

Second Report of Confidential Review of Maternal Deaths, Kerala



Why Mothers Die

Kerala
2006-2009
Observations,
Recommendations

Editors:

VP Paily, K Ambujam, Betsy Thomas



Maternal Fetal Medicine Committee
Kerala Federation of Obstetrics & Gynaecology

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Title:

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Observations, Recommendations

Edited by **Dr.V P Paily, Dr.K. Ambujam, Dr.Betsy Thomas**

Year of Publication 2012

©Kerala Federation of Obstetrics & Gynaecology (KFOG)

Published by

Kerala Federation of Obstetrics & Gynaecology (KFOG)

TOGS Academia, East Sooryagramam,

Jubilee Mission Medical College P.O., East Fort,

Thrissur, Kerala, India 680 005

The cases discussed here were provided to us without identification points like name, hospital number etc and on the assurance that anonymity will be maintained. The editorial team or assessors will not be able to answer queries about any particular case because of the above reasons and also because the concerned documents were destroyed after review process was completed.

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Design : Smriti design, Thrissur

Printing : Anaswara Offset, Cochin

ISBN 978-81-908706 - 3 -4

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Preface

While preparing the second edition of the book “Why Mothers Die – Kerala” covering the years 2006 to 2009, we were not overjoyed. The first edition for the years 2004-2005 had given us the baseline data regarding maternal mortality in Kerala. We analyzed the underlying facts and published our recommendations for action by the government, society and the community of obstetricians. Now is the time to ponder over and take stock of the changes that have taken place since the publication of the first edition.

Coincidentally, the whole world is now focusing on Maternal Mortality Ratios (MMR) as part of the Millennium Development Goals. The target date of 2015 is only three years away. We are anxiously waiting to see if India will achieve the target MMR of 109 / 100,000 live births. The latest MMR of 212 (Sample Registration System) for the years 2007-2009 gives us optimism that if India really tries, the target MMR of 109 by 2015 is achievable. But, is there sufficient commitment shown at the central level? The basic first requirement is to allot funds to support this initiative. But even now the share of India’s GDP spent on health remains pitifully low. While developed countries spend 6-10% of their GDP on health, India is spending in the range of 1-2%. Insurance coverage is at its infancy in India and most insurance agencies do not cover maternity care. All these factors leave the patient and her family having to spend on maternity care from their own pockets. Obviously the poor and unemployed will not be able to afford appropriate and timely care.

Let us look at the state of Kerala where there is the widespread perception that we have already achieved this particular Millennium Development Goal (MDG # 5) long ago. But have we? It all depends on how we look at things. Kerala was reporting an MMR of 87 even in 1992. Given that the MDG # 5 is to reduce MMR by 75% of what it was in 1990, our goal should be to reach an MMR of 22. But Kerala’s MMR as per the SRS for 2007-09 (published in June 2011) is still 81. This would imply that we have not made much progress on this front since 1990. Obviously, these figures do not tally. What are the facts? There is the need for a reliable, properly conducted study.

For a few years now, studies have shown that home deliveries have virtually disappeared from Kerala. The Family Health Survey 3 also agreed with this. If

that is the case, there should be no reason to depend on any sample survey to learn about the number of births and deaths in Kerala because the registration of births and deaths is compulsory and not registering these events is punishable. However, it is clear that the administrative machinery is terribly slack in this, especially regarding registration and compilation of maternal deaths.

Knowing the actual number of mothers dying is important but more important is to know why they die – the medical and social factors related to each death. Only with that information we can devise the appropriate strategies to tackle maternal mortality. The best method to acquire that information - recognized all over the world - is the confidential review of maternal deaths. The usual strategy of setting up an open enquiry, finding the culprit and punishing them, will not work in a complex situation like maternal death, which may be the final tragic outcome of action or inaction on the part of many players including the deceased and her family, the administrative hierarchy, the medical team and society at large. That is why we started the confidential review of maternal deaths in 2004 and still continue with it.

But our observations over the last seven years do not give us reasons to be happy. The drawback regarding maternal mortality audit in the state and the reasons for maternal death remain more or less the same. A compelling order making it mandatory on the part of hospital administrations to report all maternal deaths for confidential review is still awaited.

What did we find through the audit? We reported that in 2004-05, the MMR in Kerala would have been just above 50 per 100,000. This was an estimate after allowing for cases not reported either to CRMD or the Director of Health Services. During the period that is the focus of this second edition, the number of reported deaths has actually increased. However, this need not represent an increase in the number of deaths but rather an increase in the reporting. Unfortunately, for these years also, we still have to provide an estimate of the overall MMR for the state as we are aware that there are still many unreported deaths.

Analysis of the causes of deaths reported to CRMD during 2006-09 show almost similar trends compared to 2004-05. Obstetric hemorrhage and hypertension lead the list of causes. But one welcome change is that deaths due to amniotic fluid embolism have stabilized. Similarly, venous thromboembolism (pulmonary embolism) has not shown any escalation. But the large number of suicides (when the deaths reported to CRMD and DHS are combined) is disturbing. Suicides are often related to dowry and domestic violence. The involvement of other government departments is necessary if we want to seriously tackle this problem. Similarly, sepsis also has shown an increase. Close observation of these trends calls for

remedial action in the form of workshops and CME programmes.

Another worrying observation is the relatively high number of maternal deaths at home. As per published data, Kerala has almost 100% institutional deliveries. But preliminary data suggest that about 28 deaths occurred at home, of which eight followed home deliveries. These are not included in CRMD figures as we only analyzed those cases with records. All the same, these deaths are disturbing. Does it mean that there are large numbers of pregnant women not covered by our health system (public or private)? These cast a shadow on the claim of near 100% institutional deliveries. The administration has to take note of this.

Unfortunately, there are other disturbing trends as well. The cesarean section rates have skyrocketed in recent years with rates above 50% in 4 districts. Among vaginal deliveries, the rate of episiotomy also remains high, at about 90% in primies. There does not seem to be any perceptible change in the provision of antenatal classes or labour companions. All these are happening despite attempts by the Kerala Federation of Obstetrics and Gynecology (KFOG) to conduct workshops on emergency obstetric care and safe labour practices. Obviously, more concerted efforts are required to improve the maternity care in our state. The KFOG is willing to continue its efforts to improve maternity care as is evidenced by this ongoing CRMD despite the lack of support from some quarters. Once again, we are publishing our recommendations in this edition. We hope that it will evoke a better response this time.

V. P. Paily
State coordinator,
Confidential Review of Maternal Deaths

Foreword

It is acclaimed by many that Kerala has achieved the Millennium Development Goal as far as the maternal health is concerned. This may appear superficially correct but when one goes to the crux of the problem, it is apparent that the rate of reduction of maternal mortality in Kerala is not proportionate to the expected levels. An MMR of 81 as reported by the Sample registration system is a phenomenal achievement by any Indian standards, but Kerala ought to have reduced it to around 20-25 by this time. The lower figures of 50 reported by the CRMD cannot be ignored as it is based on solid data reported from the field. We should not waste our time debating on the discrepancies of the reported data, but it is time to chalk out strategies to bring down MMR to single digit figure.

Kerala Federation of Obstetrics and Gynaecology has been piloting studies as well as training programmes to achieve this ultimate goal. The CRMD programme is an earnest attempt to analyse the root causes of maternal mortality and the factors which foster higher MMR such as discrimination against women and substandard care. I am sure that as an organization we have succeeded in bringing out reforms in the maternal healthfront in Kerala. I only wish that the Government make an earnest attempt to understand the sincere motives of the federation and lend unconditional support for its CRMD activity.

I would like to congratulate Dr.Paily and the whole team who went into the preparation of this book. This publication may be of immense help for the obstetrician to improve their standards and for the administrators to realize the ground realities of Kerala healthcare system and rationalise its organization.

Dr.V.Rajasekharan Nair

Acknowledgements

Confidential Review of Maternal Deaths in Kerala is continuing because of the determination and commitment of many of the colleagues across the state. We are grateful to all of them for their dedication to this noble cause. To start with, we would like to acknowledge Dr. Munir Islam, Dr. Ardi Kaptiningsih, Dr. Mathews Mathai and Dr. Arvind Mathur of WHO who played a vital role in developing the present form of the review. Prof. Hugh Philpott from South Africa and Dr. Gwyneth Lewis, National Coordinator of Confidential Enquiry into Maternal Deaths in the UK who guided us in developing the reporting forms deserve special mention.

The government of Kerala supported the confidential review of maternal deaths from its inception and the help is gratefully acknowledged here. But the services of Dr. P.K Jameela, Director of Health Services has to be specially mentioned. She was a great inspiration and support. Dr.Jameela helped us by providing details of the deaths reported to the Director of Health services and stimulating the whole department of health to support the audit. We could undertake the training programme for community health centre doctors, nurses and nursing assistants only because of support from her and the National Rural Health Mission. Dr.Rajmohanam, Professor of pediatrics took lot of pains to streamline the reporting format. We are grateful to him.

Among the large number of clinicians who got involved in analyzing the cases, the non obstetrician assessors have been most inspiring. We have a team each at Thiruvananthapuram, Thrissur and Kozhikode. They have given their time and professional expertise not only to analyse the cases but also in bringing out this book. Their help is gratefully acknowledged.

Many obstetrician colleagues, senior as well as middle level, were involved in scrutinizing the case records and participated in the periodic meetings to arrive at a final diagnosis of each case. They attended these meetings without receiving any remuneration. In fact they had to spend not only for travel but even for mailing the case records. This book is the result of the hours they spent for analyzing and reporting the cases and finally writing the chapters.

There is a large contingent of people involved in running the CRMD. Some of them are listed at the end of this book under executive committee members, assessors and district coordinators. The help from all these clinicians is gratefully

acknowledged. CRMD is possible only if we get the records of the patients. It is not easy to allow somebody else to go through one's case records, especially after a tragedy has occurred. In spite of this, most of the obstetricians and their hospital administrators agreed to our request and forwarded the notes to us. We are grateful to them.

The CRMD is the flagship of the Kerala Federation, perhaps the most important service the federation is doing to the community. All the office bearers have given full support to the audit process. We would specially like to mention and thank the immediate past presidents Dr.Narayanan and Dr.N S Sreedevi, secretary general Dr.Jayandhi Raghavan, vice president Dr.Neena Thomas, joint secretary Dr.Sangeetha Menon and treasurer Dr.Rajalakshmy Janardhanan and other members of the managing council.

Dr.V Rajasekharan Nair has always been a guide and philosopher for us. He has helped in procuring the support from the government, acting as the Liaison officer of the Federation with the government. We are grateful to him for writing the foreword as well.

Dr.Sheela Paily continued to coordinate the activities even after Dr.Paily handed over the chairperson's post to Dr.Ambujam. She, with help from our office staff Mr. Ravindran and Ms Sumitha, continued to keep track of the records and provided them for analysis. This book would not have come out without their help. Smriti David has become part of the activities of Kerala Federation of Obstetrics and Gynecology. We are grateful to him for bringing out this book in spite of our giving the matter at the last possible moment.

The CRMD committee needs funds for its activities including the printing of this book. The financial help comes from workshops on vaginal surgery as well as the observership fees of doctors attending the department of O&G of Mother Hospital. We acknowledge the support of the entire department and the management of Mother hospital especially Dr.Vasanthi Jayaraj, Dr.Prashanth, Dr.Philo Akkarappatty and Head Nurse Suseela.

But we are indebted the most to the contributors to this book who are very busy clinicians. Sparing the time to write the chapter would have been really difficult. On top of all that they have gracefully accepted the alterations in the text made by the editors to maintain the general style of the book and due to constraints of cost and space.

The support of our families and colleagues is gratefully acknowledged.

Dr.V P Paily
Dr.K Ambujam
Dr.Betsy Thomas

ACRONYMS USED AND THEIR EXPANSIONS

ABG	Arterial Blood Gas
ACC	American College of Cardiology
ACE	Angiotensin Converting Enzyme inhibitors
ACN	Acute Cortical Necrosis
AF	Atrial Fibrillation
AFE	Amniotic Fluid Embolism
AFLP	Acute Fatty Liver of Pregnancy
AFV	Amniotic Fluid Volume
Ag	Antigen
AHA	American Heart Association
ALSO	Acute Life Support in Obstetrics
AMI	Acute Myocardial Infarction
AMTSL	Active Management of Third Stage of Labour
APLA	Anti Phospho Lipid Antibody
AR	Aortic Regurgitation
ARB	Angiotensin Receptor Blocker
ARDS	Acute Respiratory Distress Syndrome
ARF	Acute Renal Failure
ARM	Artificial Rupture of Membranes
AS	Aortic Stenosis
ASA	American Society of Anesthesiologists
ASD	Atrial Septal Defect
ATN	Acute Tubular Necrosis
AVM	Arterio Venous Malformation
AVR	Aortic Valve Replacement
BMI	Body Mass Index
BMV	Balloon Mitral Valvotomy (PTMC & BMV are same)
BOH	Bad Obstetric History
BUN	Blood Urea Nitrogen

CAB	Compression, Airway, Breathing
CCR	Cardio Cerebral Resuscitation
CEMD	Confidential Enquiry into Maternal Deaths
CHF	Congestive Heart Failure
CKD	Chronic Kidney Disease
CME	Continuing Medical Education
COA	Coarctation Of Aorta
CTG	Cardio Toco Graph
CVP	Central Venous Pressure
CVT	Cerebral Venous Thrombosis
DC	Direct Current
DHS	Director of Health Services
DIC	Disseminated Intravascular Coagulation
DMO	District Medical Officer
DVT	Deep Vein Thrombosis
EAS	Extra Amniotic Saline
ECC	External Cardiac Compression
EDD	Expected Date of Delivery
EF	Ejection Fraction
EFM	Electronic Fetal Monitoring
EMOCALS	Emergency Obstetric Care and basic Life Support
FFP	Fresh Frozen Plasma
FH	Fetal Heart
FHR	Fetal Heart Rate
FIGO	International Federation of Obstetrics & Gynecology
FRC	Functional Residual Capacity
FTND	Full Term Normal Delivery
GA	General Anesthesia
GDM	Gestational Diabetes Mellitus
GFR	Glomerular Filtration Rate
GO	Government Order
HAV	Hepatitis A virus
HBeAg	Hepatitis B early antigen
HBV	Hepatitis B virus
HCV	Hepatitis C virus

HELLP	Hemolysis Elevated Liver enzymes and Low Platelets
HEV	Hepatitis E virus
HICC	Hospital Infection Control Committee
HIV	Human Immunodeficiency Virus
HOCM	Hypertrophic Obstructive CardioMyopathy
HTN	Hypertension
HUS	Hemolytic Uremic Syndrome
ICD	Inter Coastal Drain
ICM	International Confederation of Midwives
ICP	Intra Cranial Pressure
ICU	Intensive Care Unit
IM	Intra Muscular
INR	International Normalised Ratio
IPPR	Intermittent Positive Pressure Respiration
IPPV	Intermittent Positive Pressure Ventilation
ITP	Idiopathic Thrombocytopenic Purpura
IUD	Intra Uterine Death
IV	Intra Venous
IVC	Inferior Vena Cava
KFOG	Kerala Federation of Obstetrics & Gynecology
LCHAD	Long Chain 3 Hydroxy Acyl Coenzyme Dehydrogenase
LDH	Lactate Dehydrogenase
LFT	Liver Function Tests
LMP	Last Menstrual Period
LMWH	Low Molecular Weight Heparin
LSCS	Lower Segment Cesarean Section
LV	Left Ventricle
LVEF	Left Ventricular Ejection Fraction
MAST	Military Anti Shock Trousers
MDG	Millennium Development Goal
MFMC	Maternal Fetal Medicine Committee
MMR	Maternal Mortality Ratio
MODS	Multiple Organ Dysfunction Syndrome
MOET	Managing Obstetric Emergencies and Trauma
MR	Mitral Regurgitation

MRI	Magnetic Resonance Imaging
MRV	Magnetic Resonance Venogram
MS	Mitral Stenosis
MV	Mitral Valve
MVP	Mitral Valve Prolapse
NIBP	Non Invasive Blood Pressure (Monitor)
NIV	Non Invasive Ventilation
NS	Normal Saline
NSAID	Non Steroidal Anti Inflammatory Drug
NASG	Non pneumatic Anti Shock Garment
NYHA	New York Heart Association
PA	Pulmonary artery
PAH	Pulmonary Artery Hypertension
PDA	Patent Ductus Arteriosus
PCOD	Poly Cystic Ovarian Disease
PDPH	Post Dural Puncture Headache
PE	Pre Eclampsia
PG E1	Prostaglandin E 1
PG E2	Prostaglandin E 2
PIH	Pregnancy Induced Hypertension
PND	Paroxysmal Nocturnal Dyspnea
PROM	Premature Rupture Of Membranes
PPCM	Peri Partum Cardio Myopathy
PPH	Post Partum Hemorrhage, Primary Pulmonary Hypertension
PR	Pulmonary Regurgitation
PRAKI	Pregnancy Related Acute Kidney Injury
PS	Pulmonary Stenosis
PSVT	Paroxysmal Supraventricular Tachycardia
PTMC	Percutaneous Trans Mitral Commissurotomy
PT	Prothrombin Time
PTT	Partial Thromboplastin Time
RBBB	Right Bundle Branch Block
RFT	Renal Fuction Tests
RHD	Rheumatic Heart Disease
RL	Ringer Lactate
ROM	Rupture Of Membranes

RRT	Renal Replacement Therapy
RSOV	Ruptured Sinus Of Valsalva
RTA	Road Traffic Accident
RVSP	Right Ventricular Systolic Pressure
RV	Right Ventricle
RUQ	Right Upper Quadrant
SAB	Sub Arachnoid Block
SAMM	Severe Acute Maternal Mortality
SEARO	South East Asia Regional Office(of WHO)
SGOT	Serum Glutamic Oxaloacetic Transaminase
SGPT	Serum Glutamic Pyruvic Transaminase
SIRS	Systemic Inflammatory Response Syndrome
SLE	Systemic Lupus Erythematosus
TC	Total Count
TOF	Tetralogy Of Fallot
TR	Tricuspid Regurgitation
TTP	Thrombotic Thrombocytopenic Purpura
UFH	Unfractionated Heparin
VSD	Ventricular Septal Defect
VTE	Venous Thrombo Embolism
VT	Ventricular Tachycardia
vWF	Von Willebrand Factor
WHO	World Health Organisation

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Contents

Preface	<i>V P Paily</i>	07
Foreword	<i>VRajasekharan Nair</i>	10
Acknowledgements	<i>VP Paily, K Ambujam, Betsy Thomas</i>	11
Acronyms and their expansions		13
List of Contributors		18

Part 1 Background & working

1. Background , continuing relevance	<i>Editors</i>	27
2. How does the system work	“	29
3. Definitions used	“	32

Part 2 In summary

4. a. Data	<i>M Deepthy, Betsy Thomas</i>	37
b. Overview & summary	<i>VP Paily, K Ambujam, Betsy Thomas</i>	47
5. Key recommendations	<i>Editors</i>	61

Part 3 Causes and Solutions

6. Hemorrhage	<i>VP Paily, K Ambujam & Betsy Thomas</i>	67
7. Hypertensive disorders	<i>VRajasekharan Nair, Deepthi Balakrishnan</i>	87
8. Amniotic fluid embolism	<i>NS Sreedevi, PV Jose</i>	97
9. Cardiovascular problems	<i>K Venugopal, Geevar Zachariah, P P Mohanan</i>	105
10. Gastrointestinal causes	<i>N Viswanath</i>	121
11. Anaesthetic causes	<i>AK Unnikrishnan</i>	135
12. Venous thromboembolism	<i>Presannakumari, P K Syamala Devi</i>	143
13. Neurological causes	<i>P C Gilvoaz & Sareena Gilvoaz</i>	153
14. Renal problems	<i>A Vimala</i>	165
15. Respiratory disorders	<i>Mathew Thomas</i>	181
16. Sepsis	<i>Sareena Gilvoaz, M Bindu</i>	191

17. Early pregnancy problems	<i>T Narayanan , Jayandhi Raghavan</i>	207
18. Less common causes	<i>Sumangala Devi</i>	211

Part 4. Practice points

19. Antenatal care	<i>Kunjamma Roy, C P Vijayan</i>	215
20. Intrapartum care	<i>Ajitha Kumari</i>	217
21. Postpartum care	<i>Sangeetha Menon</i>	221
22. Cesarean section	<i>PK Sekharan</i>	223
23. Instrumental deliveries	<i>Betsy Thomas</i>	235
24. Obstetric practice in the periphery	<i>Lalithambica Karunakaran</i>	239
25. Common pitfalls in obstetric practice	<i>C Nirmala</i>	245
26. Blood and blood components use	<i>Suseela Innah</i>	253
27. Fluid resuscitation in collapse/ shock	<i>P M Jayaraj & Vasanthi Jayaraj</i>	265
28. Severe maternal morbidity	<i>K Ambujam, Lola Ramachandran</i>	271
29. Emergency Obstetric Care & Life Support	<i>M Bindu & Neetha George</i>	277

Part 5 Annexures

Useful investigations	<i>Betsy Thomas</i>	283
Drugs: Useful Dosages & Preparations	<i>Betsy Thomas</i>	285

Part 6 Appendices

Executive committee of CRMD		295
List of obstetric assessors		295
List of Non obstetric assessors		296
List of District coordinators		296

Why Mothers Die
Kerala - 2006-09

PART-ONE

1

**Background &
working**

Chapter One:

Background, Continuing relevance

This second report represents the continuation of work done by the Kerala Federation of Obstetrics & Gynecology (KFOG) for auditing the maternal deaths in Kerala. The first report published in 2009 was acclaimed as a great contribution in understanding the extend of the problem and analyzing the cause of maternal deaths. In addition to the medical causes, it analysed the social, environmental and economic factors that contributed to the deaths. We thought of continuing the same pattern in this report also.

The Start

In January 2003, the South East Asia Regional Office (SEARO) of WHO convened a workshop in New Delhi for countries within the region to consider various types of maternal mortality audits. This book's editor, Dr. V. P. Paily, was a participant at this workshop. As a follow-up to the workshop, SEARO helped to organize a similar workshop in Kerala to study the matter further. Dr. Gwyneth Lewis from the United Kingdom and Dr. Hugh Philpott from South Africa participated in this workshop and guided the Kerala representatives in the discussions and decision-making. It was decided at that meeting to conduct a trial run of a Confidential Review of Maternal Deaths (CRMD) - similar in approach to the United Kingdom CEMD - for one month in December 2003. After revising the data collection and analysis methodology based on feedback received, the actual review began from 1st January, 2004.

The first report showed that the number of deaths may be less than what is projected in the basic sample surveys even after allowing for under reporting. But we observed that there were many avoidable maternal deaths in the state and that there are segments of the society and areas where maternal deaths are much more common than the state average.

On continuing with the audit in the subsequent years, we find that the situation has not improved much. This is in spite of the fact that, unlike the rest of the country, Kerala has many factors that can help it achieve a much lower MMR figures. The high female literacy, the widespread availability of transportation and

communication facilities and the large number of hospitals spread across the state are only some of them.

We continued the same pattern of auditing during the period under report. The principles of confidential review enunciated in the confidential enquiry into maternal deaths in the United Kingdom were followed. Our aim was to identify the cause and avoidable factors anonymously rather than put the blame on an individual or hospital. This will help to bring out the facts behind the tragedies and help in formulating strategies to prevent them recurring in the future.

During the period covered by this report (2006 -09) the numbers of maternal deaths reported to us have increased. We do not feel that this is due to an increase in the number of deaths but only due to an improvement in the reporting pattern. But still we are concerned that there are many deaths going unreported. Similarly, for large number of deaths, the records are not available for analysis. Unless these snags in reporting are sorted out, the reliability of the reports will be eroded. We are glad that recently there has been attempt on the part of the government to streamline the reporting process. Unfortunately, the concerned order was not explicit enough and compliance is poor even now. We hope that government machinery will tighten the grip on this aspect. More stringent order compelling the reporting and putting the responsibility on the hospital administration who should enroll the help of the concerned obstetrician seems to be the only way to overcome the problem.

There was no perceptible improvement in the maternal health care scenario in the reporting years, going by the number and pattern of maternal deaths. If at all unhealthy trends like very high cesarean section rates are emerging. The need for a proper audit has only increased. The KFOG feels committed to pursue its efforts in this regard and hope that there will be widespread recognition and support for the same.

Chapter Two

How does the system work

Maternal death auditing had been going on in the state for some time. Initially it was in a fragmented manner. Government medical colleges used to have departmental meetings to review the maternal deaths. The Director of Health services started to audit maternal deaths about 15 yrs ago. A team deputed by the DMO(District Medical Officer) used to go to the hospital and conduct the audit. Since the inception of the Kerala Federation of Obstetrics & Gynecology in 2002, its maternal fetal medicine committee started to be involved in the process of maternal deaths audit.

Even before that, a pilot study was done in 2001 to find out the facts. All these got consolidated as the confidential review of maternal deaths in 2004 with support from WHO and the Department of Health, government of Kerala .

The government issued an order directing all medical officers under whom maternal death occurs to send the information to the state coordinator of confidential review (Order No 39938 /FW2/ 03/H&FWD dtd 19-12-03). The specified forms developed by the maternal fetal medicine committee were used for this. In addition to the forms, the concerned doctors were asked to send anonymised photocopies of the case records to the state coordinator. In 2010 the government issued orders streamlining the reporting process further (GO (Rt)1108/2010/H&FWD dtd 25-3-10). The reporting forms were made into four parts.

Form A :

Identifying points like name of the deceased, treating doctor and hospital.

Form B:

Details of the case records are to be entered in this and the form is forwarded to the DMO. At present there is some ambiguity regarding the despatch of this form to the CRMD. Director of Health Services has agreed to issue fresh orders regarding the despatch of the form B and anonymised case records to the CRMD coordinator. Unless form B and copy of case records are provided, confidential review cannot be done.

Form C:

This is to be filled by the treating doctor and to be presented to the audit team from the DMO's office when they visit the hospital for audit. In the case of the five government medical colleges, the principal has to organize this audit. In the audit team from the DMO's office as well as from the principal of the medical college, there will be representatives of the KFOG.

Form D:

This is to be filled up by the doctor who treated the deceased mother. He/She will reflect on the case and comment on any alternative approach in the management, that would have helped to avoid the death.

The report of the audit from DMO's office as well as the Principal's office will ultimately reach the health secretary. As can be seen, these audits are not confidential (in the sense that the audit team knows the identity of the deceased, the treating team and hospital.) Our apprehension is that unbiased conclusions will not be possible. But this system has the advantage that deficiencies, if any found regarding facilities etc will be immediately known and remedial actions can be taken. However, this is not a substitute for confidential review as done by the CRMD committee. The government also recognized this and advised to continue the CRMD as in the past.

The process of data collection and review was detailed in the first report covering the year 2004 – 05. However, it is repeated here briefly. Information about the death reaches the state coordinator through different means. By now most of the obstetricians are aware of CRMD and they take the lead in informing the CRMD committee. The district representatives of CRMD may also inform the state coordinator. Other sources of the information are newspaper reports or even hearsay.

Government had promised to make available the reporting form to each hospital. It is supposed to be available for downloading from the internet. If the CRMD coordinator knows about the death and the forms are not submitted by the treating doctor, reporting forms will be sent to the concerned doctor with request to return form A, B, and D along with anonymised case records.

Once the details are available, the state coordinator or chair person of maternal fetal medicine committee will assign a number to it and thereafter the case is known only by that number. No reference to name of the deceased, treating doctor or hospital will be made in any discussions. The form A with such details will be filed separately by the state coordinator. All these records will be destroyed before the findings are compiled and published.

There are different sets of experts to go through the records to arrive at a conclusion regarding cause of death and lessons to be learnt. There are two groups of assessors. One comprises the zonal coordinators and senior professors at state medical colleges. They constitute the Executive Committee. The second group is larger in number and are considered “general assessors.” Each set of case notes is sent to two assessors: one set to a member of the Executive Committee and the other to a general assessor. They study the case notes, fill up the Assessor form and return all documents to the State Coordinator. In cases where a non-obstetric cause is suspected, an assessor from the concerned specialty will also be involved in the assessment. We have a team of volunteers from specialties like medicine, cardiology, nephrology, neurology, gastroenterology and psychiatry, who serve as non-obstetrician assessors.

The State co-ordinator then compiles the forms and prepares a list of the cases with a summary of each case for presentation at the quarterly meeting of the assessors. The assessor who studied the case and all the executive committee members will have a chance to comment on the case. A final diagnosis on the possible cause of death is then recorded at the meeting.

After extracting the data and before publication of the findings, all the case records will be destroyed.

Of late we have encouraged junior obstetricians as well as senior post graduate students also to attend the quarterly meeting of assessors. The idea is for the younger generation of obstetricians to have a first hand knowledge about the process so that it will be continued.

We want to state again that all assessors and executive committee members give their services free. There is no TA or DA paid. Even the postal charges are borne by them. Many of them acknowledge that this is also a great learning experience for them.

Chapter Three

Definitions Used

Maternal Death

“Maternal death” is defined as the death of a woman while pregnant or within 42 days of the end of pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes. For the purpose of this study, late maternal deaths occurring after 42 days of termination of pregnancy but before the end of one year have not been considered in the analysis.

Four types of maternal deaths are noted:

1. **Direct:** Deaths resulting from obstetric complications of the pregnant state (pregnancy labour and puerperium), from interventions, omissions, incorrect treatment, or from a chain of events resulting from any of the above.
2. **Indirect:** Deaths resulting from a previously existing disease or a disease that developed during pregnancy and was not due to direct obstetric causes, but which was aggravated by the physiological effects of pregnancy e.g. heart disease complicating pregnancy.
3. **Coincidental (Fortuitous):** Deaths from unrelated causes that happen to occur during pregnancy or puerperium e.g. a motor vehicle accident.
4. **Late Maternal Death:** Death occurring between 42 days and one year after abortion, miscarriage or delivery that are due to direct or Indirect maternal causes.

This study does not include late maternal deaths.

Suboptimal Care

The term “suboptimal care” is used where it is felt that a different management strategy would have resulted in a different outcome. In the British confidential enquiry report, the term used is “substandard care.”

The British report subdivides suboptimal care into “Major” and “Minor.”

1. **Major:** A different management strategy would reasonably have been expected to alter the outcome.
2. **Minor:** A different management might have made a difference but the mother’s survival was unlikely in any case.

In this report, we have not tried to classify suboptimal care into these two categories of Major and Minor. However, in the assessment of individual cases, we have tried to indicate whether suboptimal care was present.

Why Mothers Die

Kerala - 2006-09

PART-TWO

2

IN SUMMARY

Chapter Four

4 A. The Data

Deepthy M, Betsy Thomas

The most often referred part of this book will be the causes of death and the circumstances which led to the same. This is the main purpose of CRMD too. In this chapter we analyse the causes of death over the four years 2006 – 2009. We have tried to get some epidemiological data also. The reliability of some of the data given is questionable as most of the time the team filling up the form may not have the necessary information. In some situations it may be an informed guess.

The causes of death are given in the first table (table 4.1). We have given separately the causes directly reported to CRMD and the ones to the DHS. When the same case is reported to the CRMD and DHS, it is included only under CRMD. For analysis, we have considered only those cases reported to CRMD, as the details of the cases are available only about them.

In interpreting the cause of death, the primary cause is taken into consideration eventhough the final cause also may be relevant; e.g. a patient who had atonic PPH and later obstetric hysterectomy recovered , but died after a few days due to pulmonary embolism. We have taken the stand that it should be included under PPH, as it was the primary cause which set the ball rolling.

Comparison has been done with the previous two years(2004- 05) data which reveals more or less the same trend. Districtwise distribution of deaths helps us to know which is the vulnerable area and then to dissipate information to prevent the avoidable maternal deaths. Analysis of epidemiological data reflects the general trend in the state.

Table 4A.1 Causes of deaths (CRMD and DHS given separately)

<i>Cause of deaths</i>	2006		2007		2008		2009		Total
	CRMD	DHS	CRMD	DHS	CRMD	DHS	CRMD	DHS	
Haemorrhage	31	16	12	18	22	15	10	7	131
Hypertensive disorders	11	4	9	8	13	8	16	12	81
Sepsis	7	3	7	4	11	11	7	7	57
Heart disease	5	4	4	7	4	11	4	7	46
Amniotic fluid embolism	10	4	5	6	5	4	7	2	43
Respiratory diseases	9		1	3	2	3	9	5	32
Hepatic diseases	7		4	6	4	3	4	1	29
Neurological causes	6	3	1	3	3	5	5	1	27
Suicide	2	2		1		6	3	5	19
Renal causes	3	2	3	2	5		2		17
Thromboembolism	1		3	4	3	1	2	1	15
Anesthetic causes	2		1		3		2	1	9
Accident (RTA)			1	1		2		2	6
Ectopic		1	1	1		2	2		7
Accidental burns including lightning	1	1	1		1	1	1		6
Anaemia	1	1	1	1					4
Anaphylaxis			1				1		2
Postpartum collapse	2				1		1		4
Surgical /gyne causes	2	2	2				1		7
HIV	1	1							2
MTP complication				1	1		1		3
PPS complication		1							1
Sickle cell crisis								1	1
Viral infection		1					1	1	3
Obstructed labour			1						1
Malignancy			1	3		3	1		8
Malaria				1					1
Homicide						2		3	5
Diabetes						1			1
Snake bite	1								1
Abortion				1					1
Unknown	7	16		32	1	16	4	30	106
Total	109	62	59	103	79	94	84	86	676

Table 4A. 2 Year wise break up of deaths

Year	No. of deaths
2006	171
2007	162
2008	173
2009	170

The commonest cause continues to be Hemorrhage except in 2009 when hypertension topped the list. Another notable feature is the third position occupied by sepsis. A comparison has been done with the CRMD 2004 – 2005 (only those cases reported to CRMD where the cause of death is confirmed are analysed in this figure)

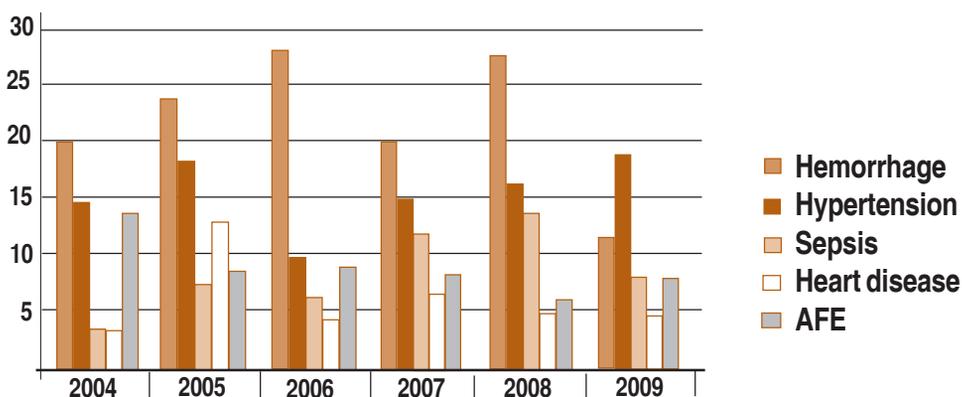


Fig.4A.1: Percentage contribution to the maternal deaths (commonest causes)

Epidemiological aspects

The various epidemiological data were analysed, eg: the district, age, religion , patient's and husband's educational status, their occupation, the type of health care providers (levels one, two , three, government or private etc), mode of delivery, type of hospital where death occurred, neonatal outcome etc.

District wise distribution of deaths

The district wise distribution shows a concentration of cases in central Kerala. It may be because the reporting is better in these districts, Nevertheless seeing the alarming figures of Malappuram and Palakkad, special workshops devoted to prevention of maternal deaths were undertaken by KFOG which, we hope will bring about a definite change.

Table 4A. 3 Districtwise distribution of deaths

District	2006		2007		2008		2009	
	CRMD	DHS	CRMD	DHS	CRMD	DHS	CRMD	DHS
Alleppey	3	1	2	1	2	3	2	1
Calicut	13	3	3	13	7	4	5	3
Ernakulam	3	6	4	4	5	8	17	9
Idukki	6	-	-	7	2	4	3	-
Kannur	11	2	5	5	7	4	4	2
Kasaragod	2	2	3	-	3	3	1	-
Kottayam	6	1	5	2	2	3	5	8
Malappuram	8	17	7	34	11	18	8	20
Palakkad	12	14	6	19	9	15	11	7
Pathanamthitta	4	1	2	2	2	5	6	2
Quilon	9	4	6	4	7	5	8	11
Thrissur	7	3	5	4	8	9	7	4
Trivandrum	20	4	8	2	12	5	9	7
Wayanad	5	4	3	5	2	8	0	12
Total	109	62	59	103	79	94	84	86

Age at Death

24.4 percentage of the deceased women were in the age group of 30 to 39 which shows the higher risk associated with late child bearing.

Table 4A .4 Age of the deceased

Age	2006	2007	2008	2009
<20	8	4	2	1
20 – 29	69	40	58	64
30 – 39	30	14	19	18
40 and above	2	1	0	1

Religion

The distribution roughly reflects Kerala's general distribution.

Table 4A.5 Religion

Religion	2006	2007	2008	2009
Hindu	63	34	50	47
Muslim	30	18	23	18
Christian	16	5	6	10
Unknown	0	2	0	9

Educational status of the deceased

The higher literacy rate in the state is reflected here but in majority the educational status is unknown. Actually only when the death has occurred we go back for such data, which most often will not be available in the case records. It is always better to have a page containing all the data like education, income, occupation etc. in the antenatal cards so that these data are more readily available.

Table 4A. 6 Educational status of the deceased

Educational Status	2006	2007	2008	2009
Primary	11	12	6	10
Secondary	19	12	19	18
Graduate	8	5	7	7
Unknown	69	30	47	49
Nil	2	0	0	0

Occupation of the deceased

Most of the deceased were un employed which indirectly indicates the socio economic status of the family . In many, the occupation is unknown, but presumably most of them were housewives too.

Table 4A. 7 Occupation of the deceased

Occupation	2006	2007	2008	2009
House wife	57	35	41	30
Teacher	1	1	2	1
Clerk	1	0	1	1

Nurse	1	0	1	1
Manual labour	1	2	1	0
Lab Technician	1	0	0	2
Other Jobs	1	0	0	0
Unknown	46	21	34	49

Husband's Educational status

Again it shows that when the educational status of the couple goes up there is more income, more earning capacity and better access to health care facilities.

Table 4A.8 Educational status of husband

Educational status	2006	2007	2008	2009
Primary	8	10	4	12
Secondary	18	13	16	14
Graduate	6	2	6	2
Nil	2	0	0	0
Unknown	75	34	53	56

Husband's Occupation

Most of the husbands were manual laborers reflecting the poor paying capacity of the family

Table 4A.9 Husband's occupation

Occupation	2006	2007	2008	2009
Manual labour	26	14	15	10
Driver	0	3	2	2
Gulf Employee	5	3	2	0
Agriculture	3	2	1	0
Salesman	1	2	0	1
Fisherman	1	2	0	1
Business	2	0	4	2
Teacher	0	0	1	1
Office Work	2	0	2	3
Other Jobs	4	0	1	4
Unknown	65	33	51	60

Distance from nearest health facility

This important information is also missing from many charts. In Kerala except in some districts like Wayanad, Idukki and Palakkad access to healthcare is not very difficult though whether all the hospitals are equipped to tackle an emergency is a big query.

Table 4A.10 distance from the nearest health facility

Distance	2006	2007	2008	2009
<5 km	19	5	8	7
5 to10 km	4	15	13	8
>10 km	8	0	3	2
Unknown	78	39	55	67

Place of antenatal care

Data show that most of the women received some form of antenatal care.

Table 4A.11 Place of antenatal care

Place	2006	2007	2008	2009
Primary Level	16	6	11	08
Secondary Level	30	27	30	34
Tertiary Level	21	8	9	14
Dr's Consult	8	4	13	11
Unknown	14	14	16	17

Type of care provider

This shows that the referral was almost equal from both sectors. Taluk hospitals and district hospitals, in spite of all the shortage of facilities, still cater to a lot of economically backward people though we cannot ignore the fact that the health care of Kerala is becoming more private oriented. While referring from the government sector, one problem which may cause the delay is the hierarchy; like from level one to two and then to three etc which may cause loss of precious time. So depending on patients' condition she should be referred to the centre with all the facilities without much delay.

Table 4A.12 Type of facility from which patient was referred

Type of facility	2006	2007	2008	2009
Government	22	16	28	22
Private Sector	30	17	27	22
Direct admission	57	26	24	29
Unknown	0	0	0	11

Type of Delivery

Maximum number of deaths were after cesarean ; that too emergency cesarean. It need not always imply that cesarean was directly related to the death, but there were other underlying risk factors in most of them. A good number remained undelivered

Table 4A.13 Type of delivery

Type of delivery	2006	2007	2008	2009
Undelivered	16	9	5	19
Spont. Vag	26	12	23	18
Vag. Breech	4	1	3	0
Vag. Forceps	4	2	2	02
Vag. Vacuum	5	5	5	03
Elective C S	10	6	4	05
Emergency C S	42	19	32	30
Abortion	1	1	5	04
Road accident	0	1	0	0
Ectopic	1	0	0	02
Vesicular mole	0	0	0	1
Unknown	0	2	0	0

Where did the death occur (Type of Hospital)

As we can see, the vast majority of the deaths have occurred at tertiary care hospitals. It is not surprising as most of them would have been referred there when things got bad. Also, the condition in which the patients reached there is important. Most of the time they reach in multiorgan failure so that even if facili-

ties were available nothing more could have been done .The deaths occurring at level two could have been due to an unexpected massive event which did not give time to act, or a delay in decision making to shift to a higher centre. From the previous edition of this book the change noted is that the number of deaths occurring at the primary level has come down showing the trend that the obstetric services provided at such centres is coming down. Also the patients might have been transferred at an early stage due to lack of facilities.

Table 4A. 14 Level of hospital

Level of hospital	2006	2007	2008	2009
Level 1	0	2	3	2
Level 2	13	5	4	6
Level 3	94	50	72	68
Brought dead	2	1	0	1
Home	0	0	0	1
Unknown	0	0	0	6

Neonatal outcome

Year after year, in majority of the cases babies survive. The numbers remaining undelivered raise the question of the feasibility of perimortem cesarean section for better resuscitation of the mother if not for salvaging the baby.

Table 4A.15 Neonatal outcome

Neonatal outcome	2006	2007	2008	2009
Alive and well	48	30	42	44
Alive but NND	10	1	6	3
Stillbirth	17	12	16	10
Abortion	1	0	5	2
Undelivered	17	10	5	17
IUD	3	0	1	3
Unknown	13	5	4	6

Deaths reported only to DHS / DMO

Inspite of repeated reminders, a substantial number of deaths are reported

only to the DHS/ DMO and not to the State coordinator. The flaw here is that the definite cause of death cannot be ascertained as it is usually reported by the field staff, whereas in CRMD the cause is assigned after a detailed analysis by the assessors.

Deaths at home (seen in DHS data)

About 28 deaths had occurred at home , eight of them following home delivery. Regarding the other 20, we do not know where delivery occurred. We were forced to classify them under unknown causes as there was no definite cause of death known to us . This is also worrying us, as we claim to have achieved almost 100 % institutional delivery in Kerala.

Chapter 4 B

Overview of Confidential Review of Maternal Deaths, 2006-2009

V.P. Paily, K. Ambujam, Betsy Thomas

The report covering the period 2006 to '09 presents the findings of confidential review of maternal deaths (CRMD) in Kerala conducted by the Kerala Federation of Obstetrics and Gynecology(KFOG). This should be seen as a continuation of the first report.

The CRMD committee is aware of the fact that the maternal deaths are still underreported despite the assurance of confidentiality. Recently the Government has issued orders for reporting and auditing maternal deaths; but data collection continues to be incomplete in many of the cases. The various causes of deaths identified are listed in Table 4A.1 When there were multiple causes of death, the case was assigned to only one cause that seemed to be the most appropriate. We have included the cases reported to us (CRMD) and the list supplied by the Director of Health Services (DHS). If a particular case was reported to the CRMD committee and the DHS, to avoid double counting, they were included only in the list of CRMD.

The MMR for Kerala as per the available data will be as follows

Table 4B.1

	2006	2007	2008	2009
Total births (Government of Kerala)	537661	538754	510151	543180
Total deaths reported (CRMD + DHS)	171	162	173	170
MMR per 100,000 births	31.8	30.06	33.91	31.29

We are conscious of the limitations of these data. First of all the denominator, number of births, are not necessarily live births. As was done in the first edition of

the book, we just accept the data given by the department of statistics knowing that even they also will not be able to provide the exact figures for live births.

Under reporting is a concern and a major draw back in calculating the MMR. If one goes through the data provided in chapter 4, some districts can be seen with unbelievably low number of maternal deaths. Obviously, maternal deaths from such districts(eg:Kasaragod) do not get reported. There are many reasons for this, the most important one being referral pattern. Most of the serious patients from Kasaragod go to Mangalore for tertiary care. Deaths occurring there are not necessarily reported to us. If we accept that there may be upto 50% under reporting, the MMR of the state can be estimated as double the figure given in the table 4B.1. It is sad that it is still an estimate!

The main purpose of the CRMD was to identify why mothers die. A realistic analysis of remedial factors is possible only when all the circumstances of death are known. Often the assessment of the final cause of death is through informed guesswork rather than hard facts. Even when case notes are available they are often incomplete. The notes from the hospital where the death occurred are usually available but the ones from the primary centre where the process started are not. In order to prevent the occurrence of similar events in the future the crucial information required is the circumstances and details of care in the primary centre. Our attempts to collect such data were unsuccessful most of the time.

The lack of autopsy to identify the cause of death is another handicap in many cases. Though it may not be important in cases where there are enough clinical features to arrive at a diagnosis, in other cases a provision for autopsy would be helpful. Autopsy in every case may not be feasible, but where an autopsy is conducted for medicolegal reasons, the results should be made available to the CRMD committee.

In spite of deficiencies in data collection and inaccuracies in diagnosis, this confidential review could identify the major causes of deaths in most cases. Some recommendations and guidelines were drawn up regarding the management of the major contributory factors to maternal deaths. These are detailed in the relevant chapters. This chapter summarizes the major causes identified and our suggestions for improvement. But before that, the actions taken by KFOG on the basis of the first report are pertinent.

Follow up action on the basis of the first report

Reports of the first two years of CRMD had brought out some valuable data, some of which were surprises. The most common causes identified were hemorrhage, hypertension and amniotic fluid embolism. It is obvious that in modern

obstetric practice with adequate facilities the outcome of these three conditions can be improved. So our first move was to address these three causes.

The KFOG took the initiative to conduct workshops to update obstetricians and labour room nurses on the prevention and management of these complications.

Obstetric hemorrhage needed to be addressed from different angles. Prophylactic steps had to be emphasized. Newer techniques to control bleeding had to be disseminated. The correct use of blood and blood components had to be taught. We did all these through workshops in different parts of the state.

But some aspects related to death due to obstetric hemorrhage are beyond KFOG to act upon except to point out the importance of these. Blood transfusion and transport facilities are integrally related to the management of obstetric hemorrhage. The society at large and the government have to take note of this.

Has the hemorrhagic deaths come down? It is difficult to say even though the number of deaths due to hemorrhage in the last year of this report was less than that of hypertension (unreported data for 2010 and 2011 put hemorrhage again on top – Editors). More details about management of this aspect of maternal deaths can be seen later in this chapter and the chapter on obstetric hemorrhage.

Regarding hypertensive disorders, the first two years' findings had shown the deficiencies in the management. The use of magnesium sulphate as the primary anticonvulsant was very low. Due to our campaign the government was kind enough to make magsulph available in all the government institutions. Parallel to this one of the pharma companies started to aggressively market the product. Certainly there is a perceptible improvement in the use of magnesium sulphate across the state.

But the delay in controlling the acute hypertension which was evident in the first report seems to be persisting. We have tried to propagate this message as well through the workshops. It is too early to see the impact in clinical practice. Details on the management of hypertensive disorders are given later.

Amniotic fluid embolism as a cause of death during the first two years was a matter of great concern. The numbers were alarmingly high and we were fairly sure that there was an iatrogenic element in the high incidence seen in our state. We addressed the issue of use of prostaglandin (both E₁ and E₂) through our workshops and conveyed the message that high and too frequent doses are dangerous. The second aspect was the use of smooth muscle relaxants in active phase of labour. Our obstetrician colleagues, by and large, seem to have received the message and acted positively. At least, the number of deaths due to amniotic fluid embolism has stabilized. More of this will be dealt with later.

In comparing the first and second reports, the other aspects of concern are the high incidence of sepsis, the continuing threat of venous thromboembolism, and the increase in the number of suicide deaths. These will be dealt with under separate headings.

- Reporting of maternal deaths is incomplete
- More clear Government orders are required for prompt reporting of all maternal deaths. The responsibility for reporting maternal deaths should be assigned to the administrator of the hospital who should use the services of the obstetrician.
- In order to identify the cause/circumstances of death, it is essential to obtain case notes from the centre where the problem leading to death started.

Observations and comments on the findings of this report

Hemorrhage

PPH still remains the most common direct cause of death. Out of the 676 deaths reported during the four year period 2006 – 2009, 131 deaths were due to obstetric hemorrhage accounting for 19.38 %. Out of the 131 deaths due to hemorrhage, reliable categorization was possible in only 75 deaths reported to CRMD, 60 of them were due to PPH (43 atonic and 17 traumatic), 8 were due to abruption, 2 due to placenta previa and 5 due to placenta previa accreta. The speed with which hemorrhage leads to DIC and irreversible shock and death is astonishing. Often, the time taken for transporting is the cause of delay. By the time the patient reaches the tertiary centre, irreversible shock would have developed and the measures taken at the higher centre turn futile.

First-aid essential before transport of patient

In the majority of cases there was no mention of any steps taken as a first aid to arrest the bleeding or in preparation for transfer to a higher centre. The committee wonders whether or not these essential first aid steps were followed or at least whether or not active management of third stage of labour was done. Documenting the initial steps and proper and safe referral are the responsibility of the referring clinician. Re-orientation and further training of medical officers and the labour room staff is urgently required to make sure that adequate first aid measures are given before referral. Nonpneumatic antishock garment (NASG) has been tried in many centres including some in Tamil Nadu. Initial reports are in favour of NASG but we would like to try in our own setting before recommending wider use.

Steps to arrest bleeding

Once excessive bleeding is identified, quick and definitive steps are essential in an organized manner to arrest bleeding. Such steps include uterotonic agents, tamponade using packs, condoms or balloon, suturing lacerations, and employing various sutures for stepwise devascularisation of uterus.

A variety of such suturing techniques are available from which the obstetrician can choose (Cafeteria approach). There is an urgent need for practicing obstetricians to update their skills in this regard. The Emergency obstetric care and Basic Life Support certification course organized by KFOG is to fill up this need. Both the obstetrician especially in small centres and the labour room nurses should undergo this training and remain updated .

Sudden onset of DIC is possible if there is amniotic fluid embolism or if the patient bleeds profusely and goes into shock. Massive blood loss is also possible with extensive lacerations of vagina, cervix or lower segment. Once the patient goes into shock the uterus becomes soft and flabby and the vicious cycle continues. Another observation is the increased incidence of bleeding when labour is induced with prostaglandins. Prostaglandin apart from ripening the cervix softens the lower uterine segment and even if the uterus seems to be contracted bleeding can be excessive from the lower segment. The lower segment is more prone for lacerations also. The situation will be further complicated if smooth muscle relaxants like drotaverin and epidosyn are used late in labour. We would even call this entity as “Lower segment PPH”.

The committee for CRMD is concerned about the routine use of drugs like drotaverin, valethamate and hyoscine even when labour is progressing normally. We feel that these agents which are smooth muscle relaxants would cause vasodilatation and thus increase the risk of amniotic fluid embolism too apart from excessive bleeding.

The committee feels that induction should be done with proper indications and mechanical methods like Foley’s catheter and extra amniotic saline should be used in unfavourable cervix before using prostaglandins. This will help to reduce the dose of prostaglandins and thereby the risk of lacerations and bleeding. Prostaglandin E₁ is cheap and is widely used nowadays. PGE₁, 25 micrograms inserted into the posterior fornix of the vagina at 4-6 hour interval, 3 – 4 times should be the standard regime. The committee also feels that prostaglandins should not be used in the midnight for induction of labour as uterine contractions can be hypertonic leading to fetal distress at odd timings. Also, prostaglandins should not be continued after cervix has become effaced. Elective induction should not be undertaken earlier than 7 days before expected date of delivery.

Every centre where delivery occurs should have a standard protocol to deal with hemorrhage. Ensuring that an emergency trolley with necessary drugs is available in each labour room is perhaps the first step so that valuable time is saved to initiate management. Immediate volume replacement by infusing IV fluids and simultaneous measures to arrest bleeding as fast as possible is the only way to save these patients. Unfortunately simple procedures like condom tamponade or packing have not yet been incorporated into the usual practice.

Ambulance service

Delay in reaching a higher centre due to lack of transporting facility is a problem in remote and hilly areas of our state. Often the patient's family is unable to pay for an ambulance due to financial constraints. Even within big cities, rush hour traffic may delay transfer from one centre to another especially if it is not in an ambulance. The government has already taken measures to fulfill this requirement by providing "108" services with trial run at Thiruvananthapuram. But organized free ambulance service for maternity cases still remains a dream.

- Hemorrhage remains the leading cause of maternal death.
- In massive hemorrhage patient may collapse very quickly.
- First aid measures to arrest bleeding like packing or tamponade is essential especially before referring to higher centres.
- Volume replacement by enough IV fluids is the first step in a bleeding patient.
- Mechanical methods of ripening cervix using EAS should precede prostaglandins in unfavourable cervix.
- Standard recommendations regarding dose and frequency of administration should be practiced when using prostaglandins. Oxytocin acceleration should be used only if contractions are poor. Once patient has reached active phase of labour, prostaglandins should not be used for acceleration of labour.
- Use of smooth muscle relaxants should be avoided when labour has entered active phase (effaced and 4cm dilated).
- All obstetricians and labour room staff should be trained in Emergency obstetric care and Basic Life Support (EMOCALS).
- Ambulance service for maternity transfer should be made widely available and free.

Hypertensive Disorders

In the four year period under study, about 12% of the maternal deaths were due to hypertensive disorders, only second to hemorrhage which has taken a toll of 19 % of maternal deaths.

In 2009, hypertensive disorders overtook hemorrhage as per the cases reported to CRMD. This may not be the actual situation because not all cases are reported to CRMD. [Data available so far for the years 2010 and 2011 indicate hemorrhage as the leading killer. (Editors)]. But preeclampsia/eclampsia continues to be a major killer of pregnant women in Kerala. A total of 81 mortalities were reported over the four year period under review out of which data from 49 cases were available for CRMD to analyse and arrive at conclusions.

Magnesium Sulphate – the preferred anticonvulsant

WHO strongly recommends magnesium sulphate as the anticonvulsant of first choice and this has been proved by many randomized controlled trials. Magnesium sulphate has been made available in the government hospitals based on our previous recommendations. Even though there is increasing tendency to use magnesium sulphate as the principal anticonvulsant in eclampsia, it is seen that nearly 46 % patients might not have received the proper dose of drug.

The committee strongly recommends full regime to be followed to prevent recurrence of convulsions. Lowering the dose or stopping with loading dose alone may lead to recurrence of seizure. For atypical cases where the diagnosis is not definite, other standard anticonvulsants may be considered. (Please refer to the chapter on neurological disorders.)

Parenteral antihypertensives essential

Intracranial bleed was the principal cause of death in 24 %, and an equal number from multiorgan dysfunction. There has been a slight reduction in deaths due to intracranial haemorrhage during this 4 year period under review compared to the previous biennium (down to 24 % from 35 %). This may be due to our previous recommendation of starting antihypertensives once hypertension is clearly documented, even if the blood pressure is lower than 160/110 which is the recommended level in standard literature. We still stick to our previous recommendation of initiating antihypertensive treatment when the blood pressure is persistently above 140/90. This is based on the assumption that the deaths due to intracranial bleed can be reduced even when there are no clear benefits for the baby. Antihypertensives should be prescribed in sufficient dose to get the desired effect.

Most hospitals lack parenteral antihypertensives and even after I.V Labetalol became available, the high cost hinders its widespread use. I.V hydralazine is not marketed in India. Often nifedipine is the only drug available. The concern in using sublingual nifedipine is the crash fall in BP and possible myocardial ischaemia. In centres without parenteral antihypertensives, a modified sublingual use may be better than no antihypertensives at all. The drug may be administered as two or three drops every two or three minutes with BP recording in between until maximum of 5mg has been used. Oral nifedipine should be started concurrently.

Another disturbing trend is the use of oral labetalol as the first line treatment even for mild hypertension. Alpha methyl dopa and calcium channel blockers should be the first line antihypertensives in mild hypertension.

High Dependency Unit/ Intensive care units

Multiorgan dysfunction took a toll of 24 % of the mothers in hypertensive disorders.

The relative increase in the contribution from hypertensive disorders may be a pointer to the fact that any further reduction demands an increase in the quality of care, especially high dependency units, care by intensivists, and the need for multidisciplinary approach to this common obstetric problem.

Multiorgan dysfunction is the final common pathway for many other causes of maternal death, especially hemorrhagic causes. Obstetric high dependency unit is a must in centres with more than 3000 deliveries per year. There should be access to ICU in case ventilatory support is needed. Obstetric medicine physicians should be groomed to develop this interest.

Timing of termination of pregnancy

Termination of pregnancy is the definitive management of hypertensive disorders. Once eclampsia has developed termination of pregnancy without delay has to be considered. The optimal mode of termination can be decided based on the nature of cervix. Prolonged inductions are to be avoided in any case once termination is decided. Monitoring should be continued even after delivery in severe cases of preeclampsia as there can be further worsening of the condition resulting in multiorgan dysfunction and death. In the present series there were some deaths in preeclamptic women due to postpartum aggravation. So antihypertensive should be continued for a few more days if indicated.

- MgSO₄ should be promoted as the first line anticonvulsant in eclampsia and severe preeclampsia. The standard recommended dose should be followed.

- Parenteral antihypertensives like labetalol and hydralazine should be made available and affordable in all centres.
- Once severe preeclampsia sets in, priority should be given to the mother's safety and pregnancy should be terminated promptly.
- More centres with obstetric high dependency units/ICU should be established.

Sepsis

The elevation of sepsis as the third most common cause of maternal deaths in the state is frightening. It was in the 12th place during the first report. Thirty two of the 57 cases included were assigned to this category by CRMD. It is a surprising coincidence that even in the recent UK confidential enquiry report sepsis was found to be more frequent than in the previous year.

Our concern is all the more because of the multidrug resistant strains of bacteria reported from intensive care units all over the world with its notorious association with Delhi. We are not in a position to say what the exact organism was, nor its sensitivity pattern in our cases. The diagnosis in our series was mostly on clinical grounds. Some cases had very rapid progression ending in shock and DIC. The importance of prevention is all the more in our set up because most of our patients cannot afford very expensive antibiotics, leave alone activated protein C. Basic principles of aseptic practice become crucial in the labour room.

But if faced with a case of sepsis the steps to be taken are the ones described by 'Surviving Sepsis' campaign. We hope that the aggressive approach described as "bundles" in the chapter on sepsis will be taken up by more and more centres.

Heart Disease

Heart disease continues to be high in the order of causes of maternal deaths. But it is gratifying to note that the overall contribution has decreased from 9% in the earlier series to 6.8% in the present. If the cases analysed by CRMD alone are taken the incidence was only 5.13% (17/331). The types of cases also have changed. The previous series had rheumatic heart disease as the major type but the present series have congenital heart disease (operated and not operated) and patients with prosthetic heart valves. These are patterns likely to persist in the future with improvement in medical care. Still there were six cases of rheumatic valvular lesion and the importance of auscultating the precordium at the first visit should be emphasized to all trainees and practitioners.

The changed pattern of heart disease brings with it new challenges. Post surgical cases and those on replaced valves may be on anticoagulants.

We feel that the advice given by our cardiology colleagues regarding pulmonary hypertension in the first edition of this book is worth repeating here. In cases which are known to deteriorate progressively (e.g. pulmonary hypertension), once fetal maturity has been ensured (34 weeks), it is worth delivering by an elective cesarean section. In the absence of scientific studies, however, it is difficult to make such a recommendation. But our own experience of grim outcomes from the policy of waiting for spontaneous labour has made us consider this recommendation favourably. Further studies are required to settle this issue.

Amniotic Fluid Embolism

Amniotic fluid embolism continues to be a cause for concern in our series. With 43 deaths out of 676, it ranks fifth in frequency. But we have to be strict with criteria to diagnose the condition as otherwise it will be labelled as a “escape” diagnosis by the obstetrician. The CRMD committee was mindful of these facts and was strict with criteria to assign a diagnosis of amniotic fluid embolism. Twenty three out of 331 cases were finally included in this category.

It is a relief that the number of cases reported in the years 2006 -09 has stabilized . The KFOG strived hard to spread the message that high doses of prostaglandins at frequent intervals was dangerous, especially when combined with smooth muscle relaxant agents like drotaverin and valethamate in active labour. Along with increasing the risk of AFE, it contributes to genital tract trauma and PPH as well. It is heartening to note that more and more obstetric colleagues have taken up this message.

Taking steps to prevent AFE is crucial but at the same time all obstetric teams should be equipped to tackle any case of collapse, if it presents unexpectedly. The availability of resuscitation equipments and drugs in the emergency trolley is crucial. All staff, especially obstetricians and labour room nurses should be trained in steps of basic life support.

Respiratory disorders

At the planning stage of this second edition in 2010, we were terribly concerned about respiratory disorders, especially H1N1 pneumonia. It had started to spread in Kerala in the second half of 2009 and caused about 24 maternal deaths in 2010. Thankfully by 2011 the condition was under control. Yet the lessons learnt like aggressive supportive therapy in pneumonia and use of oseltamivir in H1N1 pneumonia (even if it is only suspected) are relevant.

The importance of controlling asthma and keeping a tight vigil and early delivery if respiratory disorders like asthma occur in 3rd trimester are emphasized in the relevant chapter.

Hepatic diseases

There were 19 patients assigned to this group. Acute liver failure due to HELLP or AFLP were the main causes but there were five deaths due to viral hepatitis. In the case of HELLP as well as AFLP, early recognition and prompt treatment are the keys to survival. Occasionally severe sepsis also may present with hepatic failure as part of multiorgan dysfunction.

Neurological Diseases

There were 15 deaths allotted to neurological causes out of 331 analysed by CRMD. However, there were other cases with neurological problems like intracranial bleed which are not allotted to this group. Such patients would have been grouped under primary conditions with which they were admitted eg: hypertensive disorder. The commonest problem identified was the delay in making a diagnosis. More liberal use of CT scan is recommended. The importance of head ache as a warning sign of post partum cerebral venous thrombosis should be highlighted.

Suicide

There were 19 cases in this group, of which five were reported to CRMD. We were not able to analyse circumstances and reasons for suicide. The impression gathered is that they were related to problems like dowry and domestic violence. This highlights the social angle of obstetric practice. Urgent attention of the government and social activists is drawn to these facts.

Renal causes

Pregnancy Related Acute Kidney Injury(PRAKI) which is the term preferred by the nephrologists (and not renal failure) was responsible for 13 maternal deaths out of 331. Actually there were many others who had PRAKI as part of multiorgan dysfunction but are assigned to other primary causes of death like bleeding and hypertension. Most of the time prompt resuscitation and adequate replacement of blood volume and blood pressure will help to avert kidney damage. Nephrotoxic drugs like aminoglycosides and NSAIDS will make the situation worse when there is threat to renal function due to various causes like circulatory collapse or sepsis. Hence in such situations their use has to be with extreme caution as discussed in the chapter on renal diseases. Kidney suffers in systemic illnesses like systemic lupus erythematosus and Antiphospholipid antibody syndrome. Infections in the urinary tract can ascend up to become acute pyelonephritis. In HELLP syndrome unless pregnancy is terminated promptly multiorgan dysfunction will

ensue. So in all these situations, vigilance in picking up the problems early and instituting specific treatment is essential to prevent permanent kidney damage.

Thromboembolism

Even though we were anxious about the possibility of higher incidence of thromboembolism in our patients due to higher cesarean section rates as well as changing life styles, the actual figures came as a relief- 9 out of 331. In our earlier edition, we had highlighted the importance of thromboprophylaxis and are pleased to note that many centres use it. There is a need for continued vigil in this regard. We felt that use of heparin for thromboprophylaxis may have to be modified for our population and hence the recommendations made in the first edition are reproduced in this book. However, for those who want to find out the western recommendations, the RCOG guidelines are referred to. We hope that all obstetricians will take up our recommendations on thromboprophylaxis.

Anesthetic causes

Thankfully the anesthetic deaths did not go up. But this is no reason for complacency. It should be recognized that obstetric anesthesia itself is high risk business and when there are complications like hypertension, diabetes, multiple pregnancy etc all the more so. The essential precautions are described in the chapter on anesthetic problems. While we welcome use of modern devices to monitor vital functions during surgery, the importance of the anesthetist by the side of the patient to pick up problem early and take prompt remedial action cannot be over emphasized. We hope that the anesthesiologist will wear the mantle of the intensivist and help to manage the critical situations that occur ever so often in obstetric practice.

Unknown causes

The large number of cases which had to be put under this group is a matter of concern – 106 out of 676. This only implies the need for proper auditing to know the real reason for maternal death. Even when we have the records, sometimes we will not be able to arrive at a diagnosis as happened in 12 cases out of 331 under the CRMD. This could be due to paucity of investigations or autopsy. It could also be due to complexity of the cases as sometimes clinical diagnosis may not be possible and the disease will rapidly progress to death.

In this edition of the book we have added some chapters on operative obstetrics as well as procedures like blood transfusion. The aim is to make this a useful reference manual for obstetric emergencies.

Recommendations

Some of the aspects we highlighted for action in the first edition need re-emphasis even in this. Even though there were attempts to have a net work of ambulance services for the entire state, it does not seem to have materialized. There have been reports that the pilot project in Thiruvananthapuram was successful. A concerted effort to spread the service to the entire state is urgently required. Also we would like to emphasize that maternity transport, for rich or poor, should be made free.

We highlighted the plight of the tribals in our first edition. The situation does not seem to have changed if we go by media reports as well as our own observations. There were at least 26 deaths out of 676 among tribals. This shows that at least 3.84% of deaths are among the tribals, where as they constitute only 1.14% of the population. It is true that ignorance, illiteracy, customs, inaccessible dwelling places, poor general health and poverty are factors against them. But the disproportionate number of maternal deaths among them calls for urgent attention.

The importance of nursing care in the labour room has to be highlighted. Labour room is the most stressful place to work because of the unpredictability of events. The status and pay of the labour room nurses have to be kept one step higher. The presence of an experienced compassionate nurse in the labour room is what makes the labour safe. The nurse's role has to be recognized and rewarded.

The obstetricians as well as labour room nurses have to be updated on management of obstetric emergencies as well as basic life support skills. The Kerala federation has initiated a certificate course for emergency obstetric care and basic life support. We hope that all those practicing obstetrics will take advantage of this.

We also would reemphasise the importance of insisting on adequate facilities where deliveries take place. With the limited resources we have, Kerala cannot afford the luxury of home delivery. In the west, when people talk about home deliveries, it is with the backup of emergency services that will come to the rescue immediately if any unexpected complication develops. For our country, for years to come, we feel that the institutional deliveries will have to be insisted on. It is mandatory on the part of the authorities, public or private, to ensure that minimum standards in terms of equipments and manpower are maintained. We restate here the recommendation to have accreditation of hospitals.

Continuation of confidential review

Those of us involved in the process of confidential review are convinced that just as in the developed world, this is the way forward for our country to improve

maternity care. Looking at maternal death alone is too narrow an approach. It has to be expanded to look at the welfare and survival of the newborn as well. Regarding maternal health itself, it is time to start audit of “near miss” cases. The Kerala Federation of obstetrics and gynecology is committed to improve maternal and new born care in our state. We hope that this will be reciprocated by the government and the society at large.

Chapter Five

Key Recommendations

The Editors

Key Recommendations

The Confidential Review of Maternal Deaths done since 2004 accepts the fact that many of the deaths were avoidable. There was room for improvement in the management of many of the cases reported. Some of the areas identified for improvement are as follows.

1. Improve the emergency care facilities in the labour room to deal with clinical situations such as an acute collapse due to hemorrhage, or amniotic fluid or pulmonary embolism. An emergency trolley with the required drugs, IV fluids, IV sets, cannula, cut down sets, endotracheal tubes, laryngoscopes, suction catheter, condom catheter, presterilised uterine pack etc. should be readily available in every labour room to save time. The obstetrician should ensure that the head nurse maintains the trolley in working order. Oxytocin, methergine and PgF2 alpha should be stored in the refrigerator to maintain the potency.
2. All practising obstetricians should acquire the skill of resuscitation of a collapsed patient by undergoing hands on training on basic life support. In many centres the obstetrician may be the sole person available on the spot and referring a collapsed patient without first aid may result in immediate death. Resuscitation should include, setting up intravenous access, endotracheal intubation, and external cardiac compression.
3. The obstetricians should keep themselves updated on latest guidelines and protocols in managing obstetric patients by attending CME programmes.
4. Establish an alarm system in labour rooms to call for help, especially in those labour rooms that have separate enclosures for the second stage of labour.

5. All staff working in the labour room including staff nurses and nursing assistants should be trained to maintain the labour ward and theatre clean. Proper sterilization and disinfection is a team responsibility in preventing sepsis.
6. Smaller centres should establish tie-up with higher centres for accepting obstetric emergencies. In emergency situations, the help of an experienced obstetrician or anaesthesiologist should be sought without delay. If a patient needs to be transported to a higher centre or if experienced obstetrician needs to attend to a serious patient, transport facility should be readily available either way. Maternity transport should be made free.
7. A protocol for referring patients to higher centres should be established. The referring hospital should inform the higher centre over the phone and get their concurrence before sending the patient. A detailed reference letter should be given mentioning the drugs given and the relevant investigations. If the patient is critical a doctor or a nurse should accompany the patient to make sure of administration of fluids and oxygen on the way.
8. Develop a network of ambulance services throughout the state. This facility should be free of cost to help the poor and needy patients. The ambulance service should also be available to bring blood and components and to transport expert teams in busy cities. Non-governmental organizations can be recruited to help out in this matter.
9. Make a concerted effort to ensure availability of blood and components to all centres where deliveries take place. A tie-up with the nearest blood bank can be established and the line of communication simplified so that valuable time is not lost when requesting for emergency supplies of blood.
10. The government should take the lead to ensure that life saving drugs are available in all centres. This includes magnesium sulphate, labetalol, hydralazine etc.
11. Establish a certification system for all hospitals to ensure that basic facilities are available and maintained throughout the year. This can be done by a team from the District Medical Office of Health or a voluntary agency working with approval from the government. A list of minimum facilities should be drawn up and published. Hospitals can be categorized according to a three tier system depending on what facilities they have.

12. Establish obstetric intensive care facilities in major centres and medical colleges. There should be ambulances with transport ventilators attached to such centres.
13. Increase the awareness of the public regarding the need for early booking and antenatal care to pick up complications early. They should be warned of the “Danger Signals” in pregnancy and asked to report immediately if there is any. Conducting regular antenatal classes will help in conveying valuable information to patients and their families.
14. While managing seriously ill patients, it is very important to document without fail all the efforts made, drugs given, vital signs, emergency investigations etc. If possible designate a junior member of the staff for solely recording these details with time.
15. Improve reporting of maternal deaths by making it mandatory. Make it the responsibility of the hospital administration to collect the data and forward it to the state coordinator.

Why Mothers Die
Kerala - 2006-09

PART-THREE

3



**CAUSES AND
SOLUTIONS**

Chapter Six

Obstetric Hemorrhage

V P Paily, K.Ambujam, Betsy Thomas

The wife of Shajahan, the empress Mumtaz Mahal had 14 children and died after her last child birth of a Postpartum Hemorrhage in 1630. So great was Shajahan's love for his wife that he built the world's most beautiful tomb in her memory – the Taj Mahal. Now, about 500 years later we strive hard to achieve the Millennium Development Goal (MDG) 5 (to reduce MMR by 75 %).

Key Summary Points

- Obstetric hemorrhage continues to be the leading cause of maternal deaths in the four years (2006, 2007, 2008 and 2009) under review. But in 2009, of the cases reported to CRMD hypertensive disorders had taken over.
- Of the various types of hemorrhage, atonic PPH is the most significant.
- From the notes available, it is not possible to ascertain whether active management of third stage of labour was universally followed. Active management of third stage should be practised in all women in labour because it reduces the incidence of atonic PPH.
- Most of the women had no identifiable risk factors for PPH.
- A systematic approach seems to be missing after PPH is recognized. Practicing PPH drill should be popularized.
- Postpartum patient should be closely monitored for at least two hours after delivery and transferred to the postnatal ward or room only after this period. It is desirable also to make sure that she empties her bladder before she is shifted.
- The doctor or nurse observing the postpartum patient, should also palpate and ascertain whether the uterus is contracted and whether the bleeding is within normal limits and record this in the chart. This is in addition to recording pulse and BP.
- Patients undergoing cesarean section should be monitored more closely for vitals, vaginal bleeding and urine output and status of uterine fundus.

- When patient is referred to higher centre, adequate steps to reduce bleeding until she reaches the higher centre should be taken. This means having an intravenous line with oxytocin flowing, rectal PgE1 and condom tamponade if it is a case of atonic PPH. In traumatic PPH, uterine and vaginal packing is advisable.
- Transport facilities for transferring these patients to higher centre have to be streamlined and made free.

Obstetric Hemorrhage: Key Recommendations

1. Management protocol for obstetric hemorrhage in the form of “PPH Drill” should be displayed in all labour rooms. All members of the staff including nurses, nursing assistants, and staff of blood bank and laboratory should rehearse the protocol like a “Fire drill” and should be aware of each one’s role. The senior obstetrician with the help of the hospital administrator should take the lead to carry out the necessary actions in a systematic manner.
2. Women with known risk factors for obstetric hemorrhage should be delivered in centres with facilities for blood transfusion, laboratory work up and surgical procedures
3. Active management of third stage of labour must be a routine.
4. Since PPH is not predictable in every case, all women in labour should have an intravenous line.
5. Initial management of PPH includes early recognition followed by prompt resuscitation and simultaneous search for the cause of bleeding.
6. When medical management has failed to stop the bleeding, condom tamponade may be tried before resorting to surgical management. Condom tamponade or packing should be done depending on the type of PPH before referring a bleeding patient to a higher centre.
7. Obstetricians in training, as well as those in service should be well versed with surgical steps to arrest bleeding.
8. Decision for surgical management especially for obstetric hysterectomy should be taken at the appropriate time and not as a last resort.
9. In anterior placenta previa with a previous cesarean scar possibility of placenta previa accreta should be considered and managed accordingly.
10. Ongoing bleeding can lead to DIC and hence prompt replacement of blood and blood components must be ensured.

11. All labour rooms should have dedicated cervical inspection sets and a well equipped and maintained trolley with all emergency drugs.
12. Patients after delivery should be closely observed for about two hours during which vital signs are recorded and uterus is palpated to ensure sustained contraction. This should be documented in the chart.
13. Make sure that bladder is emptied before patient is shifted to the ward or room.
14. All the steps taken to manage PPH should be systematically documented.
15. Clinical Audit programme should be practised in each centre. Audit should include near miss cases and positive steps in the management should be appreciated.

Summary of findings

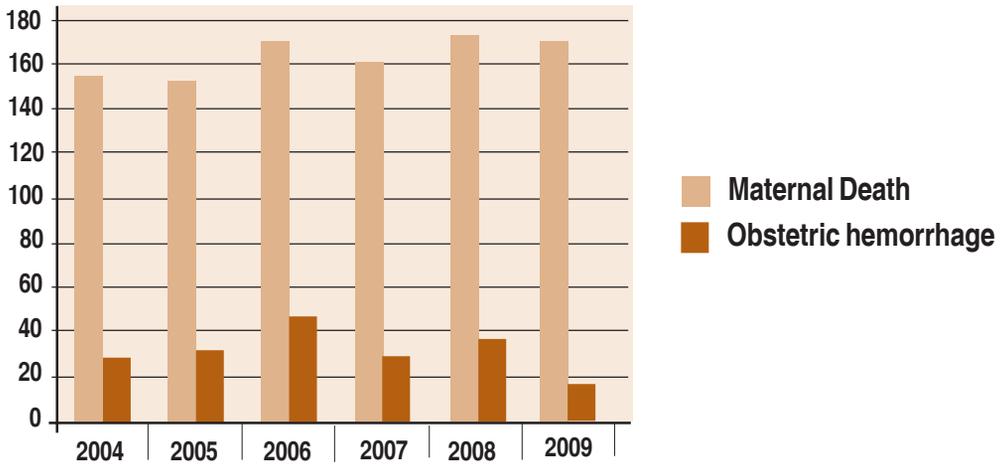
The commonest cause of maternal death continues to be obstetric hemorrhage. Out of the 676 deaths reported during the four year period 2006 – 2009, 131 deaths were due to obstetric hemorrhage accounting for 19.38 %. The yearwise break up is shown in the following table.

Table 6.1 Maternal Mortality due to obstetric hemorrhage

	Pilot study	CRMD +DHS					
	2001	2004	2005	2006	2007	2008	2009
No.of maternal deaths	105	154	153	171	162	173	170
No. of deliveries		552740	574857	537661	538754	510151	543190
Hemorrhage	23	29	32	47	30	37	17
% hemorrhage	22%	20%	21%	27.49%	18.5%	21.39%	10%

Each year of the assessment period, hemorrhage continued to top the list except in 2009 when hypertensive disorders stood first contributing to 16.48 % (28 out of 170).

Figure 6.1 Ratio of PPH as a cause of maternal death



Out of the 131 deaths due to hemorrhage, reliable categorization was possible in 75 deaths reported to CRMD, 60 of them were due to PPH (43 atonic and 17 traumatic including 2 rupture uterus) eight due to abruption , two due to placenta previa and five due to placenta previa accreta.

Table 6.2 Deaths due to hemorrhage

Type of hemorrhage	Number
Atonic PPH	43
Traumatic PPH	17
Abruptio Placentae	8
Placenta Previa	2
Placenta Previa Accreta	5
Total	75

Out of these 75 patients 31 had undergone obstetric hysterectomy and 10 out of them had relaparotomy. Most of the patients were diagnosed to be in DIC.

Table 6.3 Type of delivery

Type of delivery	Number
Normal	36
Instrument	8
Elective LSCS	12
Emergency LSCS	19
Total	75

All the eight instrumental deliveries were with vacuum except in one case of atonic PPH where it is not clear which instrument was used. It does not mean that vacuum is associated with increased mortality; majority of our obstetricians use only vacuum.

Table 6.4 Method of induction among the deceased

Method of induction	Number
PG E1	11
PG E2	4
Extra amniotic saline	2

This table also shows the trend of induction methods in our state.

Table 6.5 Deaths after LSCS

Type of PPH – Post LSCS	Number
Atonic	22
Traumatic	6

In the cases of traumatic PPH after cesarean, the primary obstetrician could have played a crucial role.

Post Partum Hemorrhage (PPH)

PPH is an obstetric emergency that can follow vaginal delivery or cesarean. There has been no significant change in the definition or classification over the past 50 years; this does not reflect the advances made in medical and surgical treatment over this period. A widely used definition currently is that proposed by the WHO in 1990 as ‘**any blood loss above 500ml from the genital tract during delivery**’. It is a major cause of maternal morbidity and mortality with sequelae such as shock, renal failure, ARDS, coagulopathy and Sheehan’s syndrome.

Atonic PPH

Uterine atony the most common cause of PPH is reported in 43 patients. It can occur after normal delivery, instrumental delivery and cesarean section. We could not find a definite association with any factors like age, parity, induction, augmentation etc. However the use of drugs like epidosin, hyoscine, and drotaverin late in active labour still continues in some parts of our state though we agree that its use has reduced compared to the previous years.

Learning from Examples- 1

Primi 23yrs, admitted at 39⁺⁵ weeks for safe confinement. Next day at 8:30 am, cervix 50 % effaced, 1 finger loose, Vertex at -3. ARM done, oxytocin started. At 3:30 pm, cervix 3-4 cm dilated, vertex -3, small caput present. Injection drotaverin and hyoscine given I V. At 7 pm, rim of cervix, head at 0 station. At 8:30 pm, no pole per abdomen, cervix fully dilated, head at 0 station, caput ++, no moulding. Patient not straining well, decided for vacuum – slipped thrice. FHS good, decided for emergency cesarean. Failed to get spinal, so done under GA. Baby 3.1 kg cried after initial resuscitation. Uterus very relaxed, lower segment tear on left side about 3 cm. Tear sutured, uterus closed, bilateral uterine artery ligation done. Atonic PPH noted. 4 packs blood given. No urine output. Patient kept in theatre till 3:40 am. Shifted to labour room with dopamine drip. At 3:45 am, she developed cardiac arrest. Intubated, shifted to surgical ICU and ventilated. But she arrested again and could not be resuscitated.

Learning points

1. Patient should not have been kept in the labour room in the latent phase itself. It causes unnecessary worry for the patient, relatives and the obstetrician, adding to maternal exhaustion and unnecessary intervention. It leads to crowding in the labour room and denial of care to those who really need it.
2. ARM was done too early. Ordinarily ARM should be done only in the active phase of labour unless there is indication like abruption.
3. Injections drotaverin and hyoscine given in active labour. Analysing the charts it is learnt that many of our obstetricians cannot get rid of this dangerous practice.
4. The decision to apply vacuum at 0 station with caput ++ is not wise. The vertex might have been above '0' station.
5. Adequate blood and fluid replacement at the right time is crucial for such patients.
6. Such a critical patient on dopamine drip and with no urine output should not have been shifted back to the labour room

Learning from Example - 2

26 year old, gravida 2, para 1, live 1; delivered normally at 12:50 pm a 3.35 kg baby. Patient developed atonic PPH. She continued to bleed inspite of injection prostodin, methergine and 200 microgram rectal misoprost. She was given one pack whole blood. Since uterus was relaxing in between and catheter was drain-

ing only 10 ml of blood stained urine, she was referred to the higher centre at 3:00 pm. She reached the higher centre at 3:35 pm in shock. Resuscitatory measures started, decided on emergency laparotomy, shifted to theatre, intubated, but suffered cardiac arrest. She was declared dead at 5 pm.

Learning points

1. There is no mention of use of condom tamponade, which was worth trying.
2. Uterine cavity was not explored for retained placental bits.
3. Uterine tamponade could have been given before shifting the patient so that ongoing bleeding could have been controlled.
4. Coagulation profile was not checked and acted upon in spite of severe bleeding.
5. One pack of blood was quite insufficient.
6. It was a good decision to refer the patient to the higher centre, but it is the duty of the primary obstetrician to make sure that the patient is safe during transport.

Learning from Examples – 3

This 23 yr old G2P1L1 was admitted at full term with labour pains at 8.30am. A drip of 25% dextrose was started. She progressed to full dilatation and delivered 3.1 Kg baby at 4pm. Usual care was given and she was shifted to her own room at 6pm. At 6.10pm she complained of head ache and weakness. Pulse was 96/mt, BP 120/70, catheterized 200ml urine. Methergine, prostodin etc given. Uterus was relaxed. Slight bleeding+. Was shifted to labour theatre. Asked for blood transfusion. Hb was 7.8gms, bleeding time 7 mts, clotting time 9 mts. Hemacel was started. Dopamine started and shifted to higher centre where she died. Further details were not available.

Looking through the notes, nurse has given hyoscine 1amp intravenously at 1pm, 2pm and 2.30pm. The fact that hemoglobin dropped to 7.8gms makes one suspect that the blood loss was not 'just slight'. Did we miss atonic PPH?

Learning points

- Starting 25% dextrose through a peripheral vein is not advisable.
- There are no notes to substantiate that she had a contracted uterus post-partum.
- The use of hyoscine is a deviation from our usual recommendations. The question whether it contributed to atonicity will go unanswered.

Traumatic PPH

There were 17 deaths assigned to this category. Compared to 2004 – 2005 period (10 out of 38)the proportion of traumatic PPH has reduced .The patients belonged to all the three groups – normal delivery, instrumental delivery and cesarean section. The interesting fact is that almost all the instrumental delivery patients who had traumatic PPH had vacuum delivery. It does not mean that forceps is safer for the mother, but that most of the obstetricians are using only vacuum.

Learning from Examples 1

25 year old primi, induced with PgE2 at term at 1.05 am. Membranes ruptured during vaginal examination at 8.15pm when cx was fully effaced and 3 cm dilated. Drotin, tramadol etc given. Labour progressed and she delivered a deeply asphyxiated baby weighing 3.7 kg. at 1.20 am the next day. While suturing third degree perineal lacerations patient collapsed. She was resuscitated and intubated. Laparotomy and internal iliac artery ligation done. Patient developed ventricular tachycardia during surgery. Defibrillated. Transfused enough blood and blood components. Patient was put on ventilator. Death 48 hours after delivery.

It is not mentioned whether vacuum was used and whether there was fetal distress. Collapse could be due to the profuse bleeding from the lacerations. Internal iliac artery ligation was done which is the right option in traumatic PPH. Unfortunately patient developed ventricular arrhythmias and deteriorated.

Learning points

1. Use of mechanical methods for ripening of cervix prior to use of prostaglandins would have made a difference.
2. Use of Prostaglandins at odd hours should be discouraged.
3. When labour is progressing normally avoid use of drugs like valethamate bromide, drotaverin etc which along with prostaglandins can make the lower segment soft and friable predisposing to tears and bleeding.
4. Look for bleeding from lacerations of the cervix and vagina before the placenta gets separated.
5. Inspect for lacerations after every instrumental delivery
6. First aid measures include running IV fluids rapidly, blood transfusion, suturing the lacerations under good light, adequate instruments and good analgesia.
7. A dedicated cervical inspection set should be available in all labour wards.

8. In unstable patients suturing should be done in the theatre under proper anesthesia.
9. Internal iliac artery ligation reduces the vaginal bleeding and should be done first before attempting to suture extensive vaginal lacerations especially if patient is unstable.
10. If patient is being referred to a higher centre pack the uterus and vagina tightly with a large pack or a mop large enough to serve as a pressure pack. The traditional roller gauze pack is quite ineffective for this purpose.

Learning from Examples 2

28 year old G₂P₁L₁, previous normal vaginal delivery, underwent emergency cesarean for CPD. Intra operatively, excessive bleeding from left angle of uterus. Extra sutures were taken. She was referred to a higher centre for persistent hypotension postoperatively. (No more details available from the primary centre.) When she reached the higher centre, pulse and blood pressure not recordable. Abdomen was distended. Urine output was nil. Paracentesis showed hemoperitoneum. Blood for clotting time- not getting clotted. Resuscitated and was taken up for laparotomy. Intraoperatively, two litres of hemoperitoneum, laceration of left broad ligament, hematoma of right infundibulopelvic and bilateral mesosalpinx were noted. Patient developed cardiac arrest towards the end of surgery and could not be revived.

Learning points

1. Traumatic PPH after LSCS is unfortunate. Intra operative left angle oozing should have made the obstetrician more vigilant. Steps should have been taken to achieve perfect hemostasis intraoperatively in the first instance itself.
2. Early suspicion and picking up of hemoperitoneum might have changed the outcome. All post operative patients should be closely monitored.
3. Timely referral with adequate fluid resuscitation and blood and blood products is another key issue in such cases.

Please refer the chapter on cesarean for further details.

Abruption

During the four year period there were 8 deaths due to abruption. The lesson learnt is that whenever there is an intrauterine death close to term without any definite cause, abruption has to be ruled out.

Learning from Examples 1

23 year old G2 P1 L1 referred from local hospital as abruption with hematemesis and fever. On arrival, temperature 103^o F, pulse 130/minute, in full dilatation, delivered still born 3.09 kg baby, developed atonic PPH, medical management failed. Embolisation done. She developed abdominal distension and shock. Laparotomy and hysterectomy done. Broad ligament hematoma evacuated. Irreversible shock, ventilatory support given. Declared dead after a few days.

Learning points

1. There is no mention about coagulation profile. Patient might have been in DIC by the time she arrived. More aggressive attempts at correction of DIC were called for.

Learning from Examples 2

This 28yr old gravida 5, para 4 was referred from the local hospital to the district hospital and from there to the medical college. She was severely anemic and findings were suggestive of abruptio placentae. She was full term pregnant but LMP and EDD were not known and did not have any antenatal care during this pregnancy. Within two hours of admission to the higher centre, with oxytocin drip she was delivered of a full term dead fetus weighing 2.5 Kg. Soon developed atonic PPH and DIC and hence proceeded with obstetric hysterectomy(total hysterectomy) along with transfusion of blood and FFP. Abdomen was closed with a drain. Postoperatively she continued to need ventilatory care and dopamine drip. Hemodialysis was initiated for renal failure. On fifth postoperative day there was problem with ventilation as no air entry was found on either side. It proved to be due to round worms which had migrated to trachea and blocked the air entry. These were cleared and air entry was re established but within a few hours her condition deteriorated and she died.

This sad story represents the culmination of many missed opportunities and tragedies. She was para 5 which is not very common in our state at present. She did not receive any antenatal care. At term, she had to be transported from the local hospital to the district hospital and finally to the medical college. How much time was lost and how even this transport was funded is not in our records but can be guessed considering the rest of the background. The pallor was obviously not just due to abruption. She would have carried the pregnancy up to full term with extreme anemia with abruption as a final blow. Round worm infestation of this magnitude indirectly suggests that she might have had hook worm as well leading to anemia. There are pointers to the living conditions and facilities like toilets and sanitation at home, leave alone nutrition.

At the higher centre where every attempt was made to save her went futile. The team there was trying to snatch her back from the claws of death but unfortunately failed. It is doubtful whether she would have come round even if the migrating round worms had not choked her. She was already in multiorgan failure.

Learning points

- Grand multiparity, absence of antenatal care and severe anemia still occur in our state, even though rarely.
- Seriously ill patients like her should be referred directly to tertiary centre bypassing secondary centre. Otherwise valuable time will be lost.
- At the higher centre, the team should be congratulated for achieving vaginal delivery and making her survive at least for a few days.
- A subtotal hysterectomy would have been enough.

We thought of citing this case even though it is a jolt to our ego and self esteem. It points to the actual situation in which some segments of the society still continue to live. Unfortunately, though this may be an extreme example, review of deaths reported across the state following home births and in transit make us question the validity of the claim of near 100% institutional deliveries and antenatal care.

Placenta previa accreta

With the alarmingly increasing cesarean section rates in the state this complication is likely to go up further in the coming years. We had five deaths due to placenta previa accreta.

Learning from Examples - 1

28 year old gravida 2, referred to Medical college hospital from local hospital at 34 weeks as previous LSCS with placenta previa . Elective cesarean was planned at 36 weeks. Developed pain abdomen and sweating at 5 a.m, 2 days prior to the planned date of cesarean. Paracentesis showed altered blood. Baby alive. Immediate laparotomy using vertical incision. 500gm clots and 500ml blood in peritoneal cavity. Scar had given way at the right end of lower segment and placenta was protruding through it. Baby delivered after separating the placenta. Placenta was adherent to lower segment and penetrating into bladder. Proceeded with hysterectomy. Rent on the bladder repaired by urologist. Profuse bleeding from plexus over bladder base was controlled. Developed cardiac arrest, resuscitated. Coagulopathy continued. 13 units whole blood, 7 FFP and 3 bags of platelets were transfused. Postoperatively the patient was on ventilator. Expired on the 6th day.

Learning points

Placenta previa accreta is a dreaded problem especially when it presents as an emergency. Placenta accreta should be suspected in anterior placenta previa with previous cesarean and ultrasonogram with doppler and if possible MRI should be done to confirm the diagnosis. Management of morbidly adherent placenta is a challenge. The whole team should be mentally and physically prepared to tackle the problem.

This was a difficult situation to start with. Rupture of one end of a scar with part of a percreta placenta protruding, will be difficult to tackle. The conventional thinking would be to enlarge the incision, deliver the fetus, tackle the placenta and do a hysterectomy. The team did exactly the same but could not salvage the situation.

We would suggest an alternative approach. This can be said to be wisdom of hind sight. This strategy is the result of trying the new technique described towards the end of this chapter. We would have put a vertical incision at the upper segment and delivered the fetus through that. Hysterectomy would have been attempted without disturbing the placenta(for details see the description later in this chapter). Subtotal hysterectomy, ensuring that the entire placenta is removed, would have been our plan. Bladder base is notorious for its vascularity and reactionary hemorrhage; the minimum that area is disturbed the better.

Learning from examples - 2

This 35 yr old Gravida 3 Para 2 with previous two cesarean deliveries had elective cesarean section at the peripheral hospital. She had placenta previa accreta. Her blood group was B negative. She developed PPH and needed relaparotomy and hysterectomy. Had hypotension on the table. Received 4 units of blood. Urine output decreased. Was referred to a higher centre. On admission there, pulse was 130/mt, BP 80 systolic, renal parameters were abnormal. Hemodialysis was started. Went on to develop metabolic acidosis and needed ventilator support. She succumbed three days later.

Learning points

- We feel that a previous cesarean with placenta previa should be approached in a systematic way which include preparation in terms of manpower and facilities (see below). A peripheral centre should not undertake such cases unless they can put together such facilities.
- The fact that she went in for renal failure indicates that she would have

sustained prolonged hypotension. Fluid and blood replacement would not have been fast enough, especially with her blood group being B negative.

- In previous cesarean with placenta previa, even if imaging studies do not suggest deeper invasion of placenta or abnormal vascularity, the possibility of accreta placenta should be kept in mind

Stepwise approach to placenta previa accreta

1. Establish the diagnosis and extent of placental invasion.
2. Counsel the patients and relatives about the seriousness of the situation without frightening them.
3. Do surgery (cesarean) as a planned procedure.
4. Arrange enough blood and blood products
5. Ensure presence of experienced obstetrician and urologist(if obstetrician is not confident enough to tackle the problem).
6. Insert bilateral ureteric catheter and a foley catheter in the bladder.
7. Use regional or regional with general anesthesia
8. Use vertical abdominal incision and classical cesarean on uterus without disturbing the placenta.
9. If accreta placenta is confirmed, decide whether to do hysterectomy with placenta in situ or to leave it behind. Do not try manual removal.
10. If decision is to leave placenta behind, tie the cord close to placenta and remove the excess length of cord, close the uterus and come out.
11. If the decision is for hysterectomy, apply tourniquet to both infundibulo pelvic ligaments and occlude the blood flow through common iliac arteries with specially developed clamps (see description towards the end of this chapter). Proceed with hysterectomy, upto the level of uterine clamps. Time elapsed from the time of common iliac clamps should be announced every 5 minutes. Try to complete the procedure in about 30 to 40 minutes.
12. By sharp dissection separate bladder from uterus and do a subtotal hysterectomy leaving behind part of the cervix below the level of placenta. Avoid unnecessary separation of bladder from vagina.
13. Double ligate all the pedicles.
14. Remove occluding clamps and tourniquets and ensure that circulation to lower limbs is re-established by palpating the femoral pulse.
15. Close the abdomen with a wide bore drain.
Keep the patient under close monitoring

Prevention of PPH

Active Management of Third Stage of Labour (AMTSL)

At the World Congress of FIGO in Chile in 2003, FIGO along with the International Confederation of Midwives (ICM) launched AMTSL to prevent PPH and increase the knowledge of Doctors, Nurses and Midwives in the medical and surgical treatment of PPH.

The three components of AMTSL :

1. Administration of uterotonic agents

At the delivery of the anterior shoulder, one of the following drugs can be given:

Oxytocin 10 units IM

Oxytocin 5 units slow I V bolus

Methyl ergometrine 0.2 mg slow I V

Syntometrine IM (Oxytocin 5 units + Ergometrine 0.5 mg)

Out of these the best oxytocic is oxytocin. The concern about methyl ergometrine is trapped placenta and blood pressure fluctuations. If the injection could not be given at the delivery of the anterior shoulder it should be given atleast within one minute of delivery (except methyl ergometrine I V).

2. Controlled Cord traction

Once cord pulsations have stopped clamp the cord close to perineum. Rub up a uterine contraction and apply upward pressure on the contracted uterus with the left hand. Apply controlled cord traction with the right hand. When the placental body is at the introitus, hold the body of the placenta and rotate it so that the placenta and membranes come out entire. Check that the removed placenta and membranes are complete.

3. Uterine massage after delivery of placenta

Gently massage the fundus of the uterus immediately after the placenta is delivered till the uterus is contracted. Palpate for a contracted uterus every 15 minutes and repeat uterine massage as needed during the first 2 hours.

Management of post partum hemorrhage- The PPH drill

Once PPH has been identified, management may be considered to involve 4 components – all of which must be undertaken simultaneously: (note the acronym CRMD relevant here **C** – Call for help, **R** – Resuscitation, **M** – Monitoring and investigations; **D**-Deal with bleeding)

1. Call for help

- Call experienced obstetricians
- Alert anesthetic Consultant
- Alert blood transfusion service
- Alert theatre staff
- Call more staff, nursing/paramedics

2. Resuscitation

- I V access (16G cannula x2)
- Head down tilt
- Oxygen by mask at 8 litres/min
- Transfuse blood as soon as possible
- Keep the woman warm
- Until blood is available, infuse in turn (as rapidly as required):
 - Crystalloid (eg: Normal Saline) maximum two litres
 - Colloid (Hydroxy Ethyl Starch preferred) maximum one litre

3. Monitoring and Investigations

- Blood for cross-match
- Full blood count
- Coagulation screen
- Continuous pulse and blood pressure recording (using pulse oximeter, ECG and automated BP recording)
- Foley's catheter to monitor urine output
- Central venous pressure monitoring (once appropriately experienced staff available for insertion)
- Consider transfer to intensive therapy unit

4. Deal with Bleeding

The commonest cause of primary PPH is uterine atony. However, careful clinical examination must be carried out with the patient in the lithotomy position and with good light to exclude traumatic PPH and retained products. A cervical inspection set must be kept ready in all labour wards.

Evaluate the 4 'T' s of PPH

1. Tone- Atonic uterus
2. Tissue- Retained products (placenta, membranes, clots)
3. Trauma- Vaginal/ cervical lacerations or hematoma, Rupture uterus
4. Thrombin- Coagulation failure

When uterine atony is the cause of the bleeding, the following measures should be taken:

- Repeat the dose of methergine and oxytocin (methergine 0.2 mg I V may be repeated every 10 minutes upto 5 doses (ie 1mg), oxytocin 5 units I V as bolus injection. Oxytocin infusion 20 units in 500 ml of normal saline at a rate that controls the uterine atony (about 60 to 90 drops/mt).
- "Rub up the fundus" to stimulate contractions (don't squeeze the fundus)
- Carboprost 0.25mg I M (may be repeated every 15 minutes upto a maximum of 8 doses)
- Rectal misoprost 600 µg may be used, is found to be less effective compared to oxytocin, but is useful while transferring pateint to other center.
- Ensure bladder is empty (Foley's catheter, leave in situ)
- Bimanual compression of the uterus
- Condom/balloon tamponade (smaller centres should transfer the patients to tertiary care centres at least at this stage. While transferring it is worthwhile to try condom tamponade or packing).

Conservative methods maybe tried while arranging for surgical methods:

- 1 Bimanual compression of the uterus
- 2 Uterine packing
- 3 Condom tamponade
- 4 Uterine artery embolisation is an option in centres with facility for the same.

Bimanual compression of the uterus

Bimanual compression of the uterus, with one hand in the vagina and the other hand applying pressure over the abdomen and making the uterus anteflex to control the blood flow temporarily.

Uterine packing

Uterine packing can be effective in controlling PPH if performed correctly.

After holding the cervix with ring forceps, thick rolls of gauze or surgical mops is fed into the uterus over the operator's fingers which are inserted along the posterior wall of the vagina into the uterine cavity.

Condom tamponade to control postpartum hemorrhage

Hydrostatic balloon tamponade using a catheter fitted with a condom to control massive post partum hemorrhage was first reported from Dhaka, Bangladesh. In an observational study during 2000 to 2001, 23 women with postpartum hemorrhage due to uterine atony with uncontrolled bleeding despite administration of uterotonics had the condom catheter placed and the bleeding was reported to have stopped within 15 minutes. Oxytocin IV drip was continued and condom catheter was removed after 24 to 48 hours. No further intervention was necessary. We have tried this method in many of our atonic PPH patients with very good results.

Steps:

- Keep the bladder empty by Foley's catheter
- Insert a sterile catheter into the condom(plastic catheter like Nelaton's), preferable to use two condoms one inside the other, and tie it tightly at the neck of the condom. Catheter should project about 4" inside the condom. An additional plastic catheter outside the condom(with the tip at one inch lower level) and tied to the other catheter will drain any collected blood beside the distended condom and warn about ongoing bleeding. Insert the condom inside the uterine cavity upto fundus by holding the tip of the catheter gently with the sponge holding forceps.
- Pack the cervix and vagina to prevent the condom from slipping down.
- Give 15° head down tilt.
- Connect the catheter (the one with tip inside condom) to a saline set and fill the condom with 300 to 500 ml of saline.
- Maintain the uterine contraction by continuous oxytocin drip and an abdominal binder.
- Condom is kept in place for about 24 hours. While removing let out the fluid stepwise 100ml per hour.

Surgical Methods

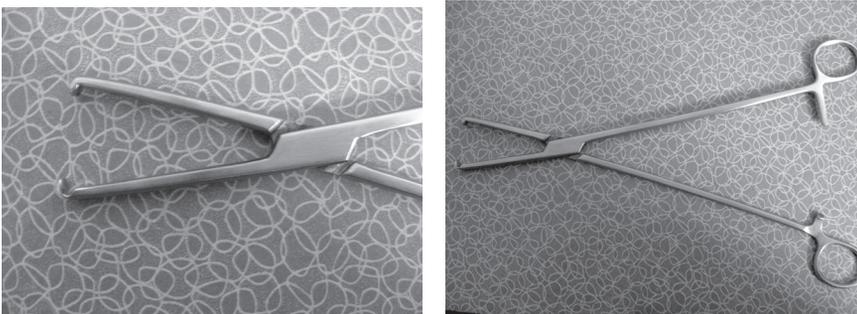
If conservative measures fail to control hemorrhage, initiate surgical methods to arrest bleeding. At laparotomy, direct intramyometrial injection of carboprost 0.5mg can be given.

- B-Lynch or Hayman's sutures (Hayman's sutures is preferable as it is easier and with less complications)
- Bilateral ligation of uterine arteries and anastomotic branch of the ovarian artery
- Bilateral ligation of internal iliac arteries. It is advisable in traumatic PPH rather than in atonic PPH in the absence of DIC (see notes on temporary common iliac occlusion later in this chapter).

Hysterectomy (resort to hysterectomy SOONER RATHER THAN LATER). During obstetric hysterectomy (preferably subtotal except in lower segment problems like placenta previa and cervical tear) arresting the bleeding at the earliest is crucial. If the situation allows, a second obstetrician should also be involved in the decision for hysterectomy. Adopt "clamp, cut and drop" technique. Pedicles can be ligated after the uterus is removed. Double ligatures have to be used for the pedicles. The seemingly avascular tissues which are cut during the procedure are also to be ligated. Out of the 31 obstetric hysterectomies which were done , 10 had to undergo relaparotomy for hemoperitoneum which added significantly to the mortality.

New strategy to control severe bleeding

Fig 6.2 Atraumatic common iliac artery clamps



Our own experience in tackling severe obstetric hemorrhage as in cases of placenta previa accreta and severe bleeding associated with conditions like rupture uterus and pelvic hematoma has made us search for simpler and practical steps to

control the bleeding which can be employed by obstetricians with average surgical skills. The established procedure of internal iliac artery ligation will not be easy in the presence of pelvic hematoma nor is it possible for most of the obstetricians. Hence we have developed a strategy of occluding the common iliac artery temporarily with specially developed clamps (see figure 6.2).

There is no need to dissect the common iliac artery for applying the clamp. Instead it can be lifted up with a Babcock clamp and the artery clamp applied with just enough tension to occlude blood flow through that. The smooth blades with a gap of about 2mm even when it is maximum tightened ensures that the arterial walls are not damaged. The only special care required is to avoid the ureters and vessels of mesosigmoid while picking up the common iliac with Babcock clamp. Occlusion of common iliac artery cuts off arterial supply to the lower limbs and pelvis. We recommend that the occlusion should not be continued for periods longer than 30 to 40 mts at a time. In tackling conditions like placenta previa accreta, the ovarian arteries also should be occluded using tourniquet.

We have so far done this in 10 cases of placenta previa accreta with very encouraging results. Bladder separation could be done by sharp dissection under vision rather than in a pool of blood which used to lead to injuries. The transfusion requirements also rapidly came down to one or two units of blood and in some cases no transfusion at all.

While the utility of this procedure is maximum in cases of placenta previa accreta, it can be employed in other cases with massive bleeding from uterus or pelvic side walls or in rupture uterus, colporrhaxis, pelvic hematoma or massive bleeding in atonic PPH or with DIC. Of course it can be employed in gynecological or urological cases and pelvic trauma where control of pelvic bleeding is needed.

It is important that the clamping of common iliac artery is limited to minimum time possible and also that on releasing the clamps the vessels are massaged to ensure that circulation is re established and that the vessels do not remain in spasm.

PPH- Key Learning Points

- Practice active management of third stage of labour in all patients irrespective of the fact whether they are having risk factors for PPH or not.
- Desirable to have an IV line with 16 – 18 G canula in all patients in the second stage.

- Despite active management of third stage, if bleeding persists, rule out traumatic PPH, retained products and inversion uterus by careful clinical examination under proper light.
- Ensure availability of dedicated cervical inspection sets in all labour wards.
- Patient who develops severe PPH should be transferred to tertiary care centres early after giving repeat dose of methergine and oxytocin IV with a drip containing 20 – 30 units of oxytocin, Inj. prostodin 250 µg IM and misoprostol 600 µg per rectum without delay.
- Use of non pneumatic antishock garments during transport is being tried. Watch out for reports of its utility.
- Patient can develop shock and may die within 2 hours of onset of post-partum hemorrhage.
- Uterine packing and balloon/condom tamponade may be tried before surgical intervention.
- Conservative surgical interventions like uterine artery ligation, B-Lynch or Hayman's brace sutures and ligation of internal iliac artery should be tried first in young nulliparous patients.
- Hysterectomy should be considered as soon as it is apparent that bleeding may pose a threat to life and not as a last resort.
- Routine tranexamic acid for PPH prophylaxis is not recommended as it is a prothrombotic drug and pregnancy itself is a thrombogenic condition.
- PPH is a leading cause of maternal mortality and many such deaths are preventable.

For blood and blood products including Recombinant factor VIIa, please refer the corresponding chapter.

For every death that is reported, we appreciate the efforts of our obstetricians in saving the lives of hundreds of women with PPH. In the unfortunate event of a maternal death we should not be put down, but let us all learn lessons which may become useful for saving the lives of many hundreds to come.

Chapter seven

Hypertensive Disorders of Pregnancy

V. Rajasekharan Nair, Deepthi Balakrishnan

Key summary points.

- Hypertensive disorders of pregnancy continue to be the second most important contributor to maternal mortality in Kerala. In the four year period under study, 12% of the maternal deaths were due to hypertensive disorders, only second to the hemorrhage which has taken a share of 19.38 % of maternal deaths.
- HELLP syndrome is the cause of death in nearly 40 % of cases of preeclampsia and eclampsia
- There has been a reduction in the incidence of cerebrovascular accidents to 24 % (compared to 34 % in the previous biennium.)
- Multiorgan dysfunction took a toll of 24 % of the mothers in hypertensive disorders.
- 87 % of deaths were due to combinations of HELLP syndrome, cerebrovascular accidents and multiorgan dysfunction.
- Continued vigil – both clinical and biochemical – may be needed following delivery in severe cases of pre eclampsia as there can be further worsening of the condition resulting in multiorgan dysfunction and death. The classical statement that delivery cures pre eclampsia should not make us complacent at least in severe cases where one should keep in mind that the pathological process can continue unabated for few more days. In the present series there were some deaths in preeclamptic women due to postpartum aggravation.
- Apart from pediatric and anesthesia colleagues, involving physicians or specialists such as nephrologist, hematologist, cardiologist or neurologist should be thought of in all severe cases of hypertension in pregnancy.

Key Recommendations

- All pregnant women should have their blood pressure checked during antenatal visits. Urine examination for albuminuria should be routinely done at every antenatal visit.
- Pregnant woman and her immediate relatives should be told regarding the warning signs of preeclampsia and eclampsia.
- Controlling blood pressure is as important as averting /controlling convulsions in severe preeclampsia and eclampsia. As we were giving lot of importance to the use of magnesium sulphate to control convulsions, the importance of controlling hypertensive crisis was not emphasised enough. Simultaneous interventions to bring down very high blood pressure along with measures to avert convulsions should be the standard approach.
- There has been a slight reduction in deaths due to intracranial hemorrhage during this 4 year period under review compared to the previous biennium. (24 % from 35 %) This may be due to our previous recommendation of starting antihypertensives once hypertension is clearly documented, even with blood pressures lower than the recommended 160/110 levels. Our previous recommendation on initiating antihypertensive treatment when the blood pressure is persistently above 140/90 holds good in the present scenario. This is based on the assumption that the deaths due to intracranial bleed can be reduced even when there is no clear benefits for the baby.
- It is observed that delay in initiating emergency measures after admission may contribute to maternal mortality, even in tertiary care set up. Triaging patients on admission and care by a dedicated team seems mandatory even in a tertiary care set up.
- The obstetric department of all hospitals should have clear written protocols for the management of both preeclampsia and eclampsia, which may be displayed in appropriate areas.
- Staff looking after the labour ward should possess resuscitation skills which are needed in cardiorespiratory collapse. It is recommended that they undergo periodic training and certification at least once in five years in Emergency obstetric care and Life Support skills organized specifically for the purpose (eg:EMOCALS by KFOG).
- The administrators should ensure supply of the essential drugs and equipments necessary for the resuscitation of collapsed patients (See appendix.).

- Alphasopa continues to be the recommended first line antihypertensive for long term control of hypertension in pregnancy. However if there is possibility of compromised liver function, as in HELLP syndrome alpha dopa should be withdrawn. Labetalol is now freely available and should be considered in all hypertensive emergencies. Hydralazine can be used whenever it is available. Nifedipine in small doses of 5-10 mg can be used to control high blood pressures, but the sublingual dose is preferably avoided. (See appendix for dosages)
- Magnesium sulphate continues to be the recommended anticonvulsant for eclampsia. For atypical cases where the diagnosis is not definite, other standard anticonvulsants may be considered.

Looking at the trend

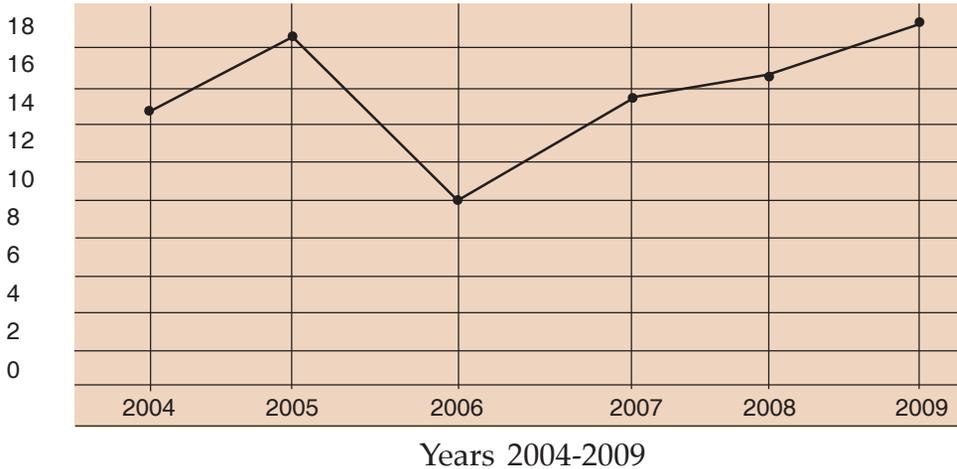
The incidence of hypertensive disorders along with other four major causes of maternal mortality are shown in table 7.1.(only those cases reported to CRMD are included.)

Table 7.1 Major causes of maternal mortality

Cause	2006	2007	2008	2009
Hemorrhage	28.4 %	20.3 %	27.8 %	11.9 %
HTN disorders	10 %	15.25 %	16.5 %	19.4 %
Sepsis	6.4 %	11.9 %	13.9 %	8.3 %
Heart diseases	4.5 %	6.8 %	5 %	4.76 %
Amniotic fluid Embolism	9.1 %	8.5 %	6.3 %	8.3 %

There are no specific trends to be highlighted, but preeclampsia/eclampsia continue to be a major killer of women in Kerala. There has been a rise in the mortality due to this problem in recent years, but the exact significance is not obvious. The relative increase in the contribution from hypertensive disorders may be a pointer to the fact that any further reduction demands an increase in the quality of care , especially high dependency units, care by intensivists, and the need for multidisciplinary approach to this common obstetric problem.

Fig 7.1 Trend in maternal mortality (%) contributed by hypertensive disorders



Summary of cases

A total of 81 mortalities with hypertensive disorder as the cause of death were reported over the four year period under review. Out of this data only 49 cases were available for CRMD to arrive at conclusions. The other cases were not considered for final analysis as the details were not available. The major causes can be summarized as follows.

Table 7. 2 Preeclampsia - causes of death (one was postpartum. The numbers do not tally because one patient would have had more than one problem.)

HELLP Syndrome	11
Cerebral bleed	7
Multiorgan dysfunction	7
Hepatic rupture	2
Pulmonary embolism	2
Acute renal failure	1
Amniotic fluid embolism	1
Abruption	1
DIC	1
ARDS	1
Encephalopathy	1
Pul edema	2
Septicaemia	1
Post partum collapse	1

Key features – preeclampsia

Out of the 49 cases scrutinised there were 27 maternal deaths from complications of preeclampsia. Development of features of HELLP was the predisposing factor in 41% of maternal deaths. Intracranial bleed was the principal cause of death in 26%, and an equal number from multiorgan dysfunction. Two deaths from hepatic hematoma rupture occurred in this series, which was not encountered in the previous biennial report of 2004 - 05. Abruptio was the reason in one case, DIC, ARDS etc became rarer entities. The principal causes of deaths in these 27 cases are tabulated. Some women had more than one factor which could account for the mortality (hence the discrepancy in the number). In one case, the condition worsened during the puerperium.

Key features – eclampsia

Table 7.3 Eclampsia -causes of death.

HELLP	8
Cerebral hemorrhage	5
MODS	5
DIC	3
Renal failure	2
Pul oedema	1
ARDS	1
PPH	1

Out of the 22 cases of eclampsia, 21 were ante/intrapartum and only one was postpartum. The principal causes leading to death of the mother is tabulated in table 7.3. Numbers do not tally because one patient would have had more than one problem.

The total number of cases of eclampsia during this four year period in Kerala could not be accounted correctly and hence any changes in the mortality trends due to eclampsia cannot be correctly calculated. Even though there is increasing tendency to use magnesium sulphate as the principal anticonvulsant in eclampsia, it is seen that nearly 46% patients might not have received the drug as seen from the records. At least one case was referred from periphery without giving the loading dose of magnesium sulphate (or any other anticonvulsant)

Key features HELLP syndrome, others

HELLP syndrome was the principal life threatening event in 40% of the maternal deaths. All the classical diagnostic features of HELLP syndrome were there

in all these cases except one. Multiorgan dysfunction was present in more than 50 % of these cases. In spite of the large number of cases of HELLP syndrome, death due to uncontrollable postpartum hemorrhage was present in only one patient. The rest of the women succumbed to multiple insults including sepsis in two cases.

Hepatic rupture, though rare has contributed to death in two women. Laparotomy and repeated attempts to control bleeding was futile, and both succumbed to continued bleeding. Both these cases were seen in preeclamptic women. Disseminated intravascular coagulation was the complicating factor in one case of preeclampsia and three cases of eclampsia. Renal failure as a cause of death was rather rare, and was seen in one preeclampsia and two cases of eclampsia. Two cases of pulmonary embolism occurred in women with preeclampsia. Pulmonary edema was the incriminating factor in two cases of preeclampsia and one case of eclampsia. ARDS was rather rare, occurred in one case of preeclampsia and in another case of eclampsia.

Learning from examples

The deadly delays continue.....

Thaddeus & Maine in 1994 highlighted the importance of the “three delays” in contributing to maternal deaths. The three delays (delay to seek care, delay to reach the facility and the delay at the facility to initiate proper treatment) continue to be a problem even in modern era, as exemplified in this vignette.

Example 1

28 year old primigravida, belonging to socially and economically weak section of the society, obviously without antenatal care, develops preeclampsia and approaches a FRU. She was found to have severe preeclampsia, with intrauterine death. Without initiating any treatment she was referred to a tertiary set up.

On admission at the tertiary care facility, she had headache, epigastric pain and her BP was 180/120. She had mild uterine contractions and was “highly uncooperative”. Diagnosed as impending eclampsia, she was started on nifedipine and atenolol and later started on magnesium sulphate. Oxytocin acceleration resulted in delivery of a still born baby. Observed for 24 hrs in labour ward. Transferred to post natal ward in the morning, but collapsed and died in the evening.

The death in this case classically depicts all the delays which could result in a maternal death even in a civilized society. The first and second delays are social factors which need particular attention by the government and the public, but the public health authorities have an equal share for not reaching out to these

deprived sections of society. The first and second delays did not result in an immediate death, but the third delay might have contributed to mortality significantly.

Some of the significant observations are :

- Poor referral from FRU, without initiating treatment.
- Diagnosed as impending eclampsia, but magnesium sulphate was started only at + 5 hrs after admission.
- 10 mg nifedipine and 50 mg atenolol were started to control the blood pressure. Better drug selection would have been appropriate.
- No thought was given to find out “ why the patient was highly uncooperative” . Probably this patient might have had a cerebral bleed already.
- Facilities such as a CT, or neurology consultation was not sought .
- No attempt was there to consider this patient as a high risk obstetric problem with aggressive treatment, which would have resulted in a different outcome.

Remember , Preeclampsia is a multisystem dysfunction

Example 2

A 29 year old second gravida, who had a cesarean section in last pregnancy was admitted to a private institution at 35 weeks gestation with symptoms of headache, vomiting and epigastric pain. She was a booked gravida in the same hospital, and her blood pressure varied between 180-200/100-110 . She was started on nicardia and eptoin and an emergency cesarean section done by 2.30 am . She developed post operative hypotension and decreased urinary output for which she was referred to a tertiary care hospital. A relaparotomy was done there about + 9 hrs , for parietal hematoma and intraperitoneal bleeding (2 litres). Abdomen closed with drains. A third laparotomy was done the next day due to continued bleeding from drains. Bilateral internal iliac artery ligation done as there was no definite bleeding sites. Renal failure persisted for which hemodialysis was done. Prolonged hospital stay with respiratory infection, renal failure, died after one month.

The following points need attention

- Surgery on an unstable patient was done at 2.30 am. It is unlikely that conditions are optimum at this time. The surgery was done without proper assessment of the severity of the disease.
- It is doubtful whether the hospital is having the necessary supporting services from other specialists.

- The problem developed in a patient who is undergoing regular antenatal check up in the same hospital.
- The drugs she received on admission are also not the ideal.
- Whether the third laparotomy was indicated at all ? The int. iliac artery ligation was a procedure of doubtful value in such a case.

This maternal loss raises several issues for which one cannot give straight forward answers, but highlights the importance of perceiving the problem of PIH on a wider perspective. Very often one may be guided by the good old dictum (modern too) that delivery is the only cure in such cases. But one has to remember that a number of target organs are insulted in severe preeclampsia. If only we can think that we are operating on a patient whose renal, hepatic, cardiovascular, neural, hematological and other systems are at stake, we can take additional precautions. One has to think whether he/she has the technical backup to tackle such multiorgan problems before delivering such a patient. We are of the opinion that in such cases, in utero transfer will be good for the mother too. Obviously there might have been good pediatric support for that doctor, but I am afraid to note that the hospital would have been poorly equipped for multiorgan dysfunction management. Repeated surgeries may be poorly tolerated by patients whose vital organs are precariously damaged. First attempt is always the best attempt. Think twice whether the odds are in your (patients') favour before putting the knife on such unstable parturients.

Recommendations to government from CRMD committee.

- Magnesium sulphate has been made available in the government hospitals based on our previous recommendations. However antihypertensives such as labetalol, and hydralazine also must be made available in the FRUs and higher levels. Injection hydralazine is not available in the market.
- In house training of the staff on life support skills is essential as acute collapse is contributing to maternal deaths in our State.

The way forward

The mortality from preeclampsia and eclampsia will (can) not be completely controlled till we understand the pathological processes involved in the initiation as well as progression of the disease. With our current understanding, we can offer only symptomatic and supportive treatment the quality of which will determine the outcome in most of these unfortunate cases. Involving other specialists, initiating early surveillance and treatment, and improving the quality of care

will go a long way in salvaging these women, till the exact etiology of this bizarre group of diseases is identified.

Appendix :

1. Maternity units should try to give attention to the following details: Supplies, equipments, drugs, staff

- Equipments** : Cot with railings, suction apparatus, oxygen cylinder/source Mouth Gag, Ambu bag, Laryngoscope, endotracheal tube Sphygmomanometer, Knee hammer, torch, stethoscope Multichannel monitor with Pulse oximeter, Defibrillator, Neonatal resuscitation trolley with supplies.
- Drugs** : Tab /Cap – Alphasol, Labetalol, Nifedipine, Hydralazine.
- Injections** : Labetalol, Magnesium sulphate, Calcium gluconate Hydrocortisone, Betamethasone, Adrenaline.
- Staff** : 1. Preferably one or more staff members should have undergone training in Life support courses .
2. 24 X 7 availability of anesthesiologist, neonatologist/pediatrician.
3. At least on call service of nephrologist/cardiologist /gastroenterologist

2. Management of Severe Hypertension:

Hydralazine (5mg I.V repeated every 20 mts) and labetalol 20mg I.V, followed by 40 mg at ten minutes, upto a cumulative dose of 300mg are the drugs of choice. Unfortunately, these are not widely available in many parts of Kerala. If these are not available nifedipine may be used to bring down BP.

3. Management of Eclampsia / Impending eclampsia

- Magnesium sulphate is the drug of choice; even if there is anuria, the loading dose can be given.
- Loading dose of 4 gm magnesium sulphate IV over 5-10 mts.+ 4gms deep IM
- Recurrent seizures – think of intracranial bleed.
- Continue magnesium sulphate for 24 hrs after last seizure.

- Monitor urine output, tendon reflexes. Stop if urine < 30ml/hr, Resp < 14/mt, SpO₂ < 95 % and with absent knee jerks.

Magnesium Sulphate Regime

Feed backs received from the practising obstetricians during EMOCALS and Quality Standards in Obstetrics training revealed that there is still some confusion regarding Mg SO₄ regime. Hence a further simplified version, as given below, is recommended by KFOG. This applies to Eclampsia and impending Eclampsia.

At zero hour 4gm IM + 4gm IV injection. (loading dose)

(The IV injection is given as 20% solution, made by adding 4gm MgSO₄ (8ml) to 12 ml of distilled water or saline and given slowly in about 5 minutes. The im dose is given at the gluteal region)

Immediately after the loading dose an IV drip is started to give MgSO₄ at the rate of 1gm per hour. (Add 4gm MgSO₄ to 100ml normal saline and give at the rate of 25ml per hour. If a micro drip set is used, 25 drops per minute will make it 25ml per hour. If it is ordinary drip set, give at the rate of 6 to 7 drops per minute.

Mg SO₄ may be continued for 24 hours after the delivery or the last fits, whichever is later.

Every four hour monitor, respiratory rate, patellar reflex and urine output.

Antidote: 10 ml of 10% calcium gluconate

Chapter 8

Amniotic Fluid Embolism

N S Sreedevi, P V Jose

Key Summary Points

- Amniotic Fluid Embolism remains one of the leading causes of maternal deaths in Kerala.
- The symptom triad, that was searched for to make a diagnosis was convulsion, coagulation problems and chest discomfort leading to collapse and cardio respiratory arrest.
- The diagnosis was made on clinical grounds alone.
- There were 27 deaths assigned to AFE out of a total of 331 deaths reported to CRMD in the four years.
- A rigid analysis shows that only for 23 cases the diagnosis of AFE will be the most appropriate.
- Once the symptoms and signs of AFE started , deterioration was rapid giving little time for rescue.
- Hyperstimulation of uterus was a common association of AFE. The use of prostaglandin (E₁ or E₂) was noted in 11 out of 23 cases.
- Artificial rupture of membranes with oxytocin also was seen associated with AFE in nine out of 23 cases.

Key Recommendations

1. Because of the association between hyperstimulation and AFE, avoid all steps that lead to hyperstimulation. In practical terms this mean adherence to the dose and time schedule of prostaglandins use (E₁ and E₂). Once cervix is effaced, prostaglandins should not be used for acceleration of labour, instead titrated doses of oxytocin alone should be used.
2. Once cervix is effaced, use of smooth muscle relaxants like valethamate, drotaverin and hyoscine will lead to vasodilatation and may increase the risk of PPH and amniotic fluid embolism.

3. Early warning signs of amniotic fluid embolism like chest discomfort in active labour should not be ignored.
4. All labour rooms should have emergency trolley which includes resuscitation equipments like laryngoscope, endotracheal tube etc.
5. If maternal cardiac arrest has occurred, perimortem cesarean will improve the chance of survival for fetus and mother. Hence this may be considered.

Summary of findings

Amniotic Fluid Embolism is an unpredictable, unpreventable and often an untreatable obstetric emergency. The management includes prompt recognition of the signs and symptoms, aggressive resuscitation efforts and supportive therapy. The following summary of findings and recommendations are made on the basis of our observations and literature review. One of the predisposing factors found is the use of oxytocic agents like prostaglandins leading to hyperstimulation. Combined use of different prostaglandins, artificial rupture of membranes and inappropriate use of oxytocin seem to increase the risk. The disease sets in rapidly and progresses quickly resulting in involvement of various systems. Early recognition and, aggressive attempts at resuscitation, could make a difference to the outcome. For achieving this, obstetricians and labour room staff have to be properly trained. The facilities for resuscitation and basic life support should be readily available in every labour room. A well maintained resuscitation trolley (or basket) which is regularly checked for expiry dates of drugs should be available in every labour room. A person should be identified for the maintenance of the emergency equipments including, laryngoscope, ambubag etc. The head nurse in charge of labour room may be the ideal choice. Another very crucial necessity is to have a mechanism evolved in each hospital for quickly summoning the help of other specialists like anesthesiologist, physician and cardiologist in case of acute collapse (eg:code blue). The availability of blood products becomes crucial in AFE as many of these cases manifest disseminated intravascular coagulopathy. Each hospital should know the quickest way to procure blood and blood products, should the need arise. Clearly written protocols for resuscitation with dose and route of administration of emergency drugs should be displayed in every labour room.

Incidence

The incidence is compared with the data collected between 1993 and 1997 from five Government medical colleges within Kerala, earlier CRMD pilot study conducted in 2001 and previous CRMD reports.

Table 8.1 Cases of AFE reported

93-97	2001	2004	2005	2006	2007	2008	2009
-	4	11	8	10	5	5	7

There was no report of amniotic fluid embolism as a cause of maternal death in the 1993-97 study. In the pilot study of 2001 there were 4 cases out of a total of 105 maternal deaths. In 2004 there were 11 out of 79 and in 2005 there were 8 out of 91 initially assigned to amniotic fluid embolism but the committee finally allotted only 10 cases to this category. In the years 2006, 2007, 2008 and 2009 the number of maternal deaths due to AFE, reported to CRMD were 27. Out of the total 27 cases reported, in four cases there is doubt regarding the diagnosis of amniotic fluid embolism. Hence further analysis will be limited to 23 cases. They are listed in table 2.

Table 8.2 List of cases

No	Age	Gr.	Gestation	Clinical Features	Induction to Death	Time Event	Mode of delivery
1	22	1	40	Dyspnoea, vomiting, collapse, convulsion	Spontaneous	8 hrs	Vaginal
2	30	3	40	Chest pain, restlessness, breathlessness Epidosisin	Pitocin Acceleration	5mts	Not delivered
3	29	2	40	Convulsion, cyanosis, DIC	PGE ₂ , PGE ₁	-	LSCS
4	27	2	40	Convulsion, BP fall, DIC	Spontaneous	3 days	Vaginal
5	33	3	40	Chest discomfort, DIC ARM	PGE ₁ 2doses,	-	LSCS
6	28	2	39	Dyspnoea, cyanosis, collapse	PGE ₁	-	Not delivered
7	28	2	40	Convulsion, Respiratory depression	ARM +oxytocin	4 hrs	LSCS
8	34	4	40	Sudden collapse	-	1 hr	Vaginal
9	31	3	40	Sudden collapse	-	-	Vacuum extraction
10	30	1	40+4 d	Convulsion, cyanosis, cardiac Arrest	PGE ₁ 3doses oxytocin	6 days 6 hr	Outlet Forceps
11	24	1	40	Fetal distress, DIC	PGE ₁	1 hr 30mts	LSCS
12	35	6	38	Respiratory distress, convulsion	-	3hrs	Not delivered

13	37	3	38	Convulsion, cyanosis collapse	-	4days	Forceps
14	30	5	40	Convulsion ,Hypotension ,DIC ARM	PGE ₁ ,oxytocin	-	Vacuum Extraction
15	37	3	39 + 4	Convulsion, cyanosis Breathlessness	ARM, oxytocin	3 hrs	Not Delivered
16	27	2	40	Breathlessness, cyanosis Echymosis	PGE ₁	6 hrs	Vacuum Extraction
17	28	3	39 + 4	Convulsion, Collapse Breathlessness	PGE ₁ , oxytocin		LSCS
18	28	3	39+4	Collapse	oxytocin	14 hrs	LSCS
19	24	2	40	Restlessness, Breathlessness, Collapse	PGE ₁ , ARM+ oxytocin	1 hr 45mts	Not Delivered
20	20	2	39 +2	Chest pain, Giddiness	ARM +oxytocin	5hrs 30mts	Vacuum Extraction
21	27	3	39	Convulsion ,Collapse	oxytocin, ARM	-	LSCS
22	30	4	38	Collapse, DIC oxytocin	PGE ₁ ,ARM	5 hrs	Normal Vaginal
23	24	1	38 + 6	Convulsion, Cyanosis	PGE ₂	2 hrs	LSCS

General observations

Age and parity

17 out of 23 were in the age group of 20-30 years. Seven out of 23 were 2nd gravida and eight were 3rd gravida

Spontaneous or induced labour

18 out of 23 cases had labour induction. Nine out of 23 had induction with PGE₁, out of these one had three and the other had two applications. Only two had PGE₁ 50 microgram used. One patient had a combination of PGE₂ and PGE₁, that too PGE₂ was instilled when cervix was already 80% effaced.

There were six cases where oxytocin was started immediately following ARM. PGE₂ was used only in two cases. The dose of vaginal PGE₁ should not be more than 25 microgram. It would be better to opt for prior ripening with mechanical methods like Foley's catheter and extra amniotic saline instillation in cases where cervix is long and firm. Once the cervix is effaced, prostaglandins should not be used for further stimulation. Instead oxytocin as a drip can be used if required. The advantage with oxytocin drip is that if there is a tendency for hyperstimulation, it can be immediately arrested by stopping the oxytocin drip.

Unlike in the previous report, in the present series the use of smooth muscle relaxants was only in one case. It was used in combination with oxytocin in a case of PROM. Many obstetricians are in the habit of using these drugs regularly in the active phase of labor. Addition of these smooth muscle relaxants which are vasodilators seem to be not only unnecessary but harmful as well. The two possible consequences of these are

1. The amniotic fluid enters the circulation through these dilated vessels, should a tear occur in the vagina or cervix.
2. The risk of excess bleeding from the cervix or vagina

Clinical presentation

AFE usually occurs during labor or in immediate postpartum period. The common scenario is a woman in late stage of labor becoming acutely dyspnoeic. A sudden change in her behaviour may be an early feature of the onset of hypoxia. Woman may complain of acute shortness of breath, sometimes with cough. There may be peripheral cyanosis. This is followed by sudden hypotension and altered mental status, confusion or coma. 50% of patients experience seizures, quickly followed by cardiac arrest. Massive DIC associated hemorrhage follows and then death. In the 23 cases we have assigned to amniotic fluid embolism, the following symptoms and signs were observed.

Table 8.3 Clinical presentation

Symptom	Number
Convulsions	12
Chest discomfort	9
Cyanosis	6
DIC	7

Diagnosis

The diagnosis should be based on the clinical findings and confirmed by demonstration of fetal squames and lanugo hair in the pulmonary vasculature. This is possible by autopsy, or by aspiration of blood from pulmonary vessels. However, fetal elements have been demonstrated in pulmonary aspirate in living patients without AFE. This gives a justification for naming this condition as “anaphylactoid reaction of pregnancy”. In most of the cases only a clinical diagnosis may be all that is possible until new lab tests become available. The complement levels are low in patients with AFE. Similarly, raised serum tryptase levels have been recorded in patients with amniotic fluid embolism. In addition fetal antigen Sialyl Tn may help to diagnose this condition.

The cluster of symptoms to be looked for to make a diagnosis are

- 1 Acute chest discomfort with dyspnoea, cyanosis and respiratory arrest.
- 2 Acute hypotension or cardiac arrest
- 3 Coagulopathy (with lab evidence or clinical features of DIC)
- 4 A brief spell of convulsions, probably the result of the acute hypoxia.

Other conditions with similar clinical presentations like drug reaction, rupture uterus, inversion of uterus or shock following massive hemorrhage should be ruled out. It has been suggested that AFE can manifest up to about 30mts after delivery.

Learning from examples

Example 1

This 30 year old 3rd gravida with 1 living child, 7yrs old and 1 abortion, was admitted with labour pains at 4 am. She was 39wks, cervix was effaced and 4 cm dilated, membranes ruptured during vaginal examination. She was started on oxytocin 5 units drip, inj.valethamate also was given half hourly for three doses. At 5.05 am cervix was 8 cm dilated and she developed chest pain and breathlessness. She was treated with oxygen by mask, betamethasone and frusemide. Soon she went into hypotension and fetal heart beat disappeared. Maternal cardiac arrest occurred. External cardiac compression was started but was not successful and she was declared dead soon.

Learning points

- Starting oxytocin immediately following rupture of membranes with a cervix effaced, 4cm dilated was not advisable.
- Injection valethamate was not necessary at this stage of labor.
- Perhaps a perimortem caesarean would have helped in resuscitation.

Example 2

29 year old woman, with one previous normal delivery two years and 9months back was admitted at term. And on date, PGE₂ was instilled at 7 in the morning when cervix was 80% effaced and 2 finger dilated. Three hours later at 10 am PGE₁ was also kept vaginally. At 12.30 pm ARM was done followed soon by oxytocin drip. At 1.40 pm cervix was fully dilated and at 1.56 pm she developed convulsions and became cyanosed. Hydrocortisone, Ambu ventilation, and IPPV

were given. Because of fetal bradycardia cesarean was done. She had cardiac arrest during cesarean section. Developed DIC and was referred to higher centre where she expired few hours later.

Learning points

- Combination of prostaglandins and oxytocin in such quick succession was not advisable.
- No need of prostaglandin when cervix is 80% effaced; use only oxytocin, if at all required.
- The minimum interval between prostaglandin administration (Six hours between PGE2 and four hours between vaginal misoprostol) should not be violated.

Conclusions

The outcome of amniotic fluid embolism is gradually changing as per literature. This may be because of more and more cases being picked up at an early stage and availability of multidisciplinary team and other facilities once the diagnosis is made. In established severe cases the results are still poor. We should strive to avoid predisposing factors mentioned in this chapter and improve the facilities for resuscitation once the diagnosis is made. All personnel working in the labour room and theatre should sharpen their resuscitation skills.

Chapter 9

Cardiovascular Diseases In Pregnancy

K.Venugopal, Geevar Zacharia, P.P. Mohanan

Key summary points

- Heart disease continues to be a major non obstetric cause for maternal mortality.
- Rheumatic heart disease, particularly mitral stenosis is the most common cardiac problem reported in the present series.
- Operated congenital heart diseases and prosthetic valve patients are increasingly presenting to the obstetrician with pregnancy and require specialised care.
- Peripartum cardiomyopathy is being recognized more frequently.

Key Recommendations

- Cardiac problems in pregnancy should be managed by a team comprising cardiologists, obstetricians and anesthetists especially in complex cases.
- High index of suspicion should be kept to rule out cardiac disease in any pregnant patient who experiences breathlessness, chest pain, palpitation or syncope in the antenatal period and they should be referred for further assessment.
- Echocardiographic evaluation which is a relatively inexpensive test along with a detailed clinical examination will help to rule out significant cardiac problems in most patients.
- Patients with Primary pulmonary hypertension, Eisenmenger Syndrome, Cyanotic congenital heart disease and patients with other cardiac diseases with severe functional disability should be advised not to conceive and undergo termination of pregnancy if already pregnant.
- Patients presenting for the first time to peripheral centres may be referred to nodal centres with facility for full cardiac evaluation early in pregnancy so that a definite strategy for management can be evolved.

- Government should consider financial assistance to patients with cardiac disease who are unable to afford surgical treatment.

Summary

Out of 331 cases of maternal deaths reported to CRMD in Kerala during the years 2006-2009, 17 were diagnosed as cardiac problems causing maternal mortality of 5.13% [46 out of 676 (6.8%) total maternal deaths]. This is in sharp contrast to the number reported during 2004-2006, 15 out of 170 (8.8%) of CRMD cases and 29/307 (9%) total cases which made cardiac problems the most common non-obstetric cause for maternal death. This drop in numbers could be attributed to increased awareness, early detection and better management of cardiac problems during pregnancy or due to under-reporting or designation of maternal cardiac deaths to other causes.

The etiological distribution of the cardiac cases were as follows.

Table 9.1 Cardiac diseases leading to maternal deaths

Rheumatic heart disease	6
Operated congenital heart disease (TOF, COA)	2
Prosthetic valve patients (AVR)	2
Primary pulmonary hypertension	1
Eisenmenger Syndrome	1
Peri partum cardiomyopathy	1
CHD, tetralogy of Fallot with brain abscess	1
Aortic aneurysm rupture (post partum)	1
Acute Pulm . edema (?Cardiogenic/?ARDS)	2

Rheumatic valvular heart disease was the major cause of cardiovascular maternal mortality. Six patients out of 17 (28%) died of RHD. Unlike the previous report, where congenital heart disease cases were not reported, there were both operated and non-operated cases of congenital heart disease in the present series. There were two cases of prosthetic valve problems and solitary cases of primary pulmonary hypertension, peripartum cardiomyopathy and aortic aneurysm rupture. There were two cases which presented with acute pulmonary edema where a definitive diagnosis of cardiogenic pulmonary edema vs ARDS was not made.

Spectrum of rheumatic valvular heart disease

There were four definite cases of rheumatic valvular heart disease. All four

patients had mitral stenosis, one patient had undergone closed mitral valvotomy in 1994 and had mild residual mitral stenosis and moderate mitral regurgitation. The other three had severe mitral stenosis.

Rheumatic mitral stenosis is the most common valvular problem confronting the obstetrician and cardiologist in pregnancy. Seventy five percentage of patients in various series elsewhere had mitral stenosis. The hemodynamic changes associated with pregnancy adversely affect mitral stenosis. The increase in heart rate and blood volume leads to increase in the trans mitral flow gradients and significantly worsen the functional status of the patient. Patients who are asymptomatic prior to pregnancy become symptomatic and may deteriorate fast with episodes of paroxysmal nocturnal dyspnea and orthopnea and occasional episodes of acute pulmonary edema. The changes are maximum during 24-26 weeks when maximum increase in blood volume may take place. Another dangerous period is the period of labour and delivery when an additional increase of 20% in cardiac output can occur. In the immediate post partum period cardiac output can rise as much as 65%. Development of atrial fibrillation will also adversely affect mitral stenosis and also may be responsible for embolic episodes. The mortality in untreated cases is 1 % which rises to 14 -17 % in the setting of atrial fibrillation. The highest risk is during intrapartum and post partum period.

Ideally if severe mitral stenosis is diagnosed before conception, patient should be treated by percutaneous valvotomy or surgery. Surgical procedures carry high fetal mortality, open heart procedures having more mortality than closed procedures. Percutaneous procedures have problems of radiation hazard to the mother and fetus and persistent hypotension due to compression of inferior vena cava. Radiation hazards can be considerably reduced by using abdominal shields. Ideal time for BMV is after 12 weeks but in unstable symptomatic patients it can be done at any time of gestation. Procedural success is 95-97%. Care should be taken to avoid development of severe mitral regurgitation as it will result in urgent mitral valve replacement with high maternal and fetal risk. Any patient diagnosed to have mitral stenosis should be carefully followed up at more frequent intervals and advised to report if any worsening of cardiac symptoms occurs. A clear strategy to intervene at any stage of pregnancy should be explained to the patients to avoid any episode of acute pulmonary edema at the time of delivery. Even in patients considered for medical management, it may be prudent to have the services of the cardiologist/anesthetist readily available at the time of delivery.

Patients with aortic stenosis, if severe as per clinical and echocardiographic findings, should be advised not to conceive prior to surgical correction of the lesion. Patients whose aortic valve lesion is severe and who has not been surgically

corrected carries a poor prognosis. If diagnosed in the first trimester they may be advised termination of pregnancy.

Regurgitant lesions like mitral and aortic regurgitation are well tolerated in pregnancy unless they are in severe heart failure or functional class IV.

Poor prognostic factors in valvular heart disease patients include LV dysfunction, severe MS, AS, prior cardiac events in previous pregnancy, transient ischemic attack, stroke or endocarditis.

Pregnancy and prosthetic valve patients

Two patients in the current series had valve replacement done in the aortic position, one for severe AR and the other for congenital heart disease VSD with AR. The management of prosthetic valve patient needs very careful evaluation and follow up in the antenatal period. The ACC/AHA guidelines 2006 say that there are three options for managing pregnant women with prosthetic valves.

- 1) UFH or LMWH between 6-12 weeks and close to term, and oral anticoagulants at other times to maintain an INR of 3
- 2) Aggressive dose adjusted UFH throughout pregnancy
- 3) Aggressive dose adjusted LMWH throughout pregnancy.

Both UFH and LMWH have to be administered with close monitoring of APTT and anti factor Xa activity. Heparin will have to be given in high doses to maintain APTT of twice control value. The regimen in consideration has to be discussed with the patient. It is also preferable to give aspirin along with this regimen. The pregnant patient with prosthetic valve should be managed by a team consisting of obstetrician, cardiologist and anesthesiologist. These patients require infective endocarditis prophylaxis with parenteral antibiotics as they belong to a high risk group of patients prone to develop infective endocarditis.

Congenital heart disease

Both cyanotic and acyanotic heart diseases were present in the current series. Cyanotic congenital heart diseases were Tetralogy of Fallot, Coarctation of Aorta operated with severe PAH and tricuspid regurgitation(TR).Cyanotic congenital heart disease has a high mortality in pregnancy and patients ideally should be advised against conception. In patients who become pregnant unknowingly, termination of pregnancy can be offered. Careful monitoring of the cardiac status, polycythemia / anemia should be done. It may be prudent to give endocarditis prophylaxis to the cyanotic heart disease patients as they come under a high risk group for development of endocarditis. Acyanotic congenital heart diseases even

if operated, should be carefully assessed as they can have life threatening arrhythmias in pregnancy. Life threatening tachy arrhythmias in pregnancy can be safely treated with cardioversion.

Pulmonary hypertension

Eisenmenger Syndrome and Primary Pulmonary hypertension are examples of precapillary pulmonary hypertension. Both diseases have high maternal and fetal mortality and pregnancy is contraindicated in these patients. These patients can deteriorate during any period of pregnancy though intrapartum and early postpartum periods are particularly risky. It is better to anticoagulate these patients as they may have a higher incidence of thromboembolic episodes. Malignant cardiac arrhythmias, right ventricular failure and pulmonary embolism are usual causes of death in these patients.

Pregnancy is not advised in this group of patients. Preventive measures like tubal ligation may be considered. If pregnancy is diagnosed early, termination of pregnancy should be recommended. Patients who continue with pregnancy should be advised to avoid stressful activities and continue their medication except ACE inhibitors. Newly introduced drugs like sildenafil and tadalafil which act through phosphodiesterase inhibition have been reported to have low teratogenicity and can be continued in pregnancy though there are no large trials in this subset of patients. The patient and bystanders should be clearly aware of the problems of maternal mortality and patients should be advised to attend tertiary care centres for delivery.

Peripartum cardiomyopathy

There was one report of a case of peripartum cardiomyopathy which was diagnosed during the last month of pregnancy who underwent emergency LSCS and was discharged on decongestive treatment. Peripartum cardiomyopathy usually is diagnosed in the last month of pregnancy or within five months after delivery. There is usually no demonstrable cause for heart failure or demonstrable cardiac problem before the last month of pregnancy. The diagnosis is established by the presence of significant systolic dysfunction, usually by echocardiography. It is more common in multipara, twin gestation and pre eclampsia. The exact etiology is unclear. It carries a high mortality 7-56%. One heartening feature about peripartum cardiomyopathy is that 50% of patients may regain almost normal left ventricular function once they recover from their initial clinical presentation. They should be on long term decongestive therapy. It is to be emphasised that they should not become pregnant again as they will deteriorate in subsequent pregnancy if the LV function is still impaired. Those who regain normal LV func-

tion may be advised to have further pregnancies after being fully evaluated with dobutamine stress echo and assessing contractile reserve. They should also be informed of the possibility of recurrence in a small subset of patients. One series has reported a recurrence of 30% in subsequent pregnancies.

Coronary Artery Disease

Though there were no reported cases of coronary artery disease in the present series, possibility of coronary artery disease should be strongly suspected in patients presenting with acute left ventricular failure. Patients with PCOD are likely to develop metabolic syndrome and are also prone for coronary artery disease .

Arrhythmias in pregnancy

Pregnancy is an arrhythmogenic state. Both ventricular and supraventricular ectopics are common and do not result in any serious maternal mortality or morbidity. Life threatening arrhythmias occur in patients with underlying serious cardiac problems. Care should be taken to rule out undiagnosed associated problems like thyroid abnormalities. Arrhythmias like atrial fibrillation which have a propensity for thrombus formation and embolic episodes, should be treated with anticoagulation. Electrical cardioversion is safe in pregnancy and can be done in patients who present with tachyarrhythmias and hemodynamic compromise. Paroxysmal supraventricular tachycardia (PSVT) can be treated by intravenous adenosine, vagal maneuvers like carotid sinus massage or electrical cardioversion.

Learning from examples

Example 1

27 years old, G5P1L1A2 was evaluated in cardiology department and was diagnosed to have RHD - moderate MS, mild AR, severe PAH and AF with dyspnea on exertion (functional class 3). She was seven months pregnant at the time of first evaluation. She was on lanoxin, amifru, atenolol, kaypen, and warfarin. It was noted in the case sheet that she should have BMV after 1 month, though the reason for postponement was not mentioned. As she was getting oral anticoagulants, the ideal procedure was to stop wafarin, switch over to heparin and take the patient for percutaneous balloon mitral valvotomy once the INR was less than 1.5. She was readmitted at nine months amenorrhea with a diagnosis of severe MS, severe PAH, and atrial fibrillation with controlled ventricular rate. Warfarin was stopped and heparin was started. She was advised PTMC three days after stopping warfarin. Patient developed convulsions and cardiac arrest from which she could not be resuscitated. An emergency LSCS was performed

to save the baby. A preterm asphyxiated baby was delivered with an Apgar score of 3 and handed over to pediatrician. The cause of convulsion and maternal death is not clear but could be an embolic coronary or cerebral event.

This is an unfortunate case, as this patient could have survived, had she undergone balloon valvotomy in the first admission itself at seven months. She had severe MS, pulmonary hypertension and atrial fibrillation at that time. It is not clear why a decision to defer BMV for one month was taken. A perimortem LSCS had to be done .

Learning points

- Mitral stenosis in pregnancy is associated with high risk, especially if they are in atrial fibrillation and have severe PAH and are severely symptomatic.
- As MS is an obstructive lesion, medical treatment is not helpful in severe cases and interventions like balloon mitral valvotomy or surgical valvotomy are essential to prevent development of acute pulmonary edema.
- Drugs which reduce the heart rate like betablockers and digoxin(in AF) are mandatory to improve symptomatic status.
- Balloon valvotomy can be performed at any stage of pregnancy, even at term if the patient has hemodynamic deterioration.
- Close supervision of these patients under cardiologists and obstetricians will reduce the chance for complications. Type of delivery should be decided in consultation.
- Patients with severe MS should be counselled to undergo PTMC before they become pregnant.

Example 2

This 23 year old woman, a known case of ASD Eisenmenger Syndrome, conceived after four months of marriage, and underwent medical termination of pregnancy. She again became pregnant one year later and decided to continue her pregnancy inspite of advice to terminate. She was taking sildenafil, and warfarin as per advice from a major cardiac centre. Sildenafil and warfarin were stopped at 14 weeks. She was symptomatic from first trimester and complained of exertional dyspnea and palpitation. Details regarding her further antenatal follow up is not available except the notes that she had no cardiac symptoms. She delivered vaginally by outlet forceps after induction with prostaglandin. She had no problems in the immediate post partum period and was discharged

after she was started on warfarin. She was readmitted with dyspnea and cough twenty days after discharge and was noted to be in severe cardiac failure. She gave history of low grade fever for two days. After admission she was started on antibiotics and diuretics and warfarin was continued. She had a sudden cardio-respiratory arrest at night from which she could not be revived.

This history has certain interesting points. The diagnosis of ASD Eisenmenger was made in the first pregnancy and she underwent termination. But during the second pregnancy she did not agree to termination and continued pregnancy after being aware of the risk. This patient could have been advised contraceptive methods to avoid the second pregnancy. Once she became pregnant she was willing to accept the risk. She delivered normally but within three weeks came back with severe heart failure and died. She could have been advised a prolonged hospital stay after delivery for optimising the drugs in the post partum period as mortality is common in the intra partum, and post partum period in Eisenmenger Syndrome. Her cardiac status may have become worse after a respiratory infection which could have been more aggressively treated.

Learning points

- Eisenmenger Syndrome has a high mortality of 40-45% in pregnancy. Among Eisenmenger Syndromes ASD has the worst outcome.
- Patients with Eisenmenger should be counselled against pregnancy.
- Advise contraceptive methods to prevent pregnancy.
- Risk of continuation of pregnancy to the mother and fetus should be clearly explained.
- Regular antenatal check up with obstetrician and cardiologist, with close communication between the two specialties is essential.
- Mode and time of delivery may be decided by joint evaluation.
- Anticipate problems in the intra partum and peripartum period.
- Longer hospitalisation and optimisation of medicines prior to discharge.
- Combined review after discharge may be done.
- Strong recommendation against future pregnancies.
- Drugs like sildenafil, diuretics and warfarin may be judiciously continued.

Example 3

22 yr old primigravida was diagnosed to have congenital heart disease VSD with AR, RSOV, Coronary AV fistula in childhood. Underwent cardiac catheterisation in 1998 and was taken up for AVR and patch closure of VSD in

1999. She had a mechanical prosthetic valve (Medtronic - Hall valve). She was on regular cardiac follow up and her anticoagulation was always under supervision. She had no cardiac symptoms during her pregnancy. It is noted that her husband was not aware of the seriousness of her cardiac problem. She was admitted at 34 weeks for safe confinement. During her first 3 months of pregnancy she was on heparin and then restarted on acitrome till she was admitted for delivery at 34 weeks when she was started on heparin again under supervision of cardiologists and cardiac surgeons. On 8/2/07 she had a normal delivery and her anticoagulation was optimal with heparin. She was started on oral anticoagulation and heparin was continued. On 12/2/07 she started having bleeding per vagina. Cardiologist advised reducing the dose of heparin. Next day she had more bleeding and a bout of vomiting. She then collapsed. She had cardiorespiratory arrest, and had oozing from multiple sites. The cause of death was not clear. It is unlikely that the death was due to mechanical prosthetic valve thrombosis or pulmonary embolism as she was adequately anticoagulated. It could be due to massive bleeding and DIC.

This patient's history is disturbing. She had excellent antenatal follow up. She was properly anticoagulated during pregnancy with supervision from cardiologists and cardiac surgeons. She delivered normally and had no post partum bleeding. Yet a few days later had a sudden collapse and died. No avoidable factors were identified. Prosthetic valve patients should be closely monitored in hospital even after delivery for optimising anticoagulation.

Learning points

- Prosthetic Valve patients should be informed about the treatment options during pregnancy.
- It is better to stop oral anticoagulation and switch over to heparin in the first trimester and restart oral anticoagulants after 1st trimester till term.
- Low molecular weight heparins are an alternative to unfractionated heparin.
- Close interaction between cardiologist and obstetrician is mandatory in the management of these patients.
- Patients should be counselled against repeated pregnancies.

Example 4

This 25 year old woman was diagnosed to have Tetralogy of Fallot in childhood and was taken up for intracardiac repair in 1989. She underwent a Re-do Trans annular patch in 1990 for pseudo aneurysm and again in 1992 for mycotic aneurysm. She was on ditide, and digoxin and has residual PS and PR. She had pul-

monary tuberculosis in 2004 and had antituberculous treatment for 6 months. She conceived spontaneously one year after marriage. She was evaluated by cardiologist and advised to continue pregnancy as the cardiac risk was assessed to be mild. Echocardiographic evaluation did not show any VSD. There was mild PS, PR and RV was enlarged. RV function was reported normal. RVSP was 63 mm Hg. ECG showed RBBB, and RVH. She had spotting at 13 weeks and was hospitalised for 15 days. At 26 weeks she was admitted with false labour pains. She developed respiratory infection, and was seen by pulmonologist. She had dyspnea and was evaluated by cardiologist. On 22/12/08 she had chest pain and dyspnea and she was shifted to ICU. Her dyspnea worsened and she had a cardiorespiratory collapse. Monitor showed ventricular tachycardia. She was initially cardioverted, but developed recurrent ventricular tachycardia and could not be revived.

This patient though hemodynamically stable, had RBBB. Post operative patients of Tetralogy with RBBB are considered to be at high risk for developing VT. She had a respiratory infection which could have aggravated her cardiac status. Another possibility is that of an undetected pulmonary embolism. It is always prudent to risk stratify adult congenital heart disease patients especially operated ones, into those with mild risk and those with significant risk. This group of patients are likely to increase in future as more and more previously inoperable cardiac conditions are being surgically treated in the neonatal period.

Learning Points

- Patients with congenital cardiac defects who have been operated in neonatal period and childhood will be increasing in number due to advances in surgical treatment.
- These patients need to be risk stratified as those likely to tolerate pregnancy without any undue risk and those who may have problems in pregnancy. The latter group is likely to be those who were operated for complex congenital cardiac defects.
- It is ideal for this group of patients to be assessed by the cardiologist before they become pregnant. If risk is high it may be prudent to advise against conception, or termination if pregnancy already occurred.
- Close interaction between obstetrician and cardiologist is essential in the management of these patients.

There were two cases where a definitive cause for death was not made. Both patients came with acute breathlessness and a presumptive diagnosis of ARDS versus Mitral valve disease was made. This could have been easily resolved with the availability of a portable echocardiographic machine. In one instance an echo evaluation could not be done as patient was too sick to be shifted. At least in major institutions facility for urgent bedside echocardiography should be available which may be life saving in certain situations.

Conclusions

Cardiac disease still continues as a major cause for mortality and morbidity in mothers. Though incidence of rheumatic fever is declining, rheumatic valvular heart disease still continues as a major problem .

Early diagnosis and interventions like balloon mitral valvotomy will reduce mortality and morbidity in patients with mitral stenosis. There is no specific time in which these procedures have to be done. In critical situations BMV can be done even at term.

Patients with prosthetic valves and those with operated congenital heart diseases will be presenting more and more to the obstetrician and will require multidisciplinary approach for management.

Diagnosis of pulmonary vascular disease either as pulmonary hypertension or following congenital L- R shunts place these patients in extremely risky group. It is better to counsel these patients not to become pregnant. They need intense observation not only during term but also in the post partum period at least for a month. Medical therapy with newer drugs like sildenafil can be continued under supervision.

Increased awareness of peripartum cardiomyopathy may help in earlier detection and management of this condition which is associated with a good prognosis in nearly 50% patients.

Establishment of nodal centres in each district where patients with heart disease can be evaluated by a multidisciplinary team which can give guidelines for management of less severe cases at secondary care centres should be considered.

Annexure 1

Approach to a patient with cardiac symptoms during antenatal check up

- Cardiac symptoms of importance are exertional dyspnea, palpitations, chest pain and syncope. Clinically assess the patient for features of heart failure and presence of cardiac murmurs.
- Assess the functional class of the patient and ask for history of prior cardiac disease, details of surgical or interventional treatment.
- Refer to cardiologist for assessing the severity of the disease and whether pregnancy can be continued.
- Assess maternal risk and fetal risk. Advise termination if heart disease carries high maternal mortality (PPH, Eisenmenger, severe CHF).
- Keep the patient on frequent regular check up to see any deterioration of symptoms. Refer to a higher centre for delivery if the heart disease carries high maternal mortality.
- Enlist the cooperation of supporting specialities like cardiologists, anaesthesiologists, and intensivists.
- Follow standard international protocols for management in situations like prosthetic valve patients.
- Anticipate probable complications and take prophylactic therapeutic measures.
- Plan mode and time of delivery in consultation with other specialists. Plan prolonged hospital stay in patients who are likely to deteriorate in post partum period.

Annexure 2

NYHA functional classification

- | | |
|---------|--|
| Class 1 | Patients with heart disease who have cardiac symptoms on unaccustomed activity |
| Class 2 | Patients who have cardiac symptoms on ordinary activities (climbing stairs). |

- Class 3 Patients who have symptoms on less than ordinary activities (having a bath).
- Class 4 Patients who have symptoms with any activity (eg.turning in bed). May be dyspneic at rest also.

Annexure 3

Cardiopulmonary resuscitation - recent changes

AHA/ International Liaison Committee on Resuscitation updated CPR guidelines 2010

Conventional Airway, Breathing, Compression (ABC) is changed to CAB (Compression, Airway, Breathing). The emphasis is now on effective chest compression. In children the earlier order of ABC is still followed. In order to encourage bystander assisted CPR, compression only technique has been advocated,. This is more likely to be accepted by the lay public than the conventional CPR with equal importance being given to airway. This is also called as the Cardio cerebral resuscitation. The exception to Compression only technique is drowning, CPR in children and infants

Annexure 4

Predictors of maternal Risk

- Prior cardiac events (Heart failure, Transient ischemic attack, Stroke prior to pregnancy).
- Prior arrhythmias (symptomatic tachycardia or bradycardia requiring treatment).
- Cyanotic heart disease
- NYHA Class 2 or more
- Valvular obstruction (mitral/aortic)
- Myocardial dysfunction with LVEF < 40%
- Severe pulmonary vascular disease with PAH

Annexure 5

Endocarditis prophylaxis

Patients with heart disease have been classified into those with high risk for endocarditis, moderate risk and mild risk.

The highest risk group include the following

- Mechanical prosthetic valves
- Natural heart valves from animals or cadavers
- Valve repair with prosthetic material
- Prior history of infective endocarditis
- Major congenital heart diseases especially cyanotic even if repaired

Moderate Risk

- Valve repair without prosthetic material
- Hypertrophic obstructive cardiomyopathy(HOCM)
- Acquired MR, AR

Mild Risk

- Mitral valve prolapse(MVP) with MR
- CAD
- Rheumatic fever
- Kawasakis disease.
- Pacemakers,

In those patients with mild or moderate risk, prophylactic antibiotics for prevention of infective endocarditis is not recommended. Antibiotics are optional in high risk patients. But in most situations antibiotic prophylaxis is routinely administered.

Annexure 6

Mechanical Prosthetic Valves ACC/AHA guidelines

Class 1 Continuous therapeutic anticoagulation

Replace warfarin with continuous intravenous heparin infusion, or dose adjusted heparin or dose adjusted LMWH subcutaneously between 6-12 weeks of pregnancy.

Upto 36 weeks, 3 options of continuous intravenous heparin or dose adjusted heparin or LMWH or warfarin should be discussed fully with the patients. Heparin has a higher incidence of thrombotic problems compared to warfarin.

LMWH should be administered twice daily subcutaneously to maintain anti Xa activity 0.7-1.2units/ ml 4hrs after administration.

UFH if used, ensure that APTT should be twice the control value.

If warfarin is used the target INR should be 3 (range 2.5-3.5)

Warfarin should be discontinued three weeks prior to planned delivery and continuous intravenous heparin given instead.

Class 2 a

It is reasonable to start UFH 4-6 hrs after delivery and begin warfarin in the absence of significant bleeding.

It is reasonable to give aspirin in low dose 75-100 mg daily in addition to anti coagulation with warfarin or heparin in the second and third trimester of pregnancy.

Class 3 (not indicated)

LMWH should not be given if anti X a levels are not monitored.

Dipyridamole should not be used instead of aspirin in view of its harmful effects on the fetus.

Annexure 7

Assessment of severity in Mitral stenosis

Functional class > 3. Episodes of PND, Orthopnea

Atrial fibrillation.

Long mid diastolic murmur

Presence of severe PAH, and heart failure

Mitral Valve area less than 1 cm² (Mod MS - valve area 1.0-1.5 with symptoms)

Chapter 10

Liver Diseases in Pregnancy

N. Viswanath

Key Summary Points

Nineteen patients with primary liver disease were analysed. Available data was sketchy in most and hence conclusions were often based on very shaky premises. Acute liver failure was the most common group. Main causes were AFLP, HELLP syndrome, however there were two cases each of fulminant hepatic failure due to B and A viruses and one due to the E virus and one (probably more) due to severe sepsis induced liver (and multiorgan) failure. Two patients with advanced hepatic neoplasm detected during pregnancy came for scrutiny.

Key recommendations

- All pregnant women should have the blood pressure checked and other investigations as recommended in the chapter of hypertensive disorders in pregnancy.
- In any patient with severe vomiting at any time or new onset vomiting, tiredness or epigastric pain, urine for bile may be done along with albumin.
- More detailed liver function studies, platelets and prothrombin time should be done when indicated.
- Closer monitoring of patients with pregnancy induced hypertension is essential. Upper abdominal pain, sudden change in clinical status (tiredness, vomiting, and apathy) should prompt checking transaminases, platelets, and renal function. If transaminases are raised, prothrombin time needs checking. Repeat these before any intervention such as induction or cesarean section, as changes for the worse may occur in a matter of a few hours.
- Any abnormality in the above tests has to be taken as an emergency and senior obstetricians should see the patient promptly. Decisions on referral to a higher center or calling in other specialists should be taken without delay and acted upon expeditiously.

- Referral should be with all the information that the treating unit has gathered so as to avoid delay in the new hospital.
- A team approach to management of critically ill pregnant mothers is necessary. Telephonic requests for consultations, telephonic discussions after consultation and daily (or more frequent) joint stock-taking could all bring down delays and reduce misunderstanding of instructions. A collective approach to a difficult clinical problem will be more rewarding than “passing the parcel” tradition.
- Frank honest discussions with the close members of the family and timely clear explanations in lay terms and unambiguous prognostication would reduce anxiety of the family and strain on the caregiver.
- Requirements for blood and blood components should be anticipated and blood bank should be alerted. If donor assistance is needed from family, they should be motivated and alerted in time.
- In almost all cases analyzed, the conclusions were at best guesses by experienced clinicians on the available (often sketchy) data. A greater effort has to be put in to get more autopsies (or at least postmortem needle necropsies from the suspected organs), so that we will learn from the losses. Teaching hospitals could lead the way.

Documentation

Documentation of clinical data and investigations leave a lot to be desired. Of course we are basing on the data that we receive and could be wrong. However there is tremendous scope for improvement and one hopes it would improve.

Suboptimal management

In a few cases this was apparent and shall be commented upon. However as noted in the earlier paragraph any assessor is hamstrung by sketchy data and the conclusions drawn therefore could be wrong.

Table 10.1 Patient details

No	Age	Gravida	Clinical features	Delivery	Days	Probable cause of death
1	20	G3A2	Admitted for safe confinement jaundice noted on day 2 in hospital	vaginal	6	Liver cell failure ?AFLP bleed , hypotension
2	29	G1	Jaundice noted on table when taken for LSCS. Bleed noted post op and referred. Arrived in hypotension	LSCS	1	No results of tests. Probably liver cell failure

Liver Diseases in Pregnancy

3	21	P1	Fever backache, came with IUD, delivered. PPH. High colored urine noted. GE referral.	vaginal	5	Liver cell failure, Acute hepatitis A with fulminant hepatic failure,
4	36	G3 P2	Fever, chills (4d). Pain abdomen. Leukocytosis, raised transaminases, MP, widal, dengue Ab -ve. USG edematous GB, pleural effusion.	LSCS & Emg laparotomy	3d	?Sepsis
5	27	G4P3	Admitted for ?BOH and HBsAg +vity. Abnormal LFT. Delivered 24 days after admission. Collapsed after delivery. Laparotomy showed haemoperitoneum and ?Hepatoma	vaginal	26d	? Hepatoma , probably bled into peritoneum
6	25	G1	Detected to have ?hilar cholangiocarcinoma in pregnancy. Vaginal delivery . 3 weeks postpartum died due to advanced malignancy	vaginal		Hilar cholangiocarcinoma, advanced disease
7	25	G2 P1	Twins, hypertension, proteinuria, LSCS & sterilization. PPH, renal failure with respiratory distress	LSCS	10 d	Ac liver failure ?AFLP
8	25	G1	PIH at 32 wks, Abnormal LFT, high uric acid, still birth. Renal failure, dialysis	vaginal	4d	PIH, Liver failure , renal failure ?AFLP
9	27	G1	Twin pregnancy, admitted with fever, icterus, pedal edema. LSCS, deteriorated. Dialysed on ventilator	LSCS	6 d	?AFLP , renal failure
10	24	G1	39 weeks, Jaundice, BP130/110. Platelets 90,000, LSCS, worsening liver and renal function	LSCS	4d	HELLP syndrome ,PIH
11	30	G2 P1	PIH at 6th mo, IUD, induced and delivered. Altered sensorium 1st post natal day, icterus	vaginal	42hrs	PIH, acute liver failure, ?HELLP syn , ??AFLP
12	35	G4 P3	Admitted at full dilatation. Expressed fresh stillbirth 1.2Kg. altered sensorium at admission. USG-contracted liver, ascites. Abnormal LFT.	Vaginal	33 hrs	Acute (on ?chronic) liver disease

13	22	G5 P1	36 wks, Jaundice 1 wk, abnormal LFT, altered sensorium, renal failure	vaginal	6 d	Hepatic dysfunction renal failure
14	24	G2 P0	Twin pregnancy at 34 wks, icteric with abnormal LFT, USG showed one live fetus, LSCS done. Went into ARF, dialysis, anemia. death	LSCS	9 d	Ac liver failure ?AFLP, HELLP syndrome second possibility ; ARF
15	22	G2 P1	Twin pregnancy, PIH, at 37 wks with Jaundice, ARF. Hb 5.7G%, BI urea 65 mg, S cr 2.0 mg, Bilirubin 10 mg, SGPT 134. Dialysed	LSCS	7d	Ac liv failure ,renal failure AFLP, ??HUS
16	19	P1	Fever ,vomiting for 3 d prior to presentation. Bilirubin 2.5mg, SGPT 6550u, PT test 58sec, con 13, platelets 160,000, HAV Ab IgM+ve	Vaginal	1d	Acute liver failure HAV related
17	25	G1	7wks amenorrhoea with vomiting. Jaundice . Bilirubin 10.1 mg%, SGPT 7000 u, HBs Ag doubtful, HBcore Ab positive, PT-108 sec, (control 11.9 sec)	no	42h	Fulminant livr failure Hepatitis B infection
18	26	primi	36 wks, jaundice noticed, suspecting AFLP. Delivered, severe PPH, HEV Ab IgM +ve	vaginal		HEV related hepatitis
19	28	G2P1	35wks, jaundice, HbsAg +ve, bilirubin 13.9, induced & delivered same day. Progressive worsening. Hepatorenal failure, expired on day 4	vaginal	4th	Exacerbation of chronic hepatitis B

Learning from Examples

Example 1 (patient 4)

36 year old 3rd gravida was admitted at 36 weeks with fever and chills of 4 days. Investigations showed leukocytosis, negative for malarial parasite, widal and IgM antibodies to dengue, leptospira. Ultrasonogram of abdomen was reported as acalculous cholecystitis and right sided pleural effusion. Other investigations not available except a note of abnormal LFT and RFT. Severe abdominal pain and

high grade fever continued. Acute cholecystitis/ hollow viscus perforation were suspected. Exploratory laparotomy and emergency LSCS done. Edema of gall bladder and bowel loops seen. No other pathology. Patient continued to be febrile, became tachypneic and had tachycardia. She steadily worsened and succumbed.

Learning Points

This patient probably had severe sepsis. This can occasionally progress to multi organ dysfunction syndrome. Investigations can often confuse and confound in these settings. Ultrasound examination in the setting of hepatocellular dysfunction may make the gall bladder look abnormal. Sonologists often interpret this as acalculous cholecystitis. Misleading abnormal looking gall bladders are often seen with viral hepatitis too. Pyrexia of unknown origin can be one of the most difficult problems to manage in clinical medicine. In pregnancy this could be even more so. However, a careful history, examination, systematic investigations including properly collected cultures could help in diagnosis and therapy. In this patient, with possibilities like hollow viscus perforation and cholecystitis being seriously considered, one wonders if a CT of the abdomen could have helped before emergency laparotomy. One admits that CT is often not considered in pregnancy and rightly too. However, in this patient the risk from radiation to fetus so near term could have been acceptable in such an ill patient with an unclear abdominal problem. One wonders if more aggressive antibiotics and a vaginal delivery if feasible would have made a difference in outcome.

Example 2 (patient 5)

27 year old fourth gravida (one live child), known to be HBsAg+ve, was admitted 26 days prior to her eventual demise, for bad obstetric history. She was found to be pale and had abnormal liver function tests. Hb 9.7g%, RBS 72mg%, Platelet count 3.1 lac, Bl urea 16mg, S. creatinine 0.6mg, Uric acid 7.6mg, Na+144, K+4.2mEq, bilirubin 0.7mg%, SGOT 117 IU, SGPT 441 IU, alk phos 436 IU, Protein 5.1 alb 3.0g%, HBsAg Positive. Ultrasonogram of upper abdomen was not done. She was induced near term and delivered a live 2.8kg baby. Patient went into a postpartum collapse 5 minutes after delivery. Paracentesis revealed hemoperitoneum, and patient was resuscitated. It was noted that there was a hard mass in the liver postpartum and ultrasonogram done showed a possible hepatoma. Patient was taken up for emergency laparotomy. It revealed blood in peritoneum with a grossly enlarged nodular liver and many umbilicated nodules. Patient died a few hours later.

Example 3 (patient 6)

25 year old primi, with an otherwise unremarkable medical history was detected to have “Type III Cholangiocarcinoma with Portal vein thrombosis”. She underwent chemotherapy after a normal vaginal delivery. However succumbed to advanced disease in three weeks postpartum.

Learning Points

Patients 5 and 6 had advanced neoplasms in pregnancy. Though neither had histologic proof (or autopsy study) this is the likely diagnosis. Many neoplasms can worsen faster during pregnancy. It is unlikely that anything that was done or not done during the care of these patients would have made a difference in the final outcome. However, in patient no.5, the care appears to have been rather suboptimal. The patient was in the hospital for more than three weeks prior to delivery. Liver function tests were abnormal. The hepatitis B positivity was also known. It is likely that patient must have had ultrasound examination of the pelvis during this time. However, an ultrasound examination of abdomen was not done antepartum. The reason given (that patient was unwilling for an abdominal ultrasound) by the treating unit is far from convincing. Evidence to argue for more detailed viral tests (HBeAg, HBV DNA PCR), or even a trial of nucleoside analogues (lamivudine, telbevudine) with her abnormal LFTs is very limited. However, an ultrasound examination of the abdomen might have shown the hepatic neoplasm (with possible metastasis). This information might have helped one to manage the postpartum collapse differently. We are aware that all this may not have made the final outcome different. However, one feels that the care was not satisfactory.

Neoplasm liver : The liver is not palpable during normal pregnancy. Detection of hepatomegaly on physical examination is an abnormal finding. Carcinoma metastatic to the liver can occur during pregnancy from primary lesions in the colon, pancreas, and breast. It is possible that the modest immunosuppressive state associated with pregnancy permits enhanced growth of tumors in this setting. Women with extensive tumor invasion of the liver may present with abdominal or back pain, rupture of the liver, or hepatic failure.

Rupture of liver: Hepatic rupture is a life threatening complication in pregnancy that tends to occur with many conditions, some unique, some incidental. HELLP syndrome, preeclamptic toxemia, hyperemesis are unique to pregnancy. Hepatic adenoma tends to grow more during pregnancy and may develop this complication with greater frequency. Hepatocellular carcinoma can bleed in anyone, whether pregnancy makes it more frequent is uncertain. In any case frank bleed into peritoneum is life-threatening and alternatives to surgery such as invasive radiology may need consideration whenever possible.

Example 4 (patient 3)

21 year old primi, had fever and backache 1week before confinement in the first hospital, when she was managed as urinary tract infection. Admitted at 35 weeks with intrauterine death. A dead fetus was delivered. Had vomiting after delivery. Developed hypotension and post-partum hemorrhage on 3rd day. High-colored urine noted. Tests revealed raised bilirubin and transaminases. Referred to a gastroenterologist. Hb 10.0g%, platelets 263,000, Prothrombin time 41 sec to a control of 12 sec. S bilirubin - Total 6.7mg%, direct 4.5%, indirect 2.2 mg%, SGOT 872U, SGPT 1780 U, Alk phosphatase 253 U (N 30-120), HBsAg- neg; HCV Ab- neg.

HAV Ab IgM positive. HEV Ab IgM neg, Bl urea 22 mg%. S creatinine 1.7 mg. Patient slipped into encephalopathy. Managed with antibiotics, nutritional support, component transfusions, and antihepatic coma regime. Patient terminally developed features of raised intracranial tension and expired.

Example 5 (patient 16)

19yr old primi, delivered normally at a local hospital. She had fever and vomiting for the previous 3 days. Icterus and worsening level of consciousness noted and referred. Hb11.6G, SGPT 6550U, Prothrombin time 58 sec (con 13 sec), Bilirubin T 7.4, D 3.6 mg , S. Creatinine 0.9 mg, platelets 160,000, HAV Ab IgM positive. She steadily worsened, developed seizures and died.

Example 6 (patient 17)

25year old primi at 7th wk of gestation was presumed to have hyperemesis. Investigations showed the following; bilirubin 10.1mg%, SGOT 3140U, SGPT 7000U, HBsAg doubtful, HAV HEV and HCV Ab negative, HBcore Ab IgM positive. Prothrombin time 108sec control 11.9 sec. She slipped into encephalopathy and died

Example 7 (patient 18)

26 year old primigravida, at 36 weeks came with three days history of jaundice and decreased urine output. Suspecting AFLP, a vaginal delivery was induced. She developed PPH and component transfusions given. Hb 10.8g%, TC 8000, P70, L30. Platelets 1.4 lacs, RBS 46 mg%, Prothrombin time 1 mt to a control of 13 sec. S creatinine 1.3 mg, bilirubin 12.1 mg, direct 11 mg, SGOT 46U, SGPT 44 U. Protein T 5.81, alb 2.1, globulin 3.21, Hepatitis E virus antibody IgM was positive. Patient continued to worsen and died

Learning Points

The four patients (examples 4,5,6 and 7) illustrate the typical presentation of fulminant hepatic failure due to classic hepatotropic viruses. Other viruses (herpes simplex, Epstein Barr virus infections) and drug induced hepatitis and liver failure could simulate this picture. However, differentiating from conditions where termination of pregnancy would make a difference (AFLP, HELLP syndrome) is very vital for effective management. But this may not often be easy. Severe sepsis due to any cause as well as the various zoonoses that have become prevalent in our state lately (leptospirosis, dengue especially DSS) all add to the difficulty in arriving at a diagnosis.

The presentation is with fever (often mistaken for UTI or viral fever), the prodrome is with vomiting and anorexia. Epigastric distress is not usually prominent early on. Total counts are usually normal or low, with occasionally transformed lymphocytes (virucytes) in the peripheral smear. Transaminases, particularly SGPT will be in thousands. Platelets are generally normal or minimally lowered but mostly above 100,000. After ensuring emergency care, a good history (how the illness started, what were the main symptoms and how the illness progressed) and examination crucial.

Check the earliest investigations and see how they evolved if earlier tests are available. All the conditions we discussed earlier may eventually progress to multiorgan failure which may be the time when they reach you. Differentiating the initial cardinal features of the disease from the background noise (that would develop as disease progresses) would help in identifying the primary causative agent. Targeted specialized investigations (including serology) are only an accessory. Sometimes tests can be misleading and would always need interpretation taking the clinical picture into consideration.

Management would be as for severe liver failure. Antiviral agents may have some role in only acute liver failure due to hepatitis B virus (nucleoside analogues) and herpes simplex virus (acyclovir). Care of the unconscious patient (airway, skin, chest), nutrition (hypoglycemia can be a problem), antibiotics and lactulose. Vitamin K, B and zinc may all need supplementation. One needs to avoid hypokalemia. A lot of doubtfully effective or ineffective therapies are there. Raised intracranial tension due to cerebral edema can be a problem. Avoiding saline infusions, managing high fever and cautious use of mannitol may be useful. Use of ventilator when indicated would help. Sepsis is another killer. This would need careful nursing and chest physiotherapy. Care of intravenous lines, endotracheal tubes are vital.

Generally people who slip into encephalopathy within 7 days of onset of prodromal symptoms do better than those who slip into encephalopathy later. Deeper

coma carries a worse prognosis. Benzodiazepines and other sedatives, ammonium chloride containing cough syrups and diuretics should all be avoided in patients with hepatitis and worsening hepatic function. Rather than levels of bilirubin, one should be wary of the patient with prolonged prothrombin time (more than 3 seconds over control) as this indicates poor hepatic function. (the traditional INR is more useful for titration of warfarin dose but not optimal for hepatic status). A patient who slips into hypoglycemia is likely to have less hepatocellular reserve. With optimum care some of the patients with fulminant hepatic failure (particularly those with shorter prodrome to coma time) would recover.

Available data indicates that pregnant women infected with viruses other than hepatitis E virus and herpes simplex virus follow a course that is no different from their nonpregnant sisters or males. To put it in another way, the disease does not affect pregnancy, nor does the pregnancy affect the disease. However, this conclusion is based on far from impeccable data. Hepatitis E and herpes simplex viral hepatitis do badly particularly in the third trimester. Termination of pregnancy is not believed to make any positive impact on the outlook of mother or child and is not advised for the indication of hepatitis .

[Editors'note: The first edition of this book had recommended to consider termination of pregnancy beyond 34 weeks at the early signs of liver dysfunction even if it is due to possible viral hepatitis. We still feel it relevant. Otherwise there is the possibility that the patient may go into labour at the peak of hepatic dysfunction and end up with unmanagable bleeding. This recommendation is based on clinical experiences and not on published evidence.]

Hepatitis A virus is an enterally transmitted RNA virus. The course of hepatitis A during pregnancy is generally similar to that in nonpregnant persons. The disease tends to be more severe in older patients. During severe illness in third trimester, there is increased risk of premature labor. In a review of 80,000 pregnancies, 13 cases of acute hepatitis A during 2nd and 3rd trimester were noted. 69 % of these developed gestational complications including premature contractions, placental separation, premature rupture of membranes, and vaginal bleeding. Eight patients developed preterm labor at a median of 34 weeks. Despite these occurrences, all children had favourable outcomes. Perinatal transmission was not noted in this group. No increased risk of fulminant hepatic failure noted in Hepatitis A.

Hepatitis E virus is an enterally transmitted acute viral hepatitis, caused by an RNA virus. It is similar to Hepatitis A virus infection in many ways. It is water-borne, but person to person spread is much less common than hepatitis A virus. Epidemiologic data on HEV in Kerala is lacking. However, clinicians observe that HEV infection is uncommon in Kerala, and most of the cases are visitors from or

recent travellers to endemic areas (Indo-Gangetic plains or other areas in the North of India). Clinical course is similar to HAV infection in the nonpregnant person. However, for reasons that are not understood, fulminant hepatic failure occurs more frequently during pregnancy resulting in an inordinately high mortality rate of 15-25% particularly in the third trimester. Increased viral replication has been documented in pregnancy. Data regarding fetal risk is sketchy. One report evaluated eight babies born to mothers with HEV infection in the third trimester. Six infants had clinical, serologic or virologic evidence of HEV infection. Two infants died within 24 hours of birth, one of whom had massive hepatic necrosis at autopsy. Understandably worse obstetric and fetal outcome is noted with HEV infection. Vaccine is in the later phase of development.

Summing up

Though the available data may not be representative, it looks as if hepatic disorders unique to pregnancy (AFLP, HELLP syndrome, hepatic disorder with PET) are a significant fraction of those causing maternal and fetal death. Identifying these early, and effectively treating them would affect the poor outcome we have in managing these conditions. As highlighted in the earlier issue, good antenatal care, early diagnosis with a high index of suspicion, aggressive management, well coordinated team effort by concerned specialists, would go a long way in getting the mortality down. Since this was dealt with in detail in the last volume, a review was avoided. However as the table at the beginning highlights these are big concerns still. Our aim should be to arrive at correct diagnosis early and manage effectively reducing avoidable delays. This time the focus was more on viral hepatitis.

The hepatitis A and E related deaths draw attention to the following issues.

- We still do not have clean water supply to all. Our environment is deteriorating. Infectious diseases appear to be on the rise.
- Should we consider screening our 15 year olds for HAV Ab IgG, and vaccinate those who are negative. This can be expensive.
- If HEV vaccine becomes available, vaccination may need serious consideration in Indo-Gangetic plains(especially the young girls).
- Hepatitis B vaccine can be administered in early pregnancy to those who are Anti HBs negative (being a vaccine containing part of surface antigen, it can be safely given in pregnancy).

HELLP Syndrome

Definition

Haemolysis, elevated liver enzymes, low platelets

Epidemiology

- Occurs in 0.2 to 0.6 % of pregnancies.
- In 10-20 % patients with severe pre eclampsia / eclampsia.
- Majority of cases between 28-36 weeks.
- In 70% prior to delivery (80% of these before 37 weeks, less than 3% between 17-20 weeks of gestation).

Disease presented postpartum in 30% patients, usually within 48 hours, rarely as late as 7 days postpartum (only 20% had pre eclampsia antepartum)

Etiology

Unknown. Abnormal vascular tone, vasospasm, coagulation may have a role in pathogenesis.

Symptoms and signs

Abdominal pain, tenderness, nausea, vomiting, malaise
Jaundice

Hypertension (BP= \geq 140/90) and proteinuria in approximately 85% pts.

Other signs include pulmonary edema (6%), ascites (8%) and acute renal failure, usually occurring in the setting of disseminated intravascular coagulation (20%).

Diagnostic criteria used are variable and inconsistent

Hemolysis; defined as presence of microangiopathic haemolytic anemia, indicated by abnormal peripheral smear (schistocytes, burr cells, echinocytes), elevated indirect bilirubin, low serum haptoglobin levels, elevated lactic dehydrogenase (LDH) levels and a significant drop in hemoglobin levels.

Elevated Liver enzymes; Serum AST (SGOT) >70 IU/ L

Low Platelets ; $<100,000$ / microlitre.

Patients may present with only two of the above. The total clinical setting is important in arriving at a diagnosis.

Investigations

Complete blood count with platelet count, Peripheral smear

Liver function tests: SGOT, bilirubin, LDH.

Imaging of the liver, ultrasonogram, CT or MRI needed sometimes.

Obstetric management

- Assessment of fetal and maternal status, intervention to stabilize when needed.
- Delivery is indicated for pregnancies ≥ 34 weeks gestation; nonreassuring tests of fetal status (eg. Fetal heart testing, biophysical profile) or presence of severe maternal disease; multiorgan dysfunction, disseminated intravascular coagulation (DIC), liver infarction or hemorrhage, renal failure, or abruptio placentae.
- Normal vaginal delivery is possible in most patients following induction. Cesarean section can be considered in very preterm gestations (under 30 weeks) when cervix is unfavourable.
- Magnesium sulphate should be considered to prevent convulsions. It may be stopped 24 hours postpartum or when remission is noted, whichever is later.

Control of hypertension

Expectant management of stable, preterm pregnancies should be considered investigational.

Table 10.2 Clinical features of AFLP and HELLP ; a comparison

	AFLP	HELLP
Glucose	decreased	normal
Creatinine	increased	Normal
Uric acid	increased	Increased
Bilirubin	^(conjugated)	^(occ unconjugated)
LDH	Normal	Increased
SGOT/SGPT	Increased	Increased
Platelets	Normal	Decreased
PTT	Prolonged	Normal
PT	Prolonged	Normal
Fibrinogen	Decreased	Normal
Histology	Microvesicular fatty infiltration	Periportal hemorrhage
Mortality (western)		
Maternal	Around 18%	About 2%
Fetal	About 24%	About 32%

Acute fatty liver of Pregnancy

Epidemiology

Prevalence 1 in 7000 to 1 in 16000 deliveries

Usually after 35 wks, even as early as 26 wks, occasionally immediate postpartum

Risk factors

Primiparity, Multiple gestation, Male fetus

Clinical presentation

Early symptoms

- Nausea and vomiting (70%)
- Epigastric or RUQ pain (60-80%)
- Malaise , anorexia
- Jaundice may be seen in 1 to 2 weeks

Late symptoms

- Fulminant liver failure
- Encephalopathy
- Renal dysfunction
- Gastrointestinal and uterine bleeding
- Pancreatitis
- Seizures
- Disseminated intravascular coagulation
- Coexistent pre eclampsia in 50% patients

Etiology;

Association between defects of fatty-acid oxidation in fetus and AFLP in mother is well reported. Toxic effects of abnormal levels of fetal long-chain fatty acids on maternal system believed to play a role in pathogenesis.

As many as 70% of cases in some studies were due to homozygous long-chain 3-hydroxyacyl coenzyme dehydrogenase (LCHAD) deficiency in fetus, with a heterozygous mother.

The best characterized mutation is a single guanosine to cytosine mutation (G1528C) in the alpha subunit of the trifunctional protein (TFP) of which LCHAD is a part.

Diagnosis.

Clinical, based on history, physical findings and laboratory investigations. Ultrasound scan may show hyperechoic liver. CT which shows decreased attenuation is more reliable than ultrasound scan, but has radiation risk. Definitive diagnosis by liver biopsy and oil red O staining and electron microscopy.

Treatment

Non pharmacologic therapy

Patient in ICU for stabilization and monitoring

Fetus is delivered; spontaneous resolution follows delivery

Mode of delivery depends on obstetric indications and clinical assessment of severity.

General management

- Decrease in endogenous ammonia through dietary restriction of proteins, oral metronidazole to decrease ammonia producing bacteria, lactulose to evacuate colonic contents.
- Intravenous glucose to maintain plasma glucose levels above 60 mg/dl as hypoglycemia is a killer in AFLP.
- Coagulopathy corrected with FFP
- Avoidance or careful use of drugs metabolized by liver.
- Avoidance and treatment of nosocomial infections.

Suggested reading

1. JR Barton, BM Sibai. Diagnosis and management of haemolysis, elevated liver enzymes, and low platelets syndrome. *Clinics in perinatology* 31 (2004) 807-833
2. FV Schiodt, WM Lee; Fulminant liver disease. *Clinics in liver disease* 7 (2003) 331-349
3. S Doshi, SD Zucker; Liver emergencies during pregnancy. *Gastroenterology clinics of North America* 32 (2003) 1213-1227
4. Handbook of liver disease (second ed) 2004 , Ed. LS Friedman, EB Keeffe

Chapter 11

Anesthetic Causes

A.K.Unnikrishnan

[*Editors note:* The key summary points, introductory remarks and key recommendations from the chapter of Anesthetic causes by Dr. A.K Unnikrishnan in the first edition of “Why Mothers Die Kerala – 2004-05” is reproduced here as we feel that they will be complementary to the present write up. Readers are requested to refer to first edition for further details including annexure on management of acute collapse and contents of emergency trolley.]

Anesthetic concerns in Obstetric Patients

Key Summary Points

- Obstetric anesthesia is more challenging than anesthesia for other surgical patients. Anatomical and physiological changes and serious obstetric complications of pregnancy are responsible for this.
- While anesthesia and surgery should be avoided if possible during period of organogenesis, concern of teratogenesis are unfounded with commonly used anesthetic agents.
- If there are no specific contraindications, regional anesthesia is preferred over general anesthesia.
- The preferred mode of labour analgesia is epidural.
- Electronic monitors are very useful; they cannot be a substitute for the anesthesiologist at the head end of the patient.
- Properly kept intraoperative records will be the best defense, should a medicolegal case arise.

The obstetric patient usually presents more challenges to the anesthesiologist than any other surgical patient. Fundamental changes occur in maternal anatomy and physiology during pregnancy due to:

- Altered hormonal activity
- Increased metabolic demands of a growing uterus, fetus and placenta

- Mechanical displacement of viscera produced by an enlarged uterus

While most of these changes confer obvious adaptive advantages for the gestation and the puerperium, some of the alterations may have a potentially adverse influence on the anesthetic administration during childbirth and for non-obstetric procedures performed during pregnancy. These include:

- Mechanical pressure by the enlarged uterus on the large vessels, the aorta and the inferior vena cava, with the potential to produce the dreaded complication of *aorto-caval occlusion syndrome*.
- Increase in intragastric pressure coupled with an incompetent lower esophageal sphincter and increased gastric acidity precipitating another near fatal complication, acid aspiration syndrome (*Mendelson's syndrome*).
- Upward displacement of diaphragm resulting in decreased functional residual capacity (FRC) which predisposes the pregnant patient to develop hypoxemia easily and thus compromises both the mother and the fetus.
- The potential for adverse effect on the fetus is when anesthetic intervention is required especially during early pregnancy when organogenesis occurs.

Thus, in many respects, the obstetric patient is unique compared to other surgical patients and demands specialized attention from the anesthesiologist.

Non-Obstetric Surgery during Pregnancy

Doctors are often confronted with concerned patients and their relatives when a non-obstetric surgery is found necessary in a pregnant patient. The questions often asked are:

1. Whether anesthesia will result in congenital defects in the fetus.
2. What is the probability of abortion/ preterm labour in such a situation.

Teratogenicity:

Since prospective clinical trials in humans are not possible, studies regarding the teratogenic potential of anesthetic drugs were done in lower mammals which showed that very high doses of certain agents (for example, nitrous oxide) can cause malformation in offspring. But these results cannot be extrapolated to the human condition. Large retrospective outcome studies in human populations comparing pregnant patients exposed to anesthetics with those not exposed, have not shown any difference in the incidence of congenital malformations in the fetus. Maternal hypotension, hypoxia, hypocarbia, etc., can however adversely affect the fetus. It is prudent, if possible, to avoid anesthetics and surgery during

pregnancy, especially during the period of organogenesis. With the current state of knowledge, it can be categorically stated that no anesthetic or drug routinely used during anesthesia can be implicated to be teratogenic.

However, their use should be confined to areas where a good support system in the form of trained personnel and necessary infrastructure is available. Despite its obvious advantages, the use of labour analgesia has not become widespread in India except in major cities and among the higher strata of society.

Key Recommendations

- Since obstetric anesthesia is more hazardous, it should not be relegated to juniors or trainees to be handled independently.
- Steps to prevent Mendelson's syndrome should be taken routinely even if regional anesthesia is planned.
- If possible avoid anesthesia during period of organogenesis even though no teratogenic effect has been reported with commonly used anesthetic agents.
- Prefer regional anesthesia rather than general anesthesia unless there is a compelling reason to choose the latter.
- Even if electronic monitoring devices are available, the anesthesiologist should stay near the patient.
- For intraoperative monitoring, a graphic record should be used in every patient.
- Anesthetist's service should be utilized for vascular access, fluid management etc in critically ill patients.

In the period, 2006 – 2009, out of the total cases of reported maternal deaths, eight deaths could be directly or indirectly attributed to anesthetic causes. In spite of advances in anesthetic practices with improved monitoring, anesthesia still remains one of the causes of maternal morbidity and mortality. We sincerely feel that there is definite scope for improvement in this area. This exercise is aimed in that direction. Here we will review the 8 cases and try to discuss what probably happened and how we can possibly rectify the mistakes, if any.

Example 1

30 year old lady, G3, P2, L1 (1st LSCS, 2nd IUD) was taken up for cesarean section. LSCS done under general anesthesia(GA)since the patient refused spinal. Intraoperative period was uneventful except for slight increase in uterine bleed

which was controlled with IM carboprost. At the end of surgery there was delay in recovery and then patient developed severe pulmonary edema and hypoxemia. She subsequently developed hypoxic encephalopathy and in spite of all efforts, expired after 48 hours.

Learning points:

From the sequence of events, we can only presume that some serious untoward incident viz. AFE, Mendelson's syndrome or other hypoxic event has occurred in the intraoperative period leading to pulmonary edema and failure to regain consciousness. We do not know whether minimum monitoring standard was adhered to in this case as the record is silent in this respect. We can only say that if prompt recognition of a problem and timely intervention was done, may be the patient could have been saved. About the choice of anesthesia, it is now well recognized that in operative obstetrics, best fetal and maternal outcomes are obtained with regional especially spinal. We must educate our patients in this aspect.

Example 2

26 year old lady G4P3, LCB 3yrs. sterilized after third delivery, admitted with abdominal pain. Ultrasonogram suggested ectopic pregnancy and she was taken up for emergency laparotomy. Due to inadequate analgesia, spinal was converted to balanced anesthesia. Intraoperative period uneventful. Tubal abortion was noted. Blood loss was only 100 ml. During reversal of neuromuscular block, severe bradycardia occurred followed by cardiac arrest. Routine CPR instituted but return of spontaneous circulation established only after 12-15mts. Patient remained unresponsive and continued to show signs of hypoxic encephalopathy and expired after one week.

Learning points

The timing of the severe bradycardia and subsequent cardiac arrest would point to the role of neostigmine used to reverse the neuromuscular block. Neostigmine has powerful cholinergic effects and in spite of addition of atropine, can rarely produce severe bradycardia. The failure of prompt return of spontaneous circulation (it took 12-15 mts) would suggest some delay in recognition and management of the problem. This obviously resulted in the subsequent development of hypoxic encephalopathy and death. Anticipation of such sudden events and prompt institution of resuscitative measures can save the patient.

Example 3

33 yr. old lady G2, P1, first was a term normal delivery. Referred to higher centre with uncontrolled PIH and GDM. She was taken up for LSCS under general anesthesia. A severely asphyxiated baby was delivered. The records do not mention about any untoward intraoperative events. During recovery she developed severe pulmonary edema and bradycardia. Resuscitated in the operation theatre and shifted to ICU where she developed repeated episodes of pulmonary edema. In spite of continued support, she expired after 3 days.

Learning points:

This obviously was a poor risk (Gr III ASA) patient. Poor control of PIH and GDM could have contributed to the over all poor outcome. However we cannot rule out some mishap occurring during the course of anesthesia. Intraoperative monitoring details are not available. These types of poor risk patients warrant extreme care in their management.

Example 4

23 year old lady, G3, P1, A1. First LSCS for CPD. Posted for elective cesarean. Spinal anesthesia failed, hence GA with balanced technique administered. Intraoperative period was uneventful. Neuromuscular block reversed with neostigmine and patient was extubated. Soon after, patient developed 'twitching' of limbs and SpO₂ started falling. Patient was reintubated and ventilated and seizure control measures were instituted. Immediately patient was shifted to a higher centre in an ambulance with AMBU ventilation. Patient developed cardiac arrest on reaching the higher centre and could not be resuscitated.

Learning points:

This patient obviously developed hypoxic brain damage as evidenced by the failure to regain consciousness and the seizure activity. One possibility is severe AFE for which very little except supportive measures can be done. Another possibility in this case is inadequate reversal from neuromuscular block. This will be evidenced by fidgety movements of limbs and SpO₂ will fall because of inadequate ventilation. Management of this condition is to continue ventilation and give more neostigmine. From the available records it is difficult to pinpoint the exact cause of the mishap. Inadequate reversal is an eminently manageable condition.

Example 5

24 year old primi admitted for safe confinement. She developed pyrexia and labour was induced. There was lack of progress and fetal distress was developing and hence decided on cesarean section. LSCS was done under spinal anesthesia. Soon after delivery, the patient developed severe hypotension followed by cardiac arrest. Resuscitated and spontaneous cardiac activity resumed. Patient was shifted to ICU. After 3 hours she developed another arrest from which she was again revived. Shifted to higher centre where she died.

Learning points:

The record provided is very sketchy. From the timing of the occurrence of cardiac arrest, the most probable cause of initial cardiovascular collapse is AFE for which only supportive measures are possible. Another possibility is severe hypotension from sub arachnoid block(SAB) which probably was not detected in time and/or was not managed aggressively. What type of monitoring was used in this case is not known. Absence of any anesthesia notes in the record is surprising. Failure to keep proper record will amount to negligence.

Example 6

24 yr. old primi, admitted at term, developed pyrexia. LSCS done under sub-arachnoid block. Soon after spinal, patient developed cardiorespiratory arrest. Resuscitated with routine measures and surgery proceeded. Delivered a deeply asphyxiated baby. CPR measures continued and surgery completed. After 3 hours she had another arrest from which she could not be revived.

Learning points:

Acute cardiovascular collapse immediately following a spinal is due to Aorto-Caval Occlusion Syndrome. This can manifest so dramatically that unless we are aware of this possibility and act appropriately, the outcome will be disastrous as has happened in this case. Putting a wedge can reduce the incidence but cannot completely prevent it. The correct management is to either *manually lift the uterus from the back (if surgery has started) or to turn the patient to the left and then give vasopressor*. There is no point in giving vasopressor if the uterus is compressing the inferior vena cava. Prompt, timely intervention only can save the patient. Spinal anesthesia (or for that matter any anesthesia) in a full term parturient is serious business and should be treated as such!

Example 7

24 yr. old lady G2, P1 first LSCS admitted with a report of IUD at 16 weeks. Labour induced with misoprost for 4 days followed by oxytocin. She was posted

for evacuation under anesthesia. Anesthetic sequence was pentazocin /promethazine / midazolam/ ketamine i.v with 100% oxygen using Bain's circuit. Towards the end of the procedure patient developed some respiratory problem followed by fall in SpO₂ and severe bradycardia. Immediately intubated ventilated and bradycardia corrected with atropine. But soon after she developed cardiac arrest. Routine CPR measures instituted. Cardiac rhythm returned but severe dysrhythmias appeared. She showed signs of DIC and MODS and was shifted to a higher centre where resuscitative measures were continued. She had hypoxic encephalopathy, DIC and serious cardiac dysrhythmias and ultimately died of MODS

Learning points:

Though the ultimate cause of death can be attributed to MODS the whole sequence of events was initiated by some intraoperative mishap. The possibility of acid aspiration in a 16 week pregnant patient was not considered and no acid aspiration prophylaxis was given. During GA in this patient, the air way should have been protected with a cuffed endotracheal tube. Failure on these two accounts could have initiated the whole unfortunate episode. A proper endotracheal balanced anesthesia, I feel, would have been a better choice for this case rather than the one which was employed.

Example 8

27 yr. old lady G2P1, first FTND. Admitted for safe confinement. At term labour induced. Reassessment revealed CPD and decided on cesarean section. LSCS done under spinal anesthesia. A healthy baby was delivered. Soon after delivery of the baby, she developed cardiac arrest. Resuscitated with routine measures and surgery completed. As the patient's condition was not satisfactory she was shifted to higher centre but on the way she died.

Learning points:

The most probable cause of this tragedy is AFE which in severe cases is invariably fatal. All that can be done is supportive measures. It was quite unwarranted to shift a very unstable patient to a distant place. The available record of the case is poorly written and will point to a suspicion of suboptimal management of a life-threatening complication.

Observations & Recommendations

1. Anesthesia still contributes, in a small way, to maternal mortality. The fact that obstetric anesthesia carries higher risk is often ignored. Obstetric anesthesia, ideally, should be handled by experienced people.
2. Acid aspiration prophylaxis must be used in pregnancy from 6 weeks onwards. Other aspiration prophylaxis like Rapid Sequence Induction must be employed in such patients.
3. Minimum Monitoring Standard (HR, ECG, SpO₂, NIBP) must be followed in all anesthetized patients. Failure to do this can land us in trouble and will amount to negligence.
4. Regional anesthesia (Spinal & Epidural) is associated with fatal complications in obstetrics and should be handled with due caution.
5. Patient records continue to be kept in a haphazard way. Often this can lead to suspicion whether proper care was given to the patient.

Chapter 12

Venous thromboembolism in pregnancy

B. Presannakumari, P K Syamala Devi

Venous thromboembolism(VTE) is a leading cause of maternal mortality. The incidence reported is 0.05-0.3 %.The incidence of fatal thromboembolism is on the decrease in developed countries. In the reports of previous CEMD in UK, of the 46 deaths from pulmonary embolism, 15 occurred antepartum and 25 occurred postpartum, 3 occurred in early pregnancy. In Kerala, confidential review of maternal deaths for the years 2004 and 2005, showed 7 cases of pulmonary embolism out of 170 cases analysed. During the years from 2006 to 2009, out of the 331 deaths reported to CRMD, 9 were presumed to be due to thromboembolism (2.6% of the total maternal deaths reported.) All the 9 deaths reported occurred postpartum. Out of the nine deaths reported, seven occurred following cesarean section, one following an abortion at 22 weeks of gestation and one followed a preterm delivery. This is in accordance with CRMD committee's perception that cesarean section increases the chance of VTE as compared to vaginal delivery.

Factors which make pregnancy a high risk condition for Venous thromboembolism

A delicate balance exists between procoagulant and anticoagulant factors in the body. However the balance tilts in favour of coagulation during pregnancy and puerperium and pregnancy per se is an independent risk factor for thromboembolism.

The incidence of DVT is five times higher in pregnancy than in age matched non pregnant women. Pregnancy and puerperium predispose to Virchow's triad of hypercoagulability, stasis and endothelial injury.

- 1. Hypercoagulability** There is elevation of coagulation factors V, VIII, X, and VWF Ag. Fibrinolytic activity is impaired during pregnancy, relating in part to elevation of placentally derived PAI -2 (plasminogen activator inhibitor). Reduced levels of natural anticoagulant protein S contribute to the state of hypercoagulability during pregnancy. Similarly about 38% of

pregnant women develop activated protein C resistance by third trimester.

The changes which make pregnancy a hypercoagulable state occur even as early as first trimester. These changes are part of the natural protection for the pregnant woman from exsanguination following abortion or delivery.

2. **Venous stasis** occurs in the lower limbs as a result of pressure of the gravid uterus on the inferior vena cava, decreased venous tone and valvular incompetence of the venous system. There is decreased flow velocity evident in early second trimester, nadir by 36 weeks, takes 6 weeks following delivery to return. Majority of DVT (90%) occurs in the left side and that too in the ileofemoral segment (70%).
3. **Endothelial injury**, Although pregnancy itself is not associated with endothelial injury, passage of fetal head through the birth canal and operative vaginal delivery including forceps and vacuum exaggerates vascular intimal trauma and trauma to pelvic veins which accounts for increased risk of VTE in the immediate post partum period.

In addition to physiological changes in the hemostatic system during pregnancy, other risk factors of VTE include cesarean section, obesity (>80 Kg), high parity (four or more), infection, pre-eclampsia, immobility, dehydration etc. Family history of VTE may also be a risk factor, since it may point to a possible underlying thrombophilia.

Inherited thrombophilia

Deficiencies of the naturally occurring anticoagulants, antithrombin, protein C and protein S can be inherited but are rare. However, protein C deficiency is reported in 1/500 healthy individuals in the UK.

Deficiencies of antithrombin, protein C and protein S are inherited in an autosomal dominant pattern, such that offspring of an affected parent has 50% chance of inheritance.

The anticoagulant activity of protein C operates by inactivation of factor V. But, when there is a single point mutation in the gene encoding factor V (Factor V Leiden mutation) the inactivation does not happen effectively and there is higher risk of VTE. The excess risk of VTE appears to be 5-10 fold in those heterozygous for mutation and approximately 80 fold in homozygotes.

Prothrombin gene mutation results in elevated mean plasma prothrombin levels. It is reported in 1.2 to 2.6% of the healthy population in the United Kingdom. This increases the risk of VTE by 2- 4 fold.

We are not aware of the prevalence of the various inherited thrombophilias in the Indian population

Hyperhomocysteinemia

Elevation of plasma homocysteine appears to confer an increased risk of venous and arterial thrombosis. Homocysteine metabolism normally occurs in 2 pathways: remethylation to methionine and trans-sulfuration to cysteine. A mutation in methylene tetra hydrofolate reductase (MTHFR) enzyme which is essential for remethylation pathway has been described by Frosst et al which is associated with a thermolabile enzyme with reduced activity. Reduced MTHFR activity can lead to accumulation of homocysteine in the circulation. However the ill effects of hyperhomocysteinemia can be countered by administration of folate.

Risk of VTE in asymptomatic carriers of inherited thrombophilia

All women with inherited thrombophilia need not be anticoagulated if they do not have a personal history of thrombophilia or have a poor pregnancy outcome. However, because of high risk for thrombosis among those who are homozygous for factor V Leiden mutation and prothrombin gene mutation or those with anti-thrombin deficiency, such patients need anticoagulation throughout pregnancy and even puerperium. Any patient who needs thromboprophylaxis in pregnancy will need the same during puerperium also.

Who should undergo thrombophilia screening?

Routine thrombophilia screening should be avoided. Women with a personal or family h/o VTE should undergo thrombophilia investigation, ideally before pregnancy to allow time to plan pregnancy management. Ideally counselling of women with thrombophilia must be done at a thrombophilia clinic where there is a hematologist and an obstetrician. Women with recurrent (three or more) early or late pregnancy losses should also be screened and counselled.

Thrombophilia screening should include coagulation screen, full blood count, antithrombin and protein C activities, total free protein S Ag, lupus anticoagulant test, anticardiolipin antibodies, and determination of factor V and prothrombin gene status.

Diagnosis of DVT

Clinical features are not reliable for the diagnosis of DVT. Edema leg and pain can occur in pregnancy in the absence of DVT. Doppler screening is a noninvasive investigation to diagnose DVT with high sensitivity in suspected patients. Hence

it should not be deferred in patients with suspected DVT. Once diagnosed, such patients should get heparin treatment.

About the women who died, Summary of Findings

There were 9 deaths reported to be due to pulmonary embolism during the year 2006-2009. Of these seven followed cesarean section. All were emergency cesarean sections. This emphasizes that the highest risk period for venous thromboembolism and pulmonary embolism in particular is during the postpartum period. Risk Factors for VTE could not be identified from the case records The BMI should be entered at booking visit. More attention is needed to elicit proper family history and advise investigations and treatment if indicated.

Table 12.1 Details of patients

Sl no:	Age	Para	Gestation	Mode of delivery	Intvl diag to death	Intvl delivery to death	Risk factors	Complications	Mode of diagnosis & investigations
1	39	P1	POD 16	LSCS	19 days	32 days	HELLP Kyphoscoliosis, Emergency CS	GDM,PIH, Partial HELLP	ECG changes, ?PE
2	29	P2	17hrs Postop	LSCS	11 hrs	28 hrs	Em LSCS	Previous CS	ECG changes ?PE
3	22	P1	POD 4	LSCS	13 hrs	5 days	Emergency LSCS	NA	Not done ?PE
4	26	P2	36 wks	Preterm del	5 days	7 days	NA	NA	ECG changes, ?PE Echo
5	19	P1	POD 2	LSCS	24hrs	3 days	Em.LSCS	NA	D-Dimer ?PE
6	24	P2		LSCS	2 hrs	30 hrs	Em.LSCS PIH, wt 89 kg	Previous CS, PIH	?PE
7	21	P1		LSCS	1 day	3 days	Em. LSCS	int.hemorrhage	?PE
8	30	P1	PostAb D 6	Abortion (PGE1)		6 days	Prolonged immobilisation	Guillian Barre syndrome	?PE
9	34	P2	POD 5	LSCS		5 days	Type 3 pl.previa, PE, bed rest, BMI raised	Previous CS, Type 3 pl previa	PIH,FDP Raised D-Dimer ? PE

POD-postoperative day/post delivery day; PE -pulmonary embolism; FDP-Fibrin degradation product

Learning from examples

Example 1

A 34 year old, G2P1L1, previous CS, Placenta previa (Type III anterior) who had emergency LSCS at 35 weeks developed severe breathlessness on 5th postoperative day. She had a weight of 82 kg at 16 weeks, major degree anterior placenta previa, fibroid complicating pregnancy and severe pre eclampsia. The clinical picture was that of massive pulmonary embolism. She died a few hours later.

Learning points

1. This patient did not receive thromboprophylaxis, even though there were clear indications for advising thromboprophylaxis - Obesity, pre eclampsia, probably bed rest prior to surgery due to the diagnosis of placenta previa and finally cesarean section.
2. She was discharged and readmitted on day 5. The treating team did not seem to have suspected pulmonary embolism as a possibility.

Example - 2

A 30 year old, G2 P1 L1, Previous CS, was admitted at 22 wks gestation with progressive weakness of both lower limbs. She was diagnosed to have Guillian Barre syndrome and was treated with plasmapheresis and FFP. Abortion was induced for intrauterine demise with PGE1 and she expelled the fetus following which her retained placental bits were removed piecemeal with sponge holding forceps.

She developed edema and pain in her right lower limb on the 6th day after expulsion of products. She was then started on heparin and antibiotics with a provisional diagnosis of DVT. Doppler study of limbs was advised but not done. From the case sheet it is not sure whether the therapeutic dose of heparin was given or not. The same day, she developed symptoms suggestive of massive pulmonary embolism and succumbed to it. Pulmonary embolism was not confirmed.

Learning points

This patient who had Guillian Barre syndrome is prone for DVT as she had weakness of lower limbs and immobilization. She was started on heparin only when she developed symptoms of DVT. If she had been started on heparin following her abortion in view of the fact that she is immobilized due to Guillian Barre syndrome, deep vein thrombosis and pulmonary embolism probably might have been prevented.

Example 3

21 year old Primigravida had emergency caesarean section for failure to progress. From the records available no risk factors other than emergency cesarean were identified. On the third day she had sudden onset of dyspnoea. She had hypotension and tachycardia and was referred to a higher centre. Internal hemorrhage was excluded by ultrasound. She was seen in the higher centre by cardiologist. Her D dimer was found to be high. She died of hypotension and cardiac arrest about 12 hours later.

Learning points

She could have been started on heparin, with a provisional diagnosis of PE. Even though there was enough time to investigate for diagnosing or excluding pulmonary embolism, it was not done. If facilities were available emergency embolectomy might have saved the life of the patient.

Key Recommendations

Venous thromboembolism risk ideally should be assessed at the time when pregnancy is diagnosed or even before, in a pre pregnancy clinic. Family history suggestive of VTE like young stroke or previous bad obstetric history of repeated abortions, early onset pre-eclampsia and/IUGR should be elicited during this assessment. This risk assessment should be done at every antenatal visit and when the patient gets into labour, irrespective of the route of delivery thromboprophylaxis is to be instituted appropriately.

Risk factors - See CRMD Recommendations

Recommendations

1. Obesity is a major risk factor for thrombosis. It is ideal to plan pregnancy after achieving ideal weight. BMI should be entered in the antenatal record at the first visit.
2. Negative D- dimer helps to rule out pulmonary embolism. When suspicious symptoms of DVT are noted, doppler studies and if necessary MRI should be employed. D dimer should not be used for diagnosing DVT.
3. When symptoms suggestive of pulmonary embolism are present ventilation perfusion scan, spiral CT scan etc should be employed. If confirmed, consider embolectomy.

4. If acute DVT or PE is diagnosed in pregnancy, the woman should receive parenteral heparin for five days along with warfarin. Intravenous heparin should be continued until PT/INR goes above two. Those who develop massive pulmonary embolism in the puerperium should be considered for thrombolytic therapy.
5. Because of high risk for thrombosis among those who are homozygous for factor V Leiden mutation, prothrombin gene mutation or antithrombin deficiency they need anticoagulation throughout pregnancy and puerperium.
6. Unindicated rest in pregnancy should not be encouraged.
7. Early ambulation and proper hydration are essential during puerperal period.
8. Elastic compression stockings should be advised for women who have large varicose veins. Pneumatic compression stockings can be used for women who are at high risk for thrombosis during cesarean.
9. Thromboprophylaxis with heparin, especially LMWH has not been found to increase the risk of hemorrhagic complications in pregnancy. Hence, it must be a practice to use heparin in pregnancy and puerperium boldly, when indicated.

[Editors' Note: We are concerned about the rising incidence of venous thromboembolism especially pulmonary embolism and would like wider use of thromboprophylaxis. However, the western recommendations do not seem to be appropriate considering its implications for day to day practice. Hence we still stick to our earlier recommendations published in the first report (2004 –'05) which is reproduced here.]

CRMD Recommendations

On the basis of the cases studied it becomes difficult to identify risk factors and make recommendations for prophylactic medication. Nor can we copy western recommendations for prophylaxis without modifications. The general feeling is that the differences in lifestyle, genetic factors for thrombophilias, food habits etc have to be considered before we adopt the western recommendations.

The general perception among obstetricians is that incidence of cerebral venous thrombosis has come down but pulmonary embolism is on the increase. Hence, we feel that thromboprophylaxis should be incorporated into our day to day practice. However, due to the absence of any hard evidence related to our population, we have been forced to make the following recommendations on the basis of the experience of the clinicians (obstetric and nonobstetric) involved in the assessment.

1. Early ambulation

Contrary to conventional practice in Kerala (indigenous medicine) we have to encourage early ambulation, after vaginal delivery as well as cesarean. Reducing episiotomy rate and using transverse incision for cesarean will help in this. When they are reluctant to move out of bed, lower limb exercises should be encouraged.

2. Early and adequate fluid intake

There seems to be a wide spread perception that drinking fluids during post partum period will inhibit the involution of the abdomen and the uterus. Hence, postpartum patients are reluctant to take fluids. This is all the more harmful because in early puerperium there is physiological diuresis. We should insist on adequate fluid intake during postpartum period.

3. Use of elastic compression stockings

These are known to reduce venous pooling and will be of special use in women with large varicose veins.

4. Change in obstetrician's concept about bed rest. It is quite common practice to enforce absolute bed rest for patients who had threatened abortion, cervical cerclage or past history of recurrent pregnancy loss. The worst scenario is seen after cervical cerclage. Patients are not even allowed to go to toilet. These practices have very little scientific support and the obstetric community should review these and incorporate appropriate changes.

5. Thromboprophylaxis should be used on the basis of risk factors (moderate risk group).

The risk factors considered

- a. Obesity (BMI >30)
- b. Age above 35
- c. Hypertension
- d. Triplets or higher order multiple pregnancy
- e. Extensive varicose veins
- f. Air travel
- g. Cesarean or midcavity instrumental delivery or cesarean hysterectomy
- h. Enforced bed rest for 4 days or more
- i. Sickle cell anaemia

It is suggested that if 3 or more of the factors exist, thromboprophylaxis is to be given starting within one hour of delivery and continued for

3-5 days or till fully ambulant. We would like to acknowledge that this is an empirical suggestion (good practice point) rather than on evidence as related to our population.

6. Patients with high risk for venous thrombosis

Anybody with history suggestive of thrombophilia in them or close relatives should be investigated for this possibility. A typical case scenario is the patient with recurrent pregnancy loss who on investigation turns out to be positive for APLA syndrome. A patient with lupus syndrome needs to be considered in this. Such patients need thromboprophylaxis in the form of aspirin and low molecular weight heparin during pregnancy and the heparin should be continued postpartum for about 6 weeks.

7. If the patient is on regional analgesia (epidural), insertion or withdrawal of catheter is to be delayed for 6-8 hrs after the last dose of heparin.

8. Whenever any patient is posted for cesarean, a risk assessment should be done regarding thromboprophylaxis and if they belong to moderate or high risk group appropriate medication be given.

RCOG Recommendations

The Royal College of Obstetricians & Gynaecologists have given guidelines regarding thromboprophylaxis which is available in the internet.

Table 12.2 Suggested thromboprophylactic doses for antenatal and postnatal LMWH which is given subcutaneously

Weight(kg)	Enoxaparin	Dalteparin
<50	20mg daily	2500units daily
> 50	40mg daily	5000 units daily
> 90	60mg daily	7500 units daily
Treatment dose	1mg/kg 12hrly antenatal 1.5 mg /kg/daily postnatal	100 units/kg/12hrly or 200units/kg daily postnatal

Conclusions

1. Venous thromboembolism is an important cause of maternal mortality and morbidity.
2. Puerperium is the highest risk period.

3. Risk assessment should be done at the initial visit. BMI should be entered.
4. If there is positive family history, thrombophilia screening should be done.
5. Encourage early ambulation in the puerperium.
6. Avoid unnecessary bed rest during pregnancy and puerperium.

Prophylaxis of DVT is easier than treatment. Pulmonary embolism is even more difficult to treat and carries a high risk of mortality.

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Chapter 13

Neurological events leading to Maternal death – An analysis

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Key summary points

- Fifteen deaths directly attributable to a neurological cause were reported to the CRMD during the period 2006-2009.
- Seizures were the commonest symptom that presented - 12 out of the 15 patients presented this way. In at least five out of 12, seizures was the direct cause of death.
- Encephalitic illness accounted for five cases.
- Intracranial hemorrhage (subarachnoid and parenchymal) were four.
- Cerebral venous thrombosis accounted for two cases.
- About 1/3 of all deaths due to neurological causes occurred in association with LSCS.
- The most frightening aspect was that the time from the first symptom to death was four days or less. Thus a neurological symptom must always be considered a red flag and a wakeup call to all concerned in the patient care.

Key Recommendations

1. Headache in a post partum patient should not be ignored. It may be a symptom of cerebral venous thrombosis.
2. CT scan as a basic investigation in suspected neurological problems should be done early. Delay in arriving at a diagnosis and instituting proper treatment may cause permanent loss of brain function. **“Time is brain”**.
3. Antiepileptic drugs should be given in adequate doses. While magnesium sulphate remains the best anticonvulsant in eclampsia, in non eclamptic causes of convulsions, other anticonvulsants should be used.

4. Eclampsia can present beyond the conventional 48 or 72 hours after delivery. Signs of preeclampsia need not be present in them.
5. Fosphenytoin, the most commonly used anticonvulsant in status epilepticus, should not be given as a drip, as extravasation can be catastrophic. It should be given as a slow bolus in a free flowing drip.
6. General principles of managing postpartum women like early ambulation and hydration are relevant from neurological angle as well in preventing complications like cerebral venous thrombosis.
7. In suspected cases of neurological problems and atypical presentations of eclampsia, involve a neurologist early.
8. Known epileptics, should discuss with their neurologist before planning pregnancy and make modifications of medications

Learning from examples

Example 1

27 years old primi – 40wks of gestation, delivered by LSCS. Post operative period was uneventful except headache on discharge. Developed seizures on day seven post partum. Went on to have status epilepticus, hypotension and tachycardia.

CT studies normal but cerebral venous thrombosis (CVT) was suspected and CT venogram was planned. ECHO: dilated cardiomyopathy.

Probable diagnosis: Status epilepticus due to CVT, Post partum eclampsia, Peripartum cardiomyopathy.

She did not improve and died on day 9 (34 hours after first symptom appeared).

Learning points

- Headache that persisted from day of discharge. Possibly a postponement of discharge and evaluation of headache might have led to earlier action.
- Never underestimate a headache. Always a red flag.

Example 2

33 year old G2 P1. First FTND. Induced with PGE2 gel at 6.10am. After 24 hours ARM was done, at 2cm dilatation – thick meconium – cesarean under spinal. Before placental separation patient had convulsion, became unconscious,

cardiorespiratory arrest, resuscitated, ventilated. CT scan of brain next day – cerebral edema with subarachnoid hemorrhage. Continued on ventilator and died on day 15.

Learning points

- A CT scan should have been taken earlier (If patient's condition permitted).
- Treatment with anticonvulsants would have helped.

Example 3

27 years old G2 P1, 16 weeks of gestation, known case of RHD, sudden onset of fever- vomiting and altered sensorium - comatose on admission – ventilated - expired on 3rd day.

No CT done. LP done, no record.

Time to death – 46hrs

Probably encephalitis

Learning points

- An earlier CT should have been done. Then we would have had a working diagnosis.

Example 4

22 year old G₃ P₁L₀A₁- admitted for BP control at 23 wks, became drowsy on day three with features of imminent eclampsia. First pregnancy had to be terminated at 24 wks for eclampsia. Second was abortion at 8 wks. She was positive for antinuclear antibodies and Rheumatoid antibodies.

Started on magsulph and induced. Delivered within 12 hours, fetus weighing 750 gms. BP high. Had a seizure on the 10th postnatal day. Another seizure on day 11 and expired on the same day.

Learning points

- The history and lab findings make one suspect possibilities of SLE with CNS vasculitis. Unfortunately detailed work up could not be done.

Example 5

25 year old, 7 months amenorrhoea, head ache, fever & giddiness. Unconscious in 24 hrs. Was being treated outside the state. Due to financial constraints

got discharged and was admitted in one of the hospitals in Kerala where she continued to be on ventilator. Later she developed ventilator associated pneumonia and expired. CT had shown bithalamic and mid brain hypodensity.

Learning points:

Unfortunately, this patient could not have complete workup. Financial matters would have played a part in this. Possibility of viral encephalitis (Japanese B variety) and thrombosis of deep venous system are to be suspected.

Example 6

30 yr old primi – 28wks gestation with seizures. Known epileptic on carbamazepine– 200mg. Developed recurrent seizures. Expired the next day.

Status Epilepticus – sudden death

Learning point

- Adequate dosage of anti epileptic drugs is mandatory. Here she was on one tablet 200mg of Carbamazepine, just enough for a 20kg child.

Example 7

29yr old, full term normal delivery doing well till 5th post natal day – develops seizures + (L) hemiplegia started on magsulph. CT showed a Intracerebral hemorrhage with early signs of herniation – ventilated. Progressively worsened and expired 10 days following delivery.

Learning point

- When focal signs are recent, a CT is mandatory as soon as possible. Magsulph works only in eclampsia

Stroke in pregnancy and puerperium

Magnitude of the problem:

- Stroke is the third leading cause of death and disability for both men and women
- The mortality from stroke is higher in women than in men (16% vs. 8%) This relates to the higher frequency of SAH in women and the greater

severity of stroke in women.

- Incidence of aneurysms and the risk of rupture is higher in women than in men. They also have a higher risk of rebleeding and a four fold increase in mortality from subarachnoid hemorrhage compared with men.
- Stroke is one of the more common causes of maternal death
- Highest risk for ischemic stroke is in late pregnancy and post partum. The post partum period increases stroke risk by 8.7 times

Causes of stroke and CVT in pregnancy and puerperium

A. Related to physiological changes in pregnancy:

Increase in blood volume

Vascular remodelling

Hypercoagulation

B. All causes related to stroke in the young.

Vascular remodelling and increased blood volume - Hemorrhagic stroke

Hypercoagulable state - ischemic stroke

The hypercoagulable state is the result of an increase in procoagulant factors and a decrease in coagulation inhibitors. Both these plus the risk of dehydration induced by hyperemesis and poor intake make ischemic stroke an increasing liability.

When to suspect a stroke?

Full blown stroke is obvious. Subtle signs are the key. Use the **FAST** test.

Face problems - asymmetric smile, mouth or eye drooping.

Arm problems - can he raise both arms? Any arm drift?

Speech problems - expression or comprehension.

Time to call for help.

A patient less responsive or in whom the bystander notices something different is a red flag.

Act Fast: Time is brain

- Neuro imaging is the first step
- CT scan is the modality of choice .In late pregnancy radiation risk to fetus is negligible.
- Contrast agents are better avoided as a routine. MRI better avoided in

early trimester

- Involve neurologist early in the illness.
- Remember you have only 4 ½ hrs to make a difference and the earlier the better.

Hemorrhagic Strokes

Epidemiology of Intracranial hemorrhage/ subarachnoid hemorrhage (ICH/SAH)

Eclampsia accounts for 30-44% of cases

Aneurysm

Arterio venous malformation (AVM)

Other vasculopathies.

Hypertension is the most common underlying factor.

Mortality and recurrence of ICH and SAH during pregnancy:

Rather high - 25-60% risk of aneurysm rupture. This increases throughout gestation and into the post partum period.

Ist trimester 6%. - Second trimester 30%. - Third trimester 55%. - Post partum 6wks - 9%

AVMs can bleed at any time.

Risk of rebleed with AVM is 25% - which is higher than in non pregnant state.

Risk of rebleed with aneurysm is 50%. - Which is similar to non pregnant

Diagnostic challenges:

- Symptoms overlap with eclampsia “both have headache, hypertension, seizures and coma”.
- SAH may also cause Albuminuria in 50% of cases.

Mode of delivery for patients with IC hemorrhage.

Ruptured aneurysm:

If clipped successfully = normal delivery.

If not clipped and rupture occurs in 3rd trimester = LSCS at 38 wks

Ruptured AVMS:

If excised before 35 weeks – normal delivery

If not excised and pelvis adequate = normal delivery without Valsalva.

If not excised and pelvis unproved - LSCS at 38 wks.

Ruptured intraparenchymal vessel

Vaginal delivery without Valsalva or LSCS at 38 weeks.

Unruptured aneurysms present a dilemma to neurosurgeon and obstetrician. Current recommendations are to treat symptomatic aneurysms more than 10mm in size or increasing in size, lobulated, or h/o previous rupture elsewhere.

Remember aneurysms increase in size with period of gestation

Learning points

- Strokes are not uncommon in late pregnancy and post partum
- Higher mortality rates compared with non pregnant states.
- Delay in detection increases mortality.
- Symptoms of ICH overlap with Eclampsia and the error is fatal.
- Involve Neurology/ Neurosurgery early.
- Many decisions need to be made jointly.

Seizures in pregnancy and the post partum period

May be pregnancy related or unrelated. Whatever the cause, they need to be evaluated and controlled rapidly. The time of onset and a well taken history often hold the key.

Differential diagnosis of a seizure occurring in a pregnant woman at or near term or in the postpartum period:

Eclampsia /Ante /Intrapartum/ Post partum

Cerebrovascular occlusions arterial or Venous.

Hypertensive encephalopathy

Reversible vasculopathy of pregnancy

Epilepsy - worsening or occurring for the first time

Brain tumour/ Abscess.

Infections - meningitis, encephalitis, HIV

Metabolic

Pseudo seizures and other rare causes

The list is long, and probably not complete, but going by commonality and common sense, a few features stand out.

A seizure in a known epileptic occurring at term or post partum, possibly is a breakthrough seizure, and needs modification of antiepileptic drug(AED) dosage.

A seizure for the first time at term or post partum is eclampsia till proven otherwise. Full eclampsia workup is advised.

Any seizure where the patient does not regain consciousness within a reasonable time needs imaging. So also, a patient not responding to Magsulph regime needs an imaging and further evaluation and perhaps add phenytoin for seizure control.

A seizure occurring > 7 days post partum is probably a CVT.

What is special about postpartum Eclampsia?

More common than we think, about 30% in some series

Can occur later than the accepted cut off point of 48 hours

Difficult to predict as the classical triad of hypertension, edema and proteinuria need not be present, and PET is diagnosed in <50%

It is the only subset of eclampsia that is not declining in incidence

Head ache and visual disturbances are almost universal presentations

When is Neuro imaging indicated in a convulsing patient?

-If refractory to mag sulf

-If classic triad is absent

-If lateralising signs exist

50% of patients may have transient changes on CT/MRI in the Parieto occipital regions, suggesting a vasospastic process

Treating the convulsing patient:

If the diagnosis of eclampsia is clear, magsulph is the treatment of choice.

If you must refer the patients to a higher facility, always give the loading dose of magsulph before sending, and mention the same in the reference letter.

Avoid giving diazepam before sending, it will confuse the picture at the next hospital and may also be risky when combined with magsulph.

If the patient continues to convulse chances are that some other etiology /additional etiology is at work. You probably are looking at a status epilepticus and is time to shift to a higher gear.

Treatment of status Epilepticus

Time is the key.

Benzodiazepines stop the ongoing seizure.

You need another drug to keep her seizure free.

You need to recognise status Epilepticus early.

First line medications control the status 80% of the time if given within 30mins, but only 40% are controlled if given after 2 hours.

Fosphenytoin is the most commonly used drug. A loading dose of 20mg/kg is given in the following manner:

Start a free flowing drip. Into the tubing inject 1cc. (75mg) of Fosphenytoin over 30 seconds. Wait for 30 seconds and repeat the process till the whole dose has been given. This ensures a rate of 150mg/mt, and the full dose can go in 10-15mins. **Do not add the drug into the drip.** If seizures persist, repeat 1/3 of the above dose. **If this too fails, there is need to go up the ladder.**

Choices are:

IV sodium valproate 40mg /kg in 100ml NS over 10mins - Repeat 20mg/kg if fits persist

IV phenobarbitone 20mg /kg loading.

IV pentobarbital 5mg. /kg - till seizures stop.

The last option requires intubation + EEG monitoring to maintain a burst suppression pattern.

Cerebral venous thrombosis (CVT)

Accounts for 2% of strokes in pregnancy in post partum. Fatality ranges from 4-36%. Widespread use of CT/MRI/MRV has changed the face of CVT, rendering early diagnosis and treatment possible.

As discussed earlier this is a prothrombotic period with hormonal and physical factors all playing a role.

Pathophysiology

Occlusion of cerebral vein – localised brain oedema – venous infarction.

Initially a cytotoxic edema caused by ischemia. Later B.B barrier breaks – Vasogenic edema.

If occlusion of major veins/ sinuses occurs – development of increased intracranial pressure(ICP) due to impaired CSF absorption occurs.

Clinical manifestations

Headache is the most common

Focal deficits

Diffuse encephalopathy – seizures

Psychiatric disturbances

Painful Ophthalmoplegia if the cavernous sinus is involved

Diagnosis of CVT

- Neuro imaging is the modality of choice - CT venogram or MR venogram
- On a plain CT, the thrombosed vessel or sinus gives rise to 3 classic signs. The cord sign, the delta sign and the empty delta sign. However CT may be normal in up to 20% of all cases of proven CVT. Hence CT venogram (CTV) or MR venogram (MRV), and sometimes even conventional angiography is needed.
- **D-Dimer** measurement is more useful in ruling out rather than ruling in CVT. The negative predictive value is most useful in patients with encephalitic signs. D-Dimer levels must always be interpreted with the clinical picture in mind. Its utility is limited if surgery has been done recently.
- Thrombophilia work up is important. One study showed that up to 75% of patients with CVT had a proven hypercoagulable state.
- Factor V Leiden mutation and hyper homocystienemia are the commonest anomalies found.

Outcome and predictors of 30 day case fatality

Impaired consciousness

Thrombosis of deep systems

Right hemisphere hemorrhage

Posterior fossa lesions.

Cause of death

Transtentorial herniation.

Status Epilepticus

Pulmonary embolism

Other medical complications.

Treatment

Acute phase – specific

Heparin - LMW or UF at full therapeutic dosage to keep a PTT > 2 x normal. The aim being to prevent extension of thrombus and treat underlying prothrombotic state, prevent DVT /Pulmonary embolism and prevent recurrence CVT.

Acute phase: - supportive

Anticonvulsants

Treatment of increased ICP- sometimes hemicraniectomy may be life saving.

Long term:

Anticoagulation with warfarin is usually given for 6 months to a year.

Lifelong if underlying prothrombotic state is seen

What about future pregnancies?

The results of 5 studies suggest that if no prothrombotic state is found, CVT even if pregnancy related should not contraindicate future pregnancies.

In summary:

- The treatment of CVT implies a high level of suspicion and early evaluation and treatment.
- Anti coagulation
- Anti convulsants
- Anti edema
- Heparin is mandatory, however hemorrhagic the CT looks. Just be sure it is a CVT. Keep checking with repeated CT's.

Reversible Posterior Leucoencephalopathy

It is now realized that sometimes there will be wide spread cerebral segmental vaso constriction and edema. The exact etiology is not known but it can occur in Eclampsia or postpartum period. Typically the lesion reverses completely. There are different types of reversible vasculopathy.

1. Reversible Posterior Leuco Encephalopathy Syndrome (RPLE syndrome)

Pathophysiology

- Sudden increase in BP
- Exceeds auto regulatory capability.
- Increased sensitivity to normal pressor agents
- Deficiency of vasodilating prostaglandins.
- Endothelial cell dysfunction.
- Affects the small vessels predominantly.

Treatment

- Treatment of Eclampsia with magnesium sulphate
- Treat Hypertension. Recommendation for BP is <105-110 diastolic and <155 mm.Hg systolic.

2. Reversible cerebral vasoconstriction syndrome.

This is a unifying term used to describe a clinical syndrome characterized by sudden severe headache, fluctuating neurologic deficits, and fully reversible segmental vasoconstriction of the medium and large cerebral arteries.

3. Postpartum cerebral angiopathy.

This is a rare condition for which no incidence rate is cited in the literature (Konstantinopoulos, Mousa, Khairallah, & Mtanos, 2004). It can cause ischemic or hemorrhagic stroke, or both, and usually occurs within the first week following a normal pregnancy and uncomplicated delivery. Although the Pathophysiology is unclear, it is believed to be caused by an inflammatory process, such as vasculitis or transient vasospasm .

Conclusions

Neurological disorders are not uncommon causes of maternal mortality. Involvement of a neurologist and investigations like CT brain should be early rather than late. The message in chapter on hypertensive disorders of pregnancy to aggressively control acute hypertension and prevent cerebrovascular accident is reiterated here. Once cerebral bleed occurs, the prognosis is grave. Importance of early ambulation and fluid intake in postpartum period should be communicated to pregnant women in antenatal period itself. Head ache in postpartum period should not be ignored.

Chapter 14

Pregnancy related Acute Kidney Injury (PR-AKI)

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Introduction

Analysis of case records has revealed that pregnancy related acute kidney injury contributes significantly to the maternal mortality in our state. In this chapter we would like to highlight the common areas where improvement is possible and discuss some of the basic pathophysiology that will help to understand the management principles. With renal replacement therapy maternal mortality due to sequelae of AKI like fluid overload, acidosis or hyperkalemia has declined. They die with renal failure and not of renal failure. Mortality is usually from the basic disease or co morbid factors.

Key summary points

- Failure to replace blood loss and maintain adequate perfusion pressure is the most common cause of PRAKI.
- Often the situation is made worse with injudicious use of nephrotoxic drugs like NSAIDS and aminoglycosides.
- PRAKI often presents as one of the important components of multiorgan dysfunction syndrome.
- In many cases, sepsis syndrome seems to be the immediate cause of mortality.
- Acute pyelonephritis, systemic lupus erythematosus(SLE), antiphospholipid antibody syndrome (APLA syndrome), immune mediated thrombocytopenia, and HUS were the important systemic diseases.
- In APLA syndrome continuation of Heparin is essential.
- Early and prompt termination of pregnancy in HELLP syndrome after correction of coagulation abnormalities will prevent maternal deaths.

Key Recommendations

1. When there is excessive blood loss as in PPH, circulatory support with I V Fluids and prompt replacement of blood and blood products is essential. Otherwise kidney function will be compromised.
2. When there is threat to renal function, as in a case of shock, the dose and interval of nephrotoxic drugs like aminoglycosides and NSAIDS should be carefully monitored.
3. Patients with sepsis should be aggressively managed with adequate fluid replacement, judicious use of vasopressors, ionotropes, measures to increase oxygen delivery and blood components.
4. Patients on immunosuppressives are at increased risk for sepsis and aseptic precautions should be strictly followed. A diligent search for infection should be done.
5. Patients with SLE should be in remission at the time of conception. Drugs like mycophenolate mofetil, Calcineurin inhibitors(CNI) have to be discontinued before planning pregnancy.
6. Though the common causes of thrombocytopenia are Gestational thrombocytopenia and HELLP syndrome, other conditions with an adverse prognosis like SLE, HUS – TTP syndrome, ITP should be suspected when platelet count is persistently low.

Learning from examples

Example 1:

This 25 yr old, G5P4L3 had normal delivery at community health centre at 10pm. Her blood group was B- negative. Due to atonic PPH she was referred to FRU and from there to medical college. By then she had developed DIC. Total hysterectomy with left salpingo oophorectomy was done at 2.40am. Relaparotomy was done six hours later. She developed pulmonary edema and renal failure. Hemodialysis was started. Anuria persisted. She died on dialysis 12 days later.

Learning points

The fact that she was in shock and DIC on arrival at medical college suggests that volume replacement did not occur promptly. The kidney failure would have been a consequence of her prolonged hypotension

Example 2

Twenty nine year old lady presented with fever of two days and dysuria. She developed acute pyelonephritis, sepsis syndrome and expired after 48 hours of hospitalization.

Learning points

Urinary tract infection is a serious complication in pregnancy. When suspected after 24 weeks of pregnancy, should be managed aggressively. Maternal complications like septic shock and Acute Respiratory Distress Syndrome in untreated pyelonephritis is as high as 20 %. Early initiation of parenteral antibiotics in this lady would have been beneficial.

Example 3

Twenty nine year old lady presented with seizures and accelerated hypertension. She had pre existing SLE. She was managed with magnesium sulphate, anti hypertensives and supportive measures. The activity of SLE was not assessed. The differential diagnosis of Neuro lupus should have been entertained.

Learning points

Pregnant women with SLE should be screened for activity and appropriate measures instituted. In this lady differential diagnosis of neuro lupus should be entertained since steroid pulses and cyclophosphamide may improve outcome. Though it is difficult to distinguish these two disorders, activity indices like low C_3 , increasing anti Ds DNA antibodies, would have given a clue.

Example 4 .

Twenty four year old lady with no h/o hypertension and other co morbid factors presented with echymotic patches, bilirubin >12mg%, elevated liver enzymes and progressive renal failure. She was managed as a case of HELLP syndrome. She was taken for plasma exchange after 3 days and expired.

Learning points

Hemolytic uremic syndrome (HUS) is a mimicker of HELLP syndrome but carries a bad prognosis. Echymotic patches, decreasing platelet count and resistance to improve after termination of pregnancy are clues. Early institution of plasmapheresis in patients with high index of suspicion may alter outcome favorably.

Example 5

Thirty one year old lady diagnosed to have APLA syndrome was on heparin. Her antenatal course was also complicated by gestational DM and hypertension. Because of persistent thrombocytopenia a diagnosis of Heparin induced thrombocytopenia was suspected. Heparin was stopped. She died due to ?Pulmonary embolism.

Learning points

She had evidence of bone marrow suppression, not thrombocytopenia alone. Heparin does not produce bone marrow suppression, but induce only thrombocytopenia. It is not clear whether this lady had APLA syndrome associated with SLE. All women with APLA should undergo detailed work up for SLE.

Example 6

Nineteen year old lady admitted with h/o Raynaud's phenomenon and fetal loss, was admitted with a reference as HELLP syndrome. In spite of appropriate measures for HELLP syndrome, she expired.

Learning points

Whenever there is a systemic symptom and bad obstetric history, regular antenatal screening for underlying systemic disease like SLE should be done.

Overview

Acute Kidney injury (AKI) has replaced the term "Acute Renal Failure" The incidence of **AKI** during pregnancy has declined from 1/3000 in 1980s to approximately 1 per 10,000.

AKI is defined as abrupt and rapid decline in glomerular filtration rate (GFR), resulting in inability of the kidney to maintain internal homeostasis.

Pregnancy produces many anatomical and physiological changes in renal and circulatory hemodynamics. This results in certain alterations in acid base balance, fluid and electrolyte homeostasis and GFR. Normal values change in pregnancy.

Definition –Is defined as plasma creatinine > 0.8mg.

Physiological changes during pregnancy

Why is plasma urea / creatinine low in pregnancy?

Renal plasma flow increases by 50-70% in pregnancy and GFR increases up to

150% of normal. With increase in GFR, plasma urea and creatinine decrease. This decrease has clinical significance in that a BUN or creatinine level in a pregnant female similar to the normal in a nonpregnant, may actually indicate underlying renal disease.

Physiological changes in blood pressure

Because of the vasodilatory effect of progesterone blood pressure decreases by about 10mm Hg with maximum fall at about 24wks. To avoid errors, 75mm Hg in the second trimester and 85 mm Hg in the third trimester are considered as upper limits of normal.

Other changes brought about by progesterone

There is fall in sodium values by about 5mmol. There will be mild respiratory alkalosis, with pH of 7.44 as normal, PCO₂ values may drop to around 30mm of mercury. As complementary mechanism serum bicarbonate levels may drop to about 22meq/L. The ureters especially the right one will be dilated.

Table 14.1 Normal laboratory parameters in pregnancy

Variable	change	Approximate normal values
Creatinine	decreased	0.5mg/dl
Blood urea	decreased	18mg/dl
GFR	increased	40 to 60% above normal
Uric acid	decreased	2-3mg/dl
Na retention over pregnancy	Increased 40 – 50%	900 to950 mmol
Plasma osmolality	decreased	By 10 milli osmoles/kg H ₂ O
PCO ₂	decreased	By 10mm Hg below baseline
pH	increased	>7.44
Serum HCO ₃	decreased	18 – 20meq/L
Serum calcium and albumin	decreased	8 -9 mg/dl
Urine glucose	variable	
Urine protein	increased	300 -500 mg/day

Pathophysiology and practical aspects of management

Table 14.2 Common causes of kidney injury

<i>Early pregnancy</i>	<i>Mid or late pregnancy</i>
Acute pyelonephritis	Post partum hemorrhage
Hyperemesis gravidarum	Abruptio placentae
Sepsis syndrome	Preeclampsia, HELLP,, AFLP
Septic abortion	Acute fulminant hepatitis
CKD exacerbation	Hemolytic uremic syndrome,
Renal transplant related AKI	APLA syndrome, Sepsis syndrome
	Renal transplant related AKI

Pathology

Most common renal lesion is acute tubular necrosis (ATN). Severe form of ATN or renal cortical necrosis can be seen in abruptio placentae, sepsis syndrome etc. ATN is reversible whereas acute cortical necrosis is irreversible.

The underlying factor is renal ischemia and commonly due to hypoperfusion and hypovolemia. If ischemia is prolonged, cortical necrosis is the result and is irreversible.

Development of cortical necrosis is not related to parity, age, severity of PIH or development of seizures. Renal Cortical necrosis is usually seen in Abruptio placentae. In order to detect Kidney injury early, AKI has been staged according to RIFLE criteria

RIFLE criteria

Risk	<i>increased creatinine x1.5 or GFR decrease > 25%</i>	<i>UO<0.5ml/kg/hrx6hr</i>	<i>High sensitivity</i>
Injury	<i>increased creatinine x2 or GFR decrease >50%</i>	<i>UO<0.5ml/kg/hr x12hr</i>	
Failure	<i>increased creatinine x3 or GFR decrease >75%</i>	<i>UO <0.3ml/kg/hr x24hrs or Anuria x 12hrs</i>	<i>High specificity</i>
loss	<i>Persistent ARF - complete loss of renal function >4weeks</i>		
ESRD	<i>End Stage Renal Disease</i>		

In pregnancy there is increased predisposition for pulmonary edema. There is an increase in preload, increase in capillary permeability and decrease in colloid osmotic pressure. At significantly lower PCWPs of 18 to 20 mm Hg there is decrease

in Cardiac output (CO) heart rate (HR) systemic vascular resistance (SVR), pulmonary vascular resistance. Hence fluid administration should be carefully titrated.

Increase in Cardiac Preload

- Increase in Capillary Permeability
- Decrease in Colloid Osmotic Pressure
- Pulmonary edema

Hence fluid deficit correction should be very judicious.

Commonest cause of hypovolemia in pregnancy is acute blood loss. The degree of hypovolemia has to be clinically assessed.

Assessment of hypovolemia and correction in hemorrhagic shock

Table 14.2 Classification of Hemorrhage/Hemorrhagic Shock

Variable	Class I	Class II	Class III	Class IV
Blood loss, mL (% blood volume)	500-1000ml <15%	500-1000ml 15-30%	1500 -2000 ml 30-40%	2000 -3000 ml >40%
Pulse rate	Normal	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Respiratory rate	Normal	20-30	30-35	>35
Urine output	Normal	20-30 mL/h	5-15 mL/h	Anuric
Mental status	Slightly anxious	Mildly anxious	Confused, anxious	Confused, lethargic
Response to fluid bolus	Yes	Yes	Transient	No

In class I, the replacement fluid should be crystalloid and blood, and in class II, blood or colloids if blood is not available, should be used. When we use crystalloids, three times the volume of blood lost should be replaced because of the distribution into interstitial space also.

Table 14.3 The composition of commonly used parenteral fluids(in 1000ml)

Solution	Dextrose	Na	K	Cl	Lactate	Ca.	others
0.9% saline		154	-	154		.	
0.45% saline		77	-	77	—		
5% dextrose	50 g	-		-		-	
5% DNS	50 g	154	-	154			
Ringer's Lactate	-	130	4	109	28	3	
Haemaccel		145	5	145		6.25	Gelatin 35 gm
Hetastarch		154		154			Starch 60g
5% albumin		160	2				50g

Key points

- Certain physiological changes occur in total body water.
- Blood loss due to causes like post partum hemorrhage lead to hypovolemic shock. There is an increased predisposition for pulmonary edema, hence deficit correction should be carefully monitored.
- When crystalloids are used for correction of blood loss, three times the assessed blood loss should be given.
- When Ringer's lactate is used, make sure the renal parameters are normal.

Hypovolemia and AKI

Since there is an increased vulnerability for pulmonary edema in pregnancy, fluid administration should be judicious. When the hypovolemia is not overt, fluid challenge should be given.

Fluid Challenge is adequate when end organ BP or cardiac output increases with increase in CVP or heart rate decreases and CVP increases without change in BP. Normal saline or Ringer's lactate is the fluid of initial choice.

Sepsis syndrome and AKI

Early goal directed therapy is to be instituted in the first 6 hours '*golden hours*'. Adequacy is assessed by clinical parameters MAP >60 mm, skin perfusion, urine output > 0.5ml/kg/hour improvement in sensorium, central venous oxygen saturation >70%, arterial lactate <2mmol/L.

Goal of therapy in first 6 hours

1. Normalize preload

- Aim to attain a Central venous pressure of 8 – 12 mm Hg or 12 – 15 mm Hg in those who are mechanically ventilated or have decreased ventricular compliance.
- Crystalloids or colloids can be used. Normal saline or Ringer's lactate is the fluid of first choice. As soon as hypo perfusion is recognized, 1000ml of normal saline or 500 ml of colloids can be infused over 30 minutes

2. Normalize perfusion pressure -

Aim – After achieving a CVP of 8 -12 mm Hg , if MAP (Mean Arterial Pressure) is less than 65mm Hg

Vasopressors – Noradrenaline and dopamine are the drugs of choice. There is no role for “renal dose Dopamine”. Phenyl ephrine, epinephrine are used only if patients do not respond to noradrenaline and dopamine.

3. Normalize cardiac index

Inotropes - Dobutamine is useful to increase cardiac index.

To prevent tissue hypoxia by matching oxygen delivery with consumption - $Scvo_2 > 70\%$

O₂ support, packed red cell transfusion to increase PCV (30 to 33%)

In sepsis associated AKI, control of infection, by judicious antibiotic therapy, and early goal directed therapy may improve outcome

Nephrotoxic AKI

Nephrotoxic drugs were contributing factors in 19% to 25% of cases of severe acute renal failure in critically ill patients

Common causes of nephrotoxic AKI in pregnancy are

- Non steroidal anti inflammatory drugs (NSAIDs)
- Aminoglycosides , Vancomycin, cephalosporins
- Loop diuretics
- Abortifacients

Non steroidal anti inflammatory drugs (NSAIDs) are commonly used for relief of pain and for it's anti-inflammatory properties.

Predisposing factors are

- Hypovolemia
- Combination with other nephrotoxic drugs like ACE inhibitors, Angiotensin Receptor Blockers (ARBs), Aminoglycosides
- Preexisting renal dysfunction.

Mechanism of AKI

Prostaglandins are vasodilatory and NSAIDs block the action of prostaglandins, thus impairing the blood flow. In patients with renal dysfunction, the afferent vasoconstriction produced by NSAIDs will reduce renal perfusion and cause hemodynamically mediated AKI. The other mechanism of renal injury is acute tubulointerstitial nephritis.

Aminoglycosides are very effective antibiotics in sepsis. They are also nephrotoxic, ototoxic and also blocks neuromuscular junction.

Dose of aminoglycosides have to be reduced in patients with decreased GFR.

The standard dose of gentamicin is 3-5mg/ kg/ day or 1-1.7mg/ kg q8h IM. To modify the dose in impaired renal function, multiply serum creatinine concentration (mg/dl) by 8 to obtain frequency of injection, or divide total scheduled dose by serum creatinine concentration and repeat this lower dose (daily or 8 hourly depending on whether daily dose or eight hourly dose was used for calculation).

Prevention

- Avoid hypovolemia
- Avoid the use of concomitant nephrotoxic drugs
- Total doses to be restricted to 5 doses.

Key points

- NSAIDs should be avoided in patients predisposed for AKI.
- Amino glycoside dose has to be adjusted to GFR.
- Concomitant use of nephrotoxic drugs should be avoided.

AKI - late pregnancy

Table 14.4 Differentiation of Preeclampsia, HELLP, AFLP, HUS, TTP

Preeclampsia onset	HELLP 3rd trimester	TTP Median 23 weeks	HUS Often post partum	AFLP Third trimester close to term
<i>primary Cl: feature</i>	<i>hypertension Edema, epigastric pain</i>	<i>neurologic Seizures, altered sensorium</i>	<i>Renal Hematuria, altered renal parameters</i>	<i>Nausea, vomiting Edema, epigastric pain</i>
<i>Purpura</i>	<i>absent</i>	<i>present</i>	<i>absent</i>	<i>absent</i>
<i>fever</i>	<i>absent</i>	<i>present</i>	<i>absent</i>	<i>absent</i>
<i>hemolysis</i>	<i>mild</i>	<i>severe</i>	<i>severe</i>	<i>mild</i>
<i>Coagulation profile</i>	<i>variable</i>	<i>normal</i>	<i>normal</i>	<i>abnormal</i>
<i>hypoglycemia</i>	<i>absent</i>	<i>absent</i>	<i>absent</i>	<i>present</i>
<i>vWF multimers</i>	<i>absent</i>	<i>present</i>	<i>present</i>	<i>absent</i>
<i>Primary treatment</i>	<i>delivery</i>	<i>plasmapheresis</i>	<i>plasmapheresis</i>	<i>delivery</i>

Management of Specific Situations

Pre-Eclampsia

Adequate prenatal care is the most important factor. Blood pressure should be kept around 140/90 to avoid placental hypoperfusion. When there is evidence of severe pre-eclampsia, pregnancy should be terminated to avoid maternal complications.

HELLP and AFLP Syndrome

In HELLP syndrome and AFLP, termination of pregnancy is the mode of treatment. Corticosteroids around 34 weeks of pregnancy has been found to be beneficial.

Classification of HELLP Syndrome on the basis of platelet count

- Class I, less than 50,000 per mm³
- Class II, 50,000 to less than 100,000 per mm³
- Class III, 100,000 to 150,000 per mm³

Management

- Control of hypertension
- Blood products
- Prophylactic transfusion of platelets at delivery does not reduce the inci-

dence of postpartum hemorrhage or hasten normalization of the platelet count. .

- Patients with DIC should be given fresh frozen plasma and packed red blood cells.
- Steroids – Betamethasone is beneficial to increase lung maturity

HUS-TTP Syndrome

In HUS - TTP syndrome, plasma exchange is the line of management and termination of pregnancy has no role. Usually, renal failure progresses to CKD and may require transplant. Mortality of HUS syndrome is high.

Systemic lupus erythematosus (SLE)

Pregnancy probably increases lupus activity.

About 50% of women will have measurable SLE activity during pregnancy.

Most of the disease activity will be mild to moderate.

15% to 30% of women will have highly active SLE in pregnancy.

Most common types of SLE activity in pregnancy:

- Cutaneous disease
- Arthritis
- Hematologic disease

Risk factors for increased lupus activity:

- Active lupus within the 6 months before conception
- Multiple flares in the years before conception
- Discontinuation of hydroxychloroquine

Lupus nephritis

Women with a prior history of lupus nephritis have 20% to 30% risk of relapse during pregnancy. For women who have worsening renal function because of SLE nephritis during pregnancy, an estimated 25% had continuing renal damage after pregnancy, despite aggressive therapy. Very few women require lifelong dialysis.

Pregnant women with SLE are at increased risk for preeclampsia

- first pregnancy
- have a history of preeclampsia

- have renal disease,
- have active SLE at conception,
- have positive anti–double-stranded DNA antibody (dsDNA) or antiribonucleoprotein antibodies,
- have low complement,
- are obese, have hypertension

Symptoms suggestive of lupus disease activity

- Constitutional symptoms may be present.
- Most patients with lupus report fatigue during pregnancy.
The likelihood of developing renal disease during **pregnancy is not increased if the patient was in remission at the time of conception.**

Differentiation of signs and symptoms of normal pregnancy from those of exacerbations of lupus.

- Differentiate malar rash from chloasma.
- Differentiate proteinuria secondary to preeclampsia from proteinuria due to lupus nephritis.
- Differentiate thrombocytopenia in pregnancy (hemolysis, elevated liver enzyme levels, and low platelet counts [HELLP] syndrome) from thrombocytopenia of lupus exacerbation, (Thrombotic thrombocytopenic purpura [TTP] or idiopathic [ITP]).
- Pedal edema and fluid accumulation in joints, especially the knees, can occur in the late stages of pregnancy and should be differentiated from the arthritis of systemic lupus erythematosus (SLE).

Risk assessment in terms of checking for antiphospholipid antibodies (for a risk of fetal loss) and for anti-Ro and anti-La antibodies (for a risk of neonatal lupus) should be performed before pregnancy.

Laboratory studies

At the first visit after or when pregnancy is confirmed, the following assessments are recommended:

- Physical examination, including blood pressure evaluation
- Renal function tests, including determination of the glomerular filtration rate, urinalysis, and tests of the urine protein–to–urine creatinine ratio
- Complete blood count
- Test for anti-Ro/SSA and anti-La/SSB antibodies

- Lupus anticoagulant and anticardiolipin antibody studies
- Anti-dsDNA test
- Complement (CH50 or C3 and C4) tests
- During the first 2 trimesters, a monthly platelet count or complete blood count is recommended.

The following evaluations are recommended at the end of each trimester of pregnancy:

- Determination of the glomerular filtration rate and measurement of the urine protein-to creatinine ratio
- Anticardiolipin antibody measurement
- Complement (CH50 or C3 and C4) test
- Anti-ds DNA antibody study

Management – A multidisciplinary approach is essential. Prednisolone and Azathioprine can be continued. CNI inhibitors and Mycophenolate mofetil have to be withdrawn when pregnancy is planned. Peripartum flares should be managed with methyl prednisolone pulses.

Anti phospholipid Syndrome (APLA)

Clinical criteria

Vascular thrombosis: One or more clinical episodes of arterial, venous, or small-vessel thrombosis, occurring within any tissue or organ

Complications of pregnancy

- One or more unexplained deaths of morphologically normal fetuses at or after 10 weeks' gestation
- One or more premature births of morphologically normal fetuses at or before 34 weeks' gestation
- Three or more unexplained consecutive spontaneous abortions before 10 weeks' gestation

Laboratory criteria

- Anticardiolipin antibodies – Anticardiolipin IgG or IgM antibodies present at moderate or high levels in the blood on two or more occasions at least 12 weeks apart

- Lupus anticoagulant - Lupus anticoagulant antibodies detected in the blood on 2 or more occasions at least 6 weeks apart, according to the guidelines of the International Society on Thrombosis and hemostasis

The clinical criteria and one lab evidence should be there to diagnose APLA

Management

Heparin is given during the antenatal period and corticosteroids are given if associated with SLE around the peripartum period.

Renal replacement therapy in AKI

Choice of modality and dose of RRT are individualized. Daily short duration hemodialysis is the accepted mode of therapy. Early dialysis is necessary in pregnant women with renal failure and should be considered when the serum creatinine reaches 3.5 mg/dL or the glomerular filtration rate (GFR) is less than 20 ml / min. Longer, more frequent dialysis (20 h/wk) is associated with the best fetal outcome. Hemodialysis may therefore be necessary at least 5 days per week. Careful avoidance of hypotension is important. Peritoneal dialysis with smaller volumes and frequent exchanges is another option.

Conclusions

Pregnancy related kidney injury should be prevented by identifying the risk factors and prompt correction. Pregnant ladies with complicating systemic illness like SLE should be screened for activity. Preconception counselling is required in such patients regarding planning of pregnancy, need for monitoring and outcome both maternal and fetal should be discussed. Specific treatment modalities are available for certain conditions like, SLE HUS- TTP syndrome, APLA syndrome etc.

Chapter 15

Respiratory diseases causing maternal mortality

Mathew Thomas

[We decided to have a chapter on respiratory diseases at the planning stages of this book in early 2010 when H1N1 pneumonia was just emerging as a killer of pregnant women. In the subsequent monsoon we lost about 24 mothers in our state due to H1N1. Thankfully in 2011 its impact declined considerably. Still, we feel that this subject deserves a separate chapter as there were significant number of maternal deaths due to respiratory causes. (Editors)]

Key summary points

- Respiratory diseases are significant causes of maternal deaths – 21 out of 331 (6.3%)
- While interpreting findings, the physiological changes of pregnancy in the respiratory system have to be kept in mind.
- The important causes were bacterial and viral (H1N1) pneumonia and bronchial asthma
- Early treatment with high dose of oseltamivir (even before virological confirmation) will make a drastic difference in outcome in H1N1 pneumonia
- Women with bronchial asthma will benefit from prepregnancy counseling and getting asthma under proper control before embarking on a pregnancy.

Key Recommendations

- Since respiratory diseases in third trimester carry worse prognosis, they should be treated more aggressively, if necessary by referral to higher centres.
- If there are reasons to suspect H1N1 pneumonia in pregnancies beyond 28 wks, treatment with oseltamivir at higher doses (150 mg twice daily) should be started without waiting for laboratory confirmation.
- When there is bacterial pneumonia, aggressive antibiotic therapy should be initiated (eg piperacillin tazobactam, meropenem or imepenem).

- When there is no response to adequate higher antibiotics, consider adding antifungals
- Use of aerosol treatment for severe degrees of asthma is strongly recommended..
- Pulse oxymetry, arterial blood gas analysis (ABG) and noninvasive ventilation should be commenced early rather than late in the management of the respiratory disorder.
- Women with respiratory disease like bronchial asthma should get their disease properly controlled before embarking on pregnancy
- In respiratory disorders when fetal lung maturity is confirmed, accelerating delivery (vaginally or by cesarean) should be considered to remove the pressure of gravid uterus on diaphragm

Learning from Examples

Example 1

This 25 year old G3P2L2, LCB 4 ½ yrs, was referred from district hospital to medical college at 36wks pregnancy, as a case of acute exacerbation of bronchial asthma, leaking p/v and abdominal pain. She had regular antenatal care. There were several episodes of breathlessness associated with bluish discoloration of fingers. Hb was 12gm%. Respiratory rate 66/mt. She had cyanosis and bilateral rales and rhonchi . Clinically gross IUGR. Nebulisation, antibiotics etc started. Echo suggested pulmonary hypertension. Seriousness explained to the relatives. Had spontaneous labour within 90mts, IUGR baby weighing 1.44 kg with good Apgar. Dyspnoea worsened, saturation dropped further, intubated and ventilated. She died 5 hours after admission.

Learning points

- She had several episodes of dyspnoea and cyanotic attacks during pregnancy which was not investigated properly to arrive at a diagnosis.
- Notes suggest that she was a known case of asthma and had cyanotic attacks in which case more aggressive management was warranted earlier. She should have been counselled against this pregnancy if pulmonary hypertension was diagnosed earlier.

Example 2

30 years old, G3P2 L2, was admitted for safe confinement. She was a known asthmatic on treatment. She developed respiratory infection while she was in the ward. Started on ampicillin and nebulisation with salbutamol. There was no improvement. The next day treatment was changed to aminophyllin, ecorlin, and taxim. She developed severe respiratory distress and stridor. There was suspicion

of retrosternal extension of multinodular goiter. She later developed respiratory arrest. So she was intubated, and ventilated. A cesarean section was done for baby's sake. Though she was continued on ventilator she died five days later.

Learning Points

- When a known asthmatic develops respiratory infection, it needs aggressive antibiotic coverage.
- There seems to have been a reluctance to investigate whether retrosternal thyroid goiter was the cause of the stridor. This might have been deferred considering the pregnancy. Radiological exposure to upper thorax and neck should not be withheld considering pregnancy, if the information is crucial for management. At least an ultrasound examination would have been safe.
- The decision to empty the uterus is to be appreciated. Perhaps this could have been done earlier.

Example 3

25 year old primi with 7 months pregnancy was seen with a history of fever of nine days, cough of eight days and loose stools. She was already diagnosed as a case of H1N1 pneumonia. Had tachypnoea, cyanosis, was started on cefixime(Taxim), azithromycin, deriphylline , high flow oxygen, methyl prednisolone 500 iv bd. Nebulisation with salbutamol and oseltamivir 75 mg (Tamiflu). Discussed with obstetrician about termination of pregnancy but the advice was not to intervene. By evening she was put on ventilator and started on vancomycin and piptaz. ABG showed metabolic acidosis. By day three fetal heart sounds were doubtful. ABG showed combined metabolic and respiratory acidosis. SPO2 came down to 12%, BP was 60 systolic. Dopamine was started. By day five ultra sound scan confirmed fetal demise. Vaginal PgE₁ induction was started. After about 24 hours dead fetus was expelled. On day seven she was started on noradrenalin drip. On day eight vasopressin drip was started. On day nine she was declared dead.

Learning Points

- This patient presented to the higher centre, nine days after the onset of fever. She had confirmed H1N1 pneumonia. Our experience as well as the literature confirm that only those cases where the treatment is started early will have good results. Unfortunately there was delay. This case happened relatively early in the epidemic of H1N1 in our state. The importance of early diagnosis and aggressive management was not widely known at that time.

- She was started on the standard dose of oseltamivir. Now it is recommended that double dose (150mg bid) is to be given for severe cases. Usually we see severe secondary bacterial infection associated with H1N1 infection. So a higher antibiotic like meropenem should have been tried.
- Earlier termination of pregnancy also would have helped with the ventilation even though it is doubtful whether it would have made any difference in the outcome.

Overview

Physiological Changes

Pregnancy induces profound changes in the mother resulting in significant alterations in anatomical and functional changes in the respiratory system. Management of respiratory diseases in pregnancy requires an understanding of these changes. Awareness of these changes is also necessary for interpreting clinical and laboratory manifestations of the disease states. Some of these important alterations in the respiratory system anatomy and physiology during pregnancy are discussed below.

1. Hormonal changes in pregnancy produce hyperemia, mucosal edema, hyper secretion, mucosal friability, capillary congestion, tissue edema and hypertrophy of mucous glands of upper respiratory tract and airway.
2. Enlarging uterus displaces the diaphragm cephalad by 4cm, increasing AP and transverse diameter of thorax and chest wall circumference.
3. There is a progressive decrease in functional residual capacity (10-20% by term), mild decrease in residual volume, expiratory reserve volume and total lung capacity. Airway function and lung compliance are not affected.
4. There is an increase in minute ventilation and alveolar ventilation due to increased production of metabolic carbon dioxide and increased respiratory drive due to high serum progesterone level. Tidal volume increase by 30-35% and respiratory rate remains relatively constant.
5. Respiratory alkalosis seen in pregnancy is due to physiologic hyperventilation. Mild hypoxemia occurs in supine position and oxygen consumption increases by about 20-33% near term. Respiratory alkalosis becomes marked in active labour due to hyper ventilation and tachypnoea caused by pain and anxiety. This may affect fetal oxygenation by reducing uterine blood flow. In some, severe pain and anxiety lead to shallow breathing with alveolar hypoventilation, atelectasis and mild hypoxemia.

Observations on the respiratory causes of maternal mortality

As per the statistics (refer tables 4A.1) respiratory diseases were responsible for 6% of the maternal mortality during the years 2006 to 2009 (4 yrs).

There were a total of 21 deaths due to respiratory diseases among the cases (331) reported to CRMD.

There were a total of 331 deaths during 2006, 2007, 2008, 2009 (4 years in total)

Table 15.1

Year	2006	2007	2008	2009
Total deaths	109	59	79	84
Respiratory causes	9	1	2	9

80% (17 deaths) of deaths occurred during the third trimester.

Table 15.2 Stages of pregnancy when death occurred

1st trimester	2 nd trimester	3rd trimester	postpartum
2	1	17	1

The common age group was 20-30 yrs (95%- 20 deaths).

Table 15.3

Age group	Number
20-30 yrs	20
30-40 yrs	1

52% (11) of deaths occurred in primigravida.

Table 15.4

G1	G2	G3	Postpartum period
11	6	3	1

16 (76%) of the mothers had received Ante natal care.

Table 15.5

No of patients who received ANC	Not known
16	5

Deaths were equal among rural and urban living (11 and 10).

Table 15.6

Urban	Rural
10	11

Most of the deaths occurred in 10 days of admission to a hospital (81% 17).

Table 15.7

0-6 hrs	6-24 hrs	1-5 days	6-10 days	11-15 days	16-20 days	21-25 days	26-30 days	31-35 days	36-40 days
1	0	8	8	3	0	0	0	0	1

The important respiratory diseases included

Table 15.8

H1N1	5
Pneumonia	9
Bronchial asthma	4
ARDS	1
PAH	1
Pulmonary Embolism	1

Important co morbidities included

Bronchial asthma (5)

Gestational diabetes (3)

Pregnancy induced hypertension and hypothyroidism (2 each).

Associated co-morbidities in these patients were as follows

Table 15.9

Bronchial Asthma	5
Gestational diabetes	3
Pregnancy Induced hypertension	2
Hypothyroidism	2
Systemic hypertension	1
Acute fatty liver of pregnancy	1
Pre-eclampsia	1

Most of the mothers (19) were ventilated.

Ventilated versus Non ventilated patient

Table 16

Ventilated	19
Non-ventilated	2

No of patients who received Antiviral – 4 patients

Time of starting Antiviral

For 2 patients – before getting H1N1 report

For 2 patients – after getting H1N1 report

Patients who received Antibiotics – All 21 patients

No of patients where surgery was preceded by event of death – 8 patients

Important causes of death were

Sepsis (6),

Acute respiratory distress Syndrome (6)

Acute severe asthma (3)

Final cause of death	
Sepsis with MODS	6
ARDS	6
Acute Severe Asthma	3
Cor-pulmonale	1
HELLP	1
Respiratory failure	2
DIC	1

Important lessons to be learnt from these data and observations

As most of the deaths occurred in the 3rd trimester, pregnant women with any active disease in the 3rd trimester should be referred early to a tertiary care centre for better care. The disease which manifests or continues in the 3rd trimester should be more carefully managed.

Suggestions

Pneumonia and H1N1 infection were the chief respiratory diseases responsible for the maternal mortality. Retrospectively we feel that at least some of these cases would have been saved by adding antiviral drugs. Now it is almost a definite indication to start oseltamivir for patients diagnosed to have features suggestive of viral pneumonia (normal white cell count, C Reactive protein and procalcitonin). It is also very clear that lives can be saved if this drug is started early and in a higher dose of 150mg twice daily. We have saved a number of pregnant ladies with very severe H1N1 infection by starting the drug in the higher dose, very early and with a good supportive treatment with oxygen. Patients with bacterial pneumonia should be treated with aggressive antibiotic therapy if they worsen.

Many of the deaths could have been prevented if higher antibiotics were used at the correct time (e.g. Piperacillin tazobactam, meropenem or imepenem) If sepsis is continuing in spite of the use of higher antibiotics for adequate number of days, antifungal treatment should be added to the treatment. To start with fluconazole would be sufficient. If patients can afford higher and newer antifungal drugs (echinocandins like caspofungin and micafungin) they should be used (but only in 2nd and 3rd trimester).

Another important disease related to maternal mortality was bronchial asthma. Currently the mainstay of treatment is aerosol treatment. Drugs like deriphyllin and aminophyllin are usually not used. The drugs of choice include levosalbutamol or fluticasone / Budesonide. These drugs should be given by frequent nebulizations especially when the patient is having acute severe asthma.

Pulse oxymetry and ABG (arterial blood gas) should be made more frequently available. There should be more use of NIV (Non Invasive Ventilation) so that mechanical ventilation can be avoided or postponed.

One patient had a diagnosis of pulmonary embolism. She had a history of preeclampsia. A diagnosis of thrombophilia is a possibility but complete investigations were not done. If such a diagnosis of thrombophilia was reached then, this patient could have been benefited by heparin thromboprophylaxis preventing the occurrence of thromboembolism.

Another patient had an uncontrolled hyperglycemia which had a definite role in the mortality. Ideal method of treating such uncontrolled hyperglycemia is by insulin infusion. If such facility is not available it may be worthwhile giving an infusion of normal saline with about 50 units of rapidly acting insulin in it. A close and frequent monitoring of the blood sugar can be done to normalize the sugar as early as possible.

Most of the patients who died due to respiratory diseases were ventilated. ABG (arterial blood gas) testing could not be done frequently in most patients. Ventilator setting and management should be by an expert to yield optimum results.

Early reference to a higher tertiary centre and early intervention by LSCS should be stressed.

If some of these points are remembered and some of these recommendations are executed the maternal mortality could be definitely decreased.

Patients with respiratory disease co morbidity (e.g. bronchial asthma) should plan their pregnancy. There should be a good respiratory assessment and proper and adequate management of the respiratory disease before becoming pregnant.

Conclusions

Respiratory causes contribute significantly to maternal death. Viral pneumonia (H1N1) is more dangerous in the third trimester. Aggressive approach to control the infection and provide respiratory support results in better outcome. Those with pre existing conditions like bronchial asthma should seek medical advice before embarking on pregnancy. When respiratory function is compromised, in the third trimester, termination of pregnancy should be considered as it will improve respiratory function.

Chapter 16

Severe Sepsis

Sareena Gilvaz, M. Bindu

Introduction

It is imperative that all practising obstetricians should understand basic infectious disease concepts to treat infected patients appropriately. More so, since the frequent source of organisms that cause post partum or post operative infections among women is the vagina which harbours a large variety of both aerobic and anaerobic organisms. This exists normally in a symbiotic relationship and often contributes to a large part of obstetric sepsis when the situation arises.

As we analysed the four years of maternal mortality between 2006- 2009 there were 32 cases of sepsis out of the total 331 maternal deaths reported to CRMD. Of these 23 were following cesareans; only two following normal vaginal delivery; three died undelivered and four following abortions, though none was due to septic / induced abortions. One was instrumental delivery.

The time of death ranged from 2 days after the event to up to 30 days. And 20 of them died within a week which makes us believe that most of them were very fulminant infections.

Their obstetric score was as follows -

- gravida 1 - 17
- gravida 2 - 10
- gravida 3 - 4
- unknown - 1

In many instances the contributing factors were prolonged inductions, withholding effective antibiotics after prolonged rupture of membranes, delayed decision making to do cesareans, hesitancy about a hysterectomy in the presence of severe sepsis with peritonitis, and the conditions needing relaparotomies. So probably we have to question ourselves as to why we are so relaxed in diagnosing and treating sepsis when we know that pregnancy and the immediate puerperium are

really immuno compromised states and sepsis takes a fulminant course under these circumstances. We think being aware of sepsis and a more aggressive attitude from us could possibly avoid many a maternal death.

Few case scenarios and lessons learnt from them

Example 1.

31 yr old, G2 A1 at 38 wks gestation had undergone emergency LSCS for severe pre eclampsia and IUGR with fetal distress, in a peripheral hospital. No mention of any intraoperative events. Next day she developed giddiness, hypotension and abdominal distension. Immediately relaparotomy was done. No hemo-peritoneum. It was a negative laparotomy and abdomen closed with a drain. Soon after relaparotomy she went into DIC and shock with an elevated PT of 125sec. Three units of blood was given and patient was referred to a higher centre.

On admission at the referral hospital, patient was drowsy, in shock, with severe pallor and features of DIC- like bleeding gums, wound site bleeding etc. Resuscitated with blood and blood products. Higher antibiotics started (Inj. Ceftriaxone and Inj.Metrogyl). She continued to be tachypnoeic, febrile, with abdominal distension and absent bowel sounds. For the next two days daily 2 units blood and 2 bags FFP were given. Antibiotic was changed from ceftriaxone to Inj.Piptaz. On the third post relaparotomy day, she developed dyspnoea along with persistent fever and was diagnosed as ARDS with features of renal compromise. She was intubated and ventilated. There was no improvement in the DIC parameters in spite of repeated daily blood and blood component therapy! On the 5th post LSCS day, she had cardiac arrest and died. Total 10 units of blood, 16 bags of FFP and three units of platelets were given.

Diagnosis: Severe pre eclampsia, IUGR, fetal distress – emergency cesarean – negative relaparotomy – DIC and shock – septicaemia – ARDS and renal failure – MODS

Learning points

- Intraoperative details are missing in the records
- All post operative abdominal distensions need not be due to internal bleeding. Confirm with scan or paracentesis before relaparotomy.
- Relaparotomy of a sick patient is a serious step. Anesthesia and surgery are poorly tolerated.
- The aggressiveness needed regarding antibiotics and use of blood components is not seen in the management –“Two units per day” is not the way to replace coagulation factors.

- Closing the abdomen with a drain in a patient with DIC was a good step which is appreciated.

Example 2.

27 yrs old G2 A1 at 37 wks gestation with gestational hypertension on tablet nicardia 10mg twice daily and long period of infertility (married life 9yrs). After admission, BP was fluctuating between 90-100 diastolic and all PIH investigations were normal. Labour was induced with PGE₂ once at 3.30pm for an unfavourable cervix. 7 hrs later cervix became favourable and hence ARM was done - clear liquor. 6hrs after ARM cervix effaced 2 finger loose, small caput, head -3 and pelvis normal. Decided for emergency LSCS (why?) in view of failed induction. A live male baby weighing 2.45kg with 9/10 Apgar. Postoperatively started on Inj.Taxim 1 gm Q12hrly. 36 hrs after LSCS she developed abnormal behaviour and was given Inj.serenace. At that time all vitals were normal. Uterus well contracted with normal bleeding per vagina.

On forth post operative day, she developed abdominal distension and breathlessness. On examination she was tachypnoeic with tachycardia, afebrile. Bowel sounds were present but sluggish. Consulted a physician and shifted to MICU. CT chest was done to rule out pulmonary embolism (CT was Normal). Intravenous antibiotics were stepped up with inj.metrogyl and Inj CP along with Inj.taxim. Within a few hours she went into shock. Intubated and dopamine drip was started. USG abdomen showed gross ascites. Paracentesis showed purulent fluid.

Immediate relaparotomy done. About 1½ L of fecal peritoneal fluid removed. Rent on terminal ileum was identified and about 8 cm ileum was resected. Uterus and tubes were normal. Blood and blood products were given. Post operatively shifted to MICU for ventilator support. She developed cardiac arrest in MICU. Condition worsened and expired on the same day (4th postoperative day)

Lessons to learn

- Mechanical ripening of cervix prior to prostaglandins makes the induction process smooth and fast. Starting prostaglandin straight away at 3.30pm was not a wise thing.
- Exactly when and how the bowel injury occurred is difficult to know. The most likely time is while opening the parietal peritoneum; all the more in cases of previous surgery.
- Before prescribing antipsychotics in the postoperative period, serious morbidities especially ones leading to hypoxia should be ruled out.

Example 3

22 yr old primi, had delivery by LSCS (Indication not known). Notes say, she had wound discharge from the 4th post operative day. She was diagnosed as having pelvic collection but relatives refused any further treatment and took her home.

9 days later she reported to the same hospital with fever, vomiting, pain abdomen and pus discharge from wound. On examination she was tachypnoeic and had tachycardia. Hb was 6.7 gm% with leucocytosis .Broad spectrum antibiotics (Inj genta, metrogyl & Ceftriaxone) was started .Repeat USG after one day of admission showed bulky uterus with evidence of pelvic abscess. Two days later she underwent laparotomy with drainage of pelvic abscess, wound had to be extended upwards to breakdown adhesions between bowel loops. Post op. antibiotics were changed to inj.amikacin instead of inj.gentamicin.

She showed good improvement after relaparotomy and was shifted to the ward. Antibiotics were stopped .On seventh post operative day following laparotomy she developed sudden gasping and became unconscious. In spite of resuscitative measure and CPR she could not be revived and declared dead within two hours. The presumed final cause of death is pulmonary embolism.

Points to ponder, lessons to learn

- Before discharge from the hospital on the 4th postoperative day ultrasound guided aspiration or colpotomy with antibiotic would have taken care of the infection. She needed a much bigger incision subsequently.
- This illustrates the importance of thromboprophylaxis in post cesarean patients especially the ones who were not ambulant.

Example 4

21 yrs old G2 A1 at 34 wks gestation referred as severe PE, treated with alphasopa, nifedipine, prophylactic MgSO₄ and Inj. betnesol. One day after admission she developed impending symptoms and decided on termination with PGE₂ gel 2 doses 6 hrs apart. After 48 hrs, emergency LSCS was done for severe PE with failed induction. Delivered 1.3 kg baby with normal Apgar. Three hours after cesarean section excessive oozing from the incision site - controlled after wound exploration.

On first postoperative day she developed fever and cough with crepitations. Second postoperative day she showed abnormal behaviour. Suspected toxic encephalopathy and broad spectrum antibiotics started. CT scan head was normal. On fourthth postoperative day she had loose stools and abdominal distension with peritonitis. USG confirmed ascites with echogenic particles and multiple septations.

After surgical consultation, only aspiration of ascites done. General condition of the patient worsened and decided for relaparotomy on 10th postoperative day. Peritoneal cavity contained one litre of pus which was drained and peritoneal toileting done. Uterus and adnexa appeared normal. Post operatively she was managed in ICU with broad spectrum antibiotics. Total 7 bags FFP and 2 units blood given. Her condition deteriorated day by day associated with disorientation and neck stiffness. She was on ventilatory support and died 6 days after relaparotomy.

Diagnosis: Postoperative peritonitis, septicaemia, ARDS, meningitis, ARF - MODS

Points to Ponder

- Prolonged induction of 48 hrs before taking decision for LSCS for severe PE with IUGR (wt.1.3kg) is questionable.
- After evidence of peritonitis, patient was on conservative management for five days before they decided on a relaparotomy. Earlier intervention would have made a difference.
- At laparotomy they have done only peritoneal toileting; a hysterectomy, removing the source of infection would have been more appropriate.
- When treating severe sepsis we must go all out in our management. A half heartedness has dire consequences - like it happened with this patient.

Example 5

32 yr old P2 L2 had undergone emergency LSCS at 39wks; indication previous LSCS in labour with unengaged head. Post operatively she had fever and treated with only ampicillin and was discharged on 7th post op day with a temperature of 100^oF. The next day (8thPOD) she was readmitted with high-grade fever, chills, dyspnoea, yellowish urine and oliguria. Total bilirubin was 7.5mg %. On admission BP was 86/66 mm Hg, dyspnoeic with abdominal distension. Two units blood and three bags of platelets were transfused. Broad spectrum antibiotics were started. Ultrasound Scan abdomen showed bulky uterus with mixed echogenic area 4.6 x 3cm on anterior uterine wall, ? focus of sepsis. There after she continued to be oliguric (urine output 250ml), drowsy with abdominal distension and sluggish bowel sounds and also on dopamine drip. Diagnosed as ARF with sepsis (bl.urea 84, S.Creatinine 3, Total bilirubin 8mg% with platelets 50000). Blood culture showed pseudomonas. Hemodialysis done on alternate days (total 10 dialysis). Antibiotics changed to cefaperazone and Vancomycin. Ten bags FFP and three bags platelets were transfused along with hemodialysis. Initially she showed improvement but later developed acute dyspnea and severe hypoxia therefore connected to ventilator. Antibiotics stepped up with Inj.Imepenum. But she deteriorated and developed cardiac arrest.

Points to Ponder

- Pseudomonas septicemia is associated with very severe consequences.
- Ampicillin seems to have been the only antibiotic she received. For post-operative sepsis, this is inadequate.
- The aggressiveness regarding antibiotics expected in a case of severe sepsis is not seen in the management of this patient.

Example 6

25 yr old, P2 L2 at term had instrumental delivery and later referred as having PPH. She was diagnosed as having inversion uterus which was corrected by hydrostatic method. On admission she was drowsy, tachypnoeic and in shock. She was intubated and resuscitated with dopamine drip. Her Hb was 6.2gm%. 5 units blood and two bags FFP were given. From second postnatal day onwards she developed high grade fever and hence broad spectrum antibiotics was started.

Even after 12th postpartum day she continued to have high grade fever but all investigations were found negative. Her uterus was 16 wks size with evidence of subinvolution. On 15th postnatal day she was referred to medical ward for the management of fever. 15 days later, (30th postnatal day) she underwent abdominal hysterectomy for sepsis and subinvolution of uterus. During surgery omentum and ovaries were found adherent to uterus. Uterus on cut section was foul smelling .Antibiotics changed to Inj.taxim, Inj.Clindamycin and inj.CP. The next postoperative day she had dyspnoea, sudden convulsion and cardiac arrest.

Lessons to learn

- *The team that diagnosed and corrected the inversion with hydrostatic method is to be congratulated..*
- *When fever persisted, the possibility of sepsis in the uterus was to be considered.*

Over View

One of the most important findings in critical care medicine in the last 2 decades is the prominent role played by the inflammatory response in the morbidity and mortality associated with severe sepsis and septic shock.

The inflammatory response of the body is initiated by some insult which could be in the form of a microbial invasion, (eg. infection) trauma etc. The systemic inflammatory response appears in the form of fever, leucocytosis, tachycardia etc and this is called the systemic inflammatory response syndrome (SIRS).

Diagnostic criteria for SIRS requires at least two of the following

1. Temperature $> 38^{\circ} \text{C}$ or $< 36^{\circ} \text{C}$
2. Heart rate > 90 beats /mt
3. Respiratory rate >20 /mt or arterial $\text{PCO}_2 < 32 \text{ mm Hg}$
4. WBC count $> 12,000/\text{mm}^3$ or $< 4000/\text{mm}^3$ or $>10\%$ immature band forms.

To simplify the whole scenario

An initial inflammatory reaction is acceptable but, when the inflammatory response by the body is excessive and overwhelming, it causes tissue damage of the host, ultimately leading to organ dysfunction. This increased inflammatory response affects the organs of the host leading to multi organ dysfunction called MODS.

The Nomenclature of Clinical syndrome associated with sepsis are

1. **SIRS** - Initial signs of systemic inflammation (eg leucocytosis , fever etc) is called systemic inflammatory response syndrome (SIRS).
2. **SEPSIS** - When SIRS occurs following an infection, it is called sepsis.
3. **SEPTIC SHOCK** - Severe sepsis with refractory hypotension is septic shock.
4. **MODS** - Severe Sepsis accompanied by two or more vital organ dysfunction is called multiorgan dysfunction syndrome (MODS) leading to multi organ failure.(common organs affected being the kidney, lungs, CVS and CNS)

Organs or systems involved in sepsis are as follows

1. In the Lungs – it causes acute respiratory distress syndrome
2. In the Kidney – It causes acute tubular Necrosis
3. Cardio vascular system - Hypotension
4. Central nervous system - Metabolic encephalopathy
5. Coagulation systems – DIC
6. Gastro intestinal Tract – Paralytic ileus
7. Liver – Acute hepatitis
8. Adrenal glands – Acute adrenal insufficiency etc

Pathogenesis of sepsis

Infection which is the initial insult leads to SIRS and overwhelming inflammatory response. Activated neutrophils adhere to surface of endothelial cells causing endothelial damage. Tissue parenchyma will be infiltrated with inflammatory mediators. MODS and multi organ failure ensue. Thus MODS is an inflammatory injury to vital organs. When four or more vital organs fail, there is 80% mortality.

Common pathogens

Most commonly isolated organisms are the gram negative pathogens such as E.coli , Klebsiella, Pseudomonas aeruginosa

Haemodynamic alterations

Initial stages of severe sepsis and septic shock is often characterised by

1. Hypovolemia and hypoalbuminemia
2. Defect in the peripheral extraction of oxygen ie, decreased O₂ uptake from the micro circulation.
3. Hyperlactemia due to impaired pyruvate metabolism.

Graded manifestations of obstetric sepsis

1. Wound discharge
2. Unhealthy lochia
3. Parametritis
4. Pelvic abscess
5. Peritonitis
6. Septicaemia
7. DIC
8. Septic shock
9. MODS

Management of severe sepsis (10 point management)

1. Fluid replacement therapy.
1000ml of crystalloids (NS, RL) in 30mts, then 500ml every 30mts or 300 - 500ml of colloids in ½ hr(5% albumin, voluven etc), So as to maintain
 - a) MAP> 65mm Hg
 - b) Central venous pressure 8-12 mmHg
 - c) Urine output 30ml /hr

2. Take early blood culture prior to antibiotic administration, then start parenteral broad spectrum antibiotics as early as possible (can start with the highest broad spectrum, then step down when culture reports come).
3. Maintain central venous oxygen saturation > 70%
4. Check serum lactate levels (if > 4mmol /L. they are at risk of septic shock).
5. Vasopressors: Use any one of the following as required and according to the case scenario. Vasopressors are to be used only after adequate fluid replacement.
 - a) Dopamine 3-10 micro gram/Kg /min. Dopamine dose to increase cardiac output and cardiac contractility. For a 50kg patient, she requires 500microgram/mt or 0.5 mg/mt. Dose of dopamine is 200 mg (1 amp) in 500 ml of normal saline. 1 ml delivers 0.4 mg or 400microgram. 50-60kg patient will require 16 – 25 drops /mt
 - b) Norepinephrine 0.2-1.3 microgram /kg/min (produces vasoconstriction and no increase in cardiac output. Often preferred)
 - c) Vasopressin is used when there is refractory hypotension. Dosage is 0.01-0.04 units /min
 - d) Dobutamine when there is a cardiogenic component.
6. Packed cell transfusion as and when required(eg: if the Hb is <8gm%)
7. Low dose steroids - 50mg I V x 6 hourly x 7days (ie hydrocortisone 200mg/ day) .This is used now a days to overcome the adrenal insufficiency seen in sepsis. This is a recent concept.
8. Adequate glycemic control. Maintain glucose >80 but<150mg%. Thus glucose and insulin to be given accordingly but watch out for hypoglycemia.
9. Use mandatory ventilatory modes (for lung protection).
10. Optional Recombinant activated protein C. Protein C is an endogenous anticoagulant with anti inflammatory property. Should be given early enough and not at an extreme stage.

Supportive therapy along with the above

1. Sedation /analgesia depending on the situation eg: Postoperative sepsis with pain.
2. Renal replacement therapy (dialysis)depending on blood chemistry. Avoid renal toxic drugs like amnioglycosides. Also decrease intake if patient is in early renal failure.
3. Bicarbonate therapy - depending on serum electrolytes.

4. DVT prophylaxis not to be forgotten
5. Stress ulcer prophylaxis must be added to the prescription
6. Blood component therapy if in DIC

In most cases of severe sepsis whether patient is with or without ventilator, we must ask for the ABG.

Antibacterial & Antibiotics

Bacteria commonly responsible for female genital tract infections:

Aerobes	Gram negative
	E Coli -
	Klebsiella
	Proteus
	Pseudomonas
	Gram positive
	Streptococcus group ABD
	Staphylococcus aureus
	Staphylococcus epidermidis
	Enterococcus
Anaerobes	Peptostreptococcus
	Peptococcus
	Clostridium
	Bacteroides

We commonly encounter during PROM and prolonged labour, the group B streptococcus, Ecoli, Klebsella etc causing chorioamnionitis and for these we can give a combination of ampicillin, gentamicin and metrogy1. But in fulminant sepsis we need to know which antibiotic covers which organism.

1. Penicillins

The effectiveness of these drugs is reduced because of beta lactamase activity of certain bacteria. To counter this, we can combine these drugs with sulbactam or clavulanic acid. Cloxacillin is good against penicillinase producing bacteria. Some drugs can be combined to increase their spectrum of activity, eg:Piperacillin, Tazobactam.

Antibiotics and their coverage:

1. Ampicillin – Mostly gram +ve staph & streptococci and enterococci
2. Amoxycillin –Close congener of ampicillin. Better oral absorption with less incidence of diarrhoea

3. Cloxacillin –good against penicillinase producing staph
4. Piperacillin –good gram negative coverage. It has good antipseudomonal plus good activity against klebsiella also

2. Cephalosporins

1st Generation - cefazolin used in surgical prophylaxis.

2nd Generation– not commonly used. Hence not being mentioned here.

3rd Generation include

- a. Cefotaxime potent action against gm –ve. Some gram +ve activity. But no action on pseudomonas and staph.
- b. Ceftriaxone – same as above . Also used in complicated UTI, skin and soft tissue infections. Also in abdominal sepsis and septicemia.
- c. Ceftazidime - highly active against pseudomonas.
- d. Cefaperazone - Active against pseudomonas. Also used in severe UTI, skin and soft tissue infection and septicemia.

Monobactam: Aztreonam used in severe septicemia, but not effective against gram positive organism.

Carbapenems: Imipenam has the broadest spectrum of activity.

Meropenam has a coverage against most aerobes and anaerobes. Used only as a reserve drug. This drug should not be abused.

3. Aminoglycosides

I. Gentamicin – Most commonly used broad spectrum gram negative action against klebsiella, E.coli, Pseudomonas, Proteus and Enterobacter.

But has no action on streptococci and anaerobes. Can be combined as follows

1. Gentamicin with Cephalosporins (1st,2nd or 3rd generation) and metrogyll
Or

2. Gentamicin with Piperacillin & salbactam & metrogyll

Thus giving a broader spectrum of activity against both gram positive and gram negative bacteria.

II. Amikacin Wider spectrum of activity. May act when resistant to gentamicin

III. Tobramycin Identical to gentamicin. Reserve alternative for gentamycin and nephrotoxicity less than gentamicin.

4. Fluroquinolones

The earlier quinolones are the ciprofloxacin .The newer ones are levofloxacin ,gatifloxacin, moxifloxacin. Rapidly emerging resistance to these antibiotics is caused by excessive use. It has reduced the value in treating serious gram negative infections particularly those due to pseudomonas aeruginosa. Therefore we must keep the higher antibiotics only for serious issues such as life threatening situations.

If colonization with Methicillin Resistant Staphylococci Aureus (MRSA) is documented, give linezolid. The emergence within hospitals of MRSA as well as VRE(vancomycin resistant enterococci) is a very worrying trend now a days.

When to stop antibiotics in sepsis

1. When fever free for 48 hrs.
2. Or when she completes the course.

Prophylactic antibiotics – what is its role

Peri operative anti microbial prophylaxis has reduced the risk of infections complicating surgical procedures. Infection rate is lowered more if the prophylactic drug like cefazolin is given prior to skin incision. But remember this is applicable only if it is a planned procedure and a clean case.

Sepsis and renal failure

Renal function is affected in most cases of severe sepsis .A general idea about this will help us in managing a patient with severe sepsis and renal failure .

Acute Renal Failure(ARF) occurs in approximately 19% of patients with moderate sepsis, 23% with severe sepsis, and 51% with septic shock when blood cultures are positive. A combination of ARF and sepsis is associated with a 70% mortality as compared with 45% mortality with ARF alone.

ARF definition

A sudden and usually reversible decrease in GFR occurring over a period of hours to days. ARF is now known as Acute Kidney Injury (AKI). ARF is classified as pre renal, renal and post –renal.

In sepsis it is often pre renal to start with .Pre renal azotemia is a fall in GFR due to decreased renal perfusion secondary to a severe deficit in blood flow in which there is minimal or no structural or cellular damage to kidney. It is a haemodynamic condition where the renal tubular function is normal. But acute tubular necrosis ATN is

characterised by renal tubular dysfunction. Septic shock produces a pre renal type of ARF (following severe hypovolemia, blood flow to renal cortex decreases but renal medullary perfusion is maintained). Cortical ischaemia produces marked decreases in GFR, concentrating ability and urine volume. This stage of severe renal functional impairment is called pre renal ARF. If cortical hypoperfusion persists, the functional impairment rapidly progresses to intrinsic renal damage i.e. ATN or renal cortical necrosis. (i.e. Kidneys lose its concentrating ability and to preserve sodium).

Oliguria: is urine output <400ml in 24hrs

Anuria: Is No or < 100ml urine output in 24hrs

Causes of ARF in pregnancy

Early pregnancy – hyperemesis, dehydration

Septic abortion

Late pregnancy – pre eclampsia and sepsis

Patients with pre renal ARF respond to inj. frusemide but not those with renal ARF.

Clinical features

When the anuria is reversible, the clinical condition is divided into 3 phases

1. Incipient phase (Time of insult to onset of oliguria) – Here marked decrease in Urine output occurs
2. Phase of oliguria /anuria – can persist from a few hrs to 3 wks
- low urine out put - Patient looks drowsy, anorexic, vomiting, abdominal distension, dry furred tongue
3. Phase of diuresis – due to delayed tubular reabsorption. Output will be very high. Therefore replace fluid accordingly and correct electrolyte imbalance also in this phase

Investigations:

Diagnosis is by clinical judgement. Investigations may support the diagnosis.

1. Blood for S. electrolytes, Osmolality BUN, S. Creatinine, TC, DC (shows leucocytosis), LFT, platelets
High sodium > 145 meq/L (N 136-145)
S.K > 5meq/L (N = 3.5-5)
S.Cl >106 meq/L (N= 100 -106)
HCO₃ <24meq/L (N=24-32)

- B.Urea >25mg % (N 20-25)
2. ECG – In hyperkalemia-tall peaked 'T' Waves , absent P waves, prolonged QRS complex to 0.2sec
 3. Urine for sodium , creatinine, osmolality & specific gravity – more of theoretical importance than practical application
 4. Blood culture and urine culture to rule out sepsis
 5. USG to rule out obstetric causes

Management

Management of ARF

Pre renal ARF usually responds well to adequate treatment. That is why initially fluid replacement is required.

1. Intravenous fluids, blood & blood components depending up on the situation. (Maintain CVP between 10-15 cm of H₂O. Increased CVP indicates ATN or acute cortical necrosis. Therefore fluids must be restricted. Low CVP indicates hypovolemia. Therefore increase IVF (1000ml in Ist 30mts & 500ml /hr) to maintain normal CVP and urine output >30ml/hr)
2. No role for low dose dopamine in ARF.
3. Deliver the obstetric patient with oliguria or anuria

Management of ATN

1. Deliver the patient.
2. Restrict fluid intake to the amount equal to urine output and insensible losses.
3. Adjust the dose of renal toxic drugs, e.g.MgSO₄, Inj.Gentamycin, Inj.Amikacin
4. Haemodialysis when conservative management fails

Indications for DIALYSIS are as follows:-

Depends upon the entire clinical picture and not on the presence or absence of certain factors.

1. Fluid overload during oliguric phase of ATN
2. Hyperkalemia (S.K >6 meq/L)
3. Severe alterations in plasma sodium concentration
4. Pericarditis
5. Uraemic encephalopathy
6. Metabolic acidosis (HCO₃ <13 meq /L)
7. Prophylactic when BUN >90-100 or bl.urea >180mg or S. creatinine >8mg% \ see in which situation the patient falls and treat accordingly

Management of incipient phase of anuria

Restore the intravascular volume.

Forced diuresis with Inj.frusemide 80-120mg IV

Management of oliguric and anuric phase

Control fluid intake

Nutrition 2000 kcal /day

Electrolyte to be balanced

Haemodialysis

Supportive therapy

Management of Hyperkalaemia

1. Restrict dietary K⁺ intake (avoid fruit juices)
2. Avoid K⁺ sparing diuretics
3. Inj. Dextrose 50ml 50% IV along with regular insulin 10units s/c (to promote K⁺ migration from extracellular to intracellular compartment .Repeat after 2-4hrs)
4. Increase carbohydrate intake
5. Ca gluconate 10% of 10ml slow IV. (To decrease cardiotoxic effect of potassium)
6. Potassium binding exchange resins for 1-2days (It exchanges potassium for sodium from blood and intestinal secretions.)
7. Haemodialysis if conservative measures fail.

Sepsis Prevention

The following are predisposing factors to obstetric sepsis and hence be careful.

1. Pregnancy with PROM or PPROM
2. Prolonged labour
3. Multiple vaginal examinations
4. Traumatic delivery
5. Manual removal of placenta
6. LSCS
7. PPH
8. Associated anaemia /mal nutrition

To prevent sepsis in the labour suites, the following steps will help.

1. Change of clothes - dedicated delivery gowns for patients in labour room
2. Prior bath with special attention to feet ,abdomen, groins and perineum

3. Labour room to get the status of an operation theatre with regular cleaning.

To aid this

- a. Preferable to have tiled washable floors and walls.
 - b. Cleanable labour cots .eg steel cots.
 - c. Mopping floor with disinfectants after every case
 - d. At least once a week wash labour room with soap and water
 - e. Use of dedicated autoclaved separate sets for each delivery case.
 - f. Regular cleaning of suction tubes and oxygen mask
 - g. Proper hand washing of medical attendants initially with soap and water followed by disinfectants.
 - h. Elbow taps in the wash area.
 - i. Initial cleaning of all instruments with bleaching powder 1tbsp (15 gm)in 1litre water.
4. Proper cleaning of perineum with betadine prior to delivery and abdomen prior to CS.

How to contain sepsis

1. Hospital infection control committee (HICC) to be mooted in every institution.
2. Have a common antibiotic policy.
3. Follow aseptic precautions and insist that all people working in the labour room follow this.
4. Limit number of invasive manipulations including vaginal examinations.
5. Good surgical techniques , gentle handling of tissues, adhering to good haemostasis and usage of prophylactic antibiotic only in elective planned surgical procedures.

Conclusion

Sepsis is emerging as a major contributor to maternal mortality. Feeling of security because of availability of antibiotics is dangerous. Bacteria resistant even to the most potent and newest antibiotics have emerged. Strict antibiotics policy, aseptic practices and vigilance to pick up sepsis early are essential to minimise sepsis deaths.

Chapter 17

Early pregnancy problems

T. Narayanan

T. Jayandhi Raghavan

Contrary to the previous clinical scenarios only few of the complications of first trimester lead to maternal mortality. Gone are the days when maternal mortality occurs due to hyperemesis gravidarum. The distant aftermath of vesicular mole when mortality may occur due to malignant transformation is also very less. Thanks to the various imageology techniques, ectopic pregnancy can be diagnosed early. However, maternal mortality due to ectopic pregnancy has not declined proportionately. The malady presented by septic abortion is still a nightmare in many of the rural areas where quacks galore. The following acumen mainly revolves around maternal mortality ensuing from ectopic pregnancy and septic abortion.

Ectopic Pregnancy

Ectopic pregnancy is a life threatening situation in early pregnancy and is one of the important causes of maternal mortality and morbidity during first 20 weeks of pregnancy.

Ultrasonography has revolutionized the management of tubal ectopic pregnancy. Early diagnosis allows medical treatment to be the first line choice in appropriate cases. Timely diagnosis and treatment can reduce the maternal mortality and 'severe acute maternal morbidity' (SAMM) commonly known as a 'near miss case', due to this life-threatening situation.

In the first report of CRMD Kerala 2004-05, six deaths occurred due to ectopic pregnancy which comes to about 1.9 %. These were either due to late diagnosis with extensive intraperitoneal bleed or missed diagnosis.

During the period 2006 – 09 in Kerala there were three maternal deaths due to ectopic pregnancy out of 331 maternal deaths analyzed.

Key Recommendations:

1. Ectopic pregnancy remains the leading cause of early pregnancy related death.
2. Tubal pregnancy accounts for 90-95 % of all ectopic pregnancies.
3. Early and correct diagnosis of ectopic pregnancy may not be made always clinically.
4. All sexually active women with a history of lower abdominal pain and vaginal bleeding should be offered ultrasonography and urine pregnancy test to exclude ectopic pregnancy.
5. Suspected cases should be hospitalised till ectopic pregnancy is ruled out. A combination of TVS and beta hCG determination is the best approach for early diagnosis of ectopic pregnancy and may often need to be repeated.
6. Early ultrasonography should be available in subsequent pregnancies for women who have had an ectopic pregnancy.
7. Timely diagnosis and treatment of ectopic pregnancy can reduce the maternal mortality and 'severe acute maternal morbidity'.
8. Women with suspected ectopic pregnancy shall be managed in centers where there are facilities for emergency surgery.
9. In the hemodynamically stable patient, a laparoscopic approach is better than open approach.
10. In cases with shock immediate laparotomy with simultaneous resuscitation will be life saving.
11. Medical management using methotrexate should be considered in suitable cases.

General observations: The incidence of ectopic gestation during the review period is 0.91%. There is a reduction in the incidence of ectopic pregnancy compared to that of CRMD 2004 -05(1.9%). This may be due to increased use of ultrasound in early pregnancy.

Clinical presentation:

Three risk factors that increase the likelihood of tubal rupture include ovulation induction, serum beta hCG level exceeding 10000 IU/L when ectopic pregnancy is first suspected, and h/o never having used contraception. Appreciation of these risk factors aids timely diagnosis and prompt surgical intervention.

There may be difference between an acute and a chronic ectopic. Acute are those with high beta hCG and carry highest risk of rupture compared to chronic variety which demonstrates static serum beta hCG.

Morbidity and mortality associated with ectopic pregnancy are directly influenced by the time interval between the onset of symptoms and start of treatment.

Septic abortion

Fortunately septic abortion was not found as cause of death in the cases reported to CRMD. However all practitioners are cautioned to take all aseptic precautions while dealing with early pregnancy termination.

With the availability of medical methods, more women go for it and has always been safer as D&E may not be needed in many of the situations. In India medical termination is approved upto 49 days of gestation, though WHO recommends up to 63 days.

The diagnosis of the septic shock is not a difficult one. In the case of septic abortion physical evidence of cervical manipulation and trauma may be present, the cervix may still be dilated and products of conception may be still traceable in the vagina. On examination cervix and uterus are tender to touch, and purulent discharge may be present .

Before initiating therapy, appropriate cultures for aerobic and anaerobic bacteria should be obtained from the involved organ and also blood cultures sent . Therapy should consist of effective cardiovascular monitoring, fluid resuscitation , effective antibiotic therapy besides dilatation and evacuation of the remaining products. If peritoneal signs are present, perforation must be suspected and laparotomy/Laparoscopy undertaken. Also important is the anticipation of concomitant multiple organ system involvement such as ARDS, Acute Renal Failure and hepatic insufficiency.

Outcome in septic shock depends in large part upon the presence of underlying risk factors such as nutritional inadequacy and presence of systemic diseases such as hypertension and diabetes.

Key recommendations

- Every centre should have an antibiotic policy
- Antibiotics are not the substitutes for strict aseptic practices. Updating and reorientation regarding infection prevention and antibiotic use is mandatory to all the staff caring for obstetric patients.

- Medication abortion is the better option for terminating early pregnancies.
- A manual vacuum aspirator (MVA) is useful in early pregnancy terminations
- An ultrasonogram is mandatory in suspected incomplete evacuations.
- Careful counseling is required to encourage for follow up and use of proper contraceptive methods.

Chapter 18

Less Common Causes of Maternal Deaths

Dr.Sumangala Devi

Among the less common causes of maternal deaths reported to CRMD 2006-09 there were

- Five cases of suicide
- Four cases of postpartum collapse
- Two cases of anemia
- Four cases of accidental burns
- Two case of anaphylaxis
- Two cases of MTP complication
- Two cases of malignancies (other than hepatic)
- One case of accident
- One case of HIV
- One case of viral fever
- One case of obstructed labour
- One case of ovarian cyst rupture
- One case of perforation of stomach

For most of these causes, there were cases reported to the director of health services as well.

Suicide

In the CRMD review 2004-05 there were six cases of suicide. Statistics from DHS reveals that nineteen women committed suicide during 2006 -09. Only five cases came to the attention of CRMD. The problem of suicide and its significant contribution in the Kerala scenario is a matter of concern. It appears that lack of family support, due to the present nuclear family trend could be one of the contributory factors. The obstetricians can do very little to prevent this unfortunate

event other than try and pick up mothers who look depressed during their antenatal visits. By counseling them and treating with the help of a psychiatrist we might be able to bring them back to normal life.

Postpartum collapse

There were four cases of postpartum collapse. We assigned this as a cause when complications like PPH, AFE, inversion uterus etc were excluded and where no other cause of collapse could be considered. Effective and timely resuscitation of a collapsed patient will definitely make a difference in the outcome. All practicing obstetricians should undergo training in basic resuscitation skills. KFOG has already started the training as EMOCALS and we hope that all obstetricians enroll and acquire skills in emergency resuscitation to save lives in a collapsed patient.

Anemia

There were a total of four cases of anemia leading to death but only two came to our review. We have included only those cases where anemia was the primary cause. There is no doubt that very low hemoglobin level would have contributed to the bad outcome. This reflects the inadequacy of antenatal care. One of the cases was a tribal woman with 8months pregnancy admitted with severe anemia and CCF. She went into preterm labour and had sudden death after delivery .This has happened inspite of the fact that there are many arrangements made by the government to give them physical as well as financial assistance for treatment. There is a need for educating the tribals and their support group to encourage effective utilization of such resources.

Burns

There were six cases of burns including two cases of lightning in this series, of which four burns came to the CRMD. Careful nursing in a special ward, good antibiotic coverage and general supportive management are mandatory. The final outcome depends on the degree of burns.

Anaphylaxis

There were two cases of anaphylaxis reported to CRMD. One followed injection fortum and the other after injection cefazolin. Obstetric patients in labour receive IV medications and are at risk of anaphylaxis. Careful history taking at the booking visit should inquire about any history of anaphylaxis in the past and record that information prominently on the antenatal card. Very often there may not be a suggestive past history. It is therefore ideal to give a test dose of the drug

before giving a full dose. This again points to the fact that immediate administration of adrenaline and resuscitative measures can save life.

MTP complications

Two out of three deaths in the review period due to complications of MTP were reported to CRMD.

One was a second gravida who had medical abortion at one and a half months amenorrhoea after which she was not followed up. Came back after 3 months with 18 wks pregnancy. The treating doctor gave her mifepristone following which she did aspirotomy as the patient started bleeding profusely. During the procedure she perforated the uterus and brought down a loop of bowel. She was referred to a tertiary care facility. There a laparotomy, resection anastomosis of bowel and closure of wound were done. There was no mention about the status of uterus.

In this case suboptimal management is the cause of the bad outcome. The patient was not followed up. If the patient was profusely bleeding through a partially dilated cervix hysterotomy from a higher centre was the right decision. At the higher centre with laparotomy hysterectomy or opening rent and complete clearing of inside of uterus with higher antibiotics might have prevented septicemia. Ideally she should have been sent to a higher centre for second trimester termination.

Second was a third gravida with 36 wks size fibroid; evacuation was done for retained products. She was transferred to ward. Two hours later she collapsed and died. There was evidence of abdominal distension and intra peritoneal bleed.

The huge fibroid might have misled the obstetrician as to the extent of uterine cavity. She should have been managed in a tertiary care centre. Deaths due to MTP have reduced drastically thanks to medical methods. Medical abortion is a better option for terminating early pregnancies. A manual vacuum aspirator (MVA) is useful in early pregnancy terminations. An USG is mandatory to make sure that the uterus is empty in suspected cases.

Careful counseling is required to encourage for follow up and use of proper contraceptive methods.

Malignancies

Of the eight deaths due to malignancies only two were reported to CRMD. One was a breast carcinoma and the other a choriocarcinoma. [Editor's note :

two different cases are included under gastro intestinal causes]. Examination of breast should be routinely done as part of antenatal check up.

Other infrequent causes

One case of HIV died of sepsis. Screening for HIV has become universal. HIV positive women should get registered and receive antiretroviral therapy to prevent perinatal transmission as well as to reduce the viral load.

There were seven cases of surgical or gynecological pathology leading to death. One case of ovarian cyst rupture led to death. USG and timely management should have prevented such a calamity.

One was a case of sickle cell crisis leading to death. Early referral to a well equipped centre could have probably saved the patient.

A case of obstructed labour ended up in death. Unfortunately the details of the case are not available. Good antenatal and intrapartum care has drastically reduced this calamity nowadays.

Road traffic accidents are a matter of great concern with the rapidly increasing incidence of accidents. From the maternal death angle there were total of six, of which one came to CRMD, these can be considered coincidental. However pregnancy can influence the chance of getting the injuries as well as outcome. Wearing seat belt is not a prevalent practice in the population as a whole, leave alone pregnant women. With the incidence going up, we have to consider promoting separate types of seat belts which are suitable for pregnant women.

Chapter 19

Suggestions to improve antenatal care

Kunjamma.M.A; Vijayan.C.P

Antenatal care is one of the best examples of Preventive health care in modern medicine. It should start with premarital and preconception counselling. The first visit should be as soon as possible after the diagnosis of pregnancy. During that visit, outline of care for the whole pregnancy should be made. While normal young pregnant women may need only about five visits (see below), anybody with problem should have additional visits planned. What is given below is only list of some of the salient points and not by any means a complete description of antenatal care.

Premarital and preconception counselling where ever possible.

- Should be encouraged in situations like diabetes, cardiovascular disease and epilepsy for partners.
- When prospective parents are elders (above 35 years) or related.
- If there is a history of congenital anomalies and recurrent pregnancy loss.

Coverage – Aim is for 100% coverage

Strategy –Identifying and correcting risk factors. Risk factors should be highlighted in the case record and appropriate competent health care should be provided throughout pregnancy, labour and puerperium.

Benefits-Reduces maternal and perinatal mortality and morbidity

First trimester visit

This is very crucial in the antenatal care. Purposes are

Diagnosis of pregnancy

Dating of pregnancy- Do the dating to the possible accuracy and record it – Never alter it based on subsequent scans.

Predicting the prospects of pregnancy

Diagnosing other pelvic pathologies.

Minimum Number of visits:

One in first trimester, two in second trimester and two in third trimester.

Minimum Investigations

Hemoglobin.(to be repeated in each trimester)
Blood grouping and Rh typing
Blood sugar (Screening,Diagnostic or both.,-to be repeated)
HIV Screening, VDRL
HBsAg Screening
Urinalysis (to be repeated)
Other investigations as and when required

What to do in each visit

Search for symptoms
Height, weight, clinical assessment on growth, symphysio-fundal height and blood pressure are to be recorded.
Examine other systems and breasts
Ultrasonogram
Dating USG in first trimester if needed
Anomalies scan at 18-20 weeks, Further as and when required

Medications

Tetanus toxoid injections (two doses at 4 to 6 weeks apart) starting in first trimester. In second pregnancy (if within 1 year) one booster dose.
Folic acid, Iron, Calcium.
Avoid self medications by the woman. Whenever getting treatment for other illnesses in pregnancy have a record of them.

Antenatal records

Should be concise and better left with the woman. Advise to carry it and to be produced wherever getting admitted

Antenatal classes ,Following points to be addressed:

Diet, exercise, employment, travel, bathing, clothing,
Bowel habits, coitus, leucorrhoea, candidiasis, bacterial vaginosis, trichomoniasis, dentition, breast care, labour, puerperium and family planning.

Chapter 20

Intrapartum Care

S Ajithakumari

Labour room is a stressful place. Apprehension about the outcome of pregnancy, stories of unexpected complications in labour and the actual labour pains work together to add to the distress of the labouring woman. The compassionate approach and professional competence of the staff are the recipes to win the confidence of the parturient and her family. Welcoming the labouring woman with a warm smile is the first and best step to ease the situation.

- All women in active labour should receive close observation and companionship.
- All pregnant women require adequate surveillance through out labour and delivery.
- Prolonged labour is associated with high levels of morbidity and mortality.

Care of Normal Labour

- On admission to labour ward, a quick elicitation of relevant history and a rapid evaluation of the general condition of the woman including vital signs –pulse, BP, respiration and temperature should be done. Look for pallor or jaundice. Her prenatal records should be reviewed. Special points highlighted should be noted.
- Assess the fetal condition
- Intermittent auscultation of FH every 30 minutes for one minute in the first stage of labour and every 5 minutes for one minute in the early second stage of labour is adequate for low risk labours. In late second stage fetal heart should be checked between each contraction. EFM remains the main stay for intrapartum fetal monitoring in high risk labours. A CTG admission test picks up the subtle changes in fetal heart rate such as shallow decelerations and poor variability. In complicated cases (eg:IUGR) continuous electronic monitoring of fetal heart may be required.

- If the membranes have ruptured, note the colour of amniotic fluid. Presence of thick meconium indicates close monitoring to detect fetal distress. Absence of drainage of amniotic fluid after rupture of membranes is an indication for assessment of AFV and possible cord compression.
- Before vaginal examination, make it a point to listen to the FH and palpate the abdomen for fetal presentation and position and descent of head, so that the findings could be correlated with the vaginal examination findings.
- In most cases the partogram is capable of standing alone as a sufficient record and hence it is an essential tool which should be made obligatory in all the obstetric units. Partogram, the pictorial documentation of labour facilitates early recognition of unsatisfactory progress of labour, enabling timely intervention. A lag time of 4 hours between the slowing of labour and need for intervention is unlikely to compromise the fetus or the mother and avoids unnecessary intervention.
- Management of Latent phase of labour should usually be conservative. Oral intake of clear liquids like water, fruit juices without pulp, carbonated beverages, clear tea, black coffee and sports drinks may be allowed during labour.
- A poor rate of cervical dilatation and /or a poor rate of descent of presenting part may indicate a potentially problematic labour. The decision to augment labour should be governed primarily by the rate of cervical dilatation after exclusion of gross disproportion or malpresentation.
- ARM should be reserved for women with abnormal labour progress or in whom CTG abnormalities are present, to assess the liquor for meconium or for internal monitoring.
- Adequate hydration, appropriate analgesia and oxytocin infusion are the other aspects of labour augmentation.
- A time limit of 6-8 hrs to terminate labour after oxytocin augmentation is recommended.
- If membrane has ruptured for more than six hours and if delivery is not close, antibiotics must be given.
- It is not advisable to request a patient to bear down or to carry out a difficult instrumental delivery at an arbitrary 1 hour unless the presenting part is at the outlet.
- Prior to attempting an instrumental vaginal delivery, an abdominal palpation should be performed to confirm that not more than 1/5th of the head is palpable. Vaginally there should be no sign of excessive caput or moulding. The head should descent with uterine contractions and bearing down

effort, and fetal ear should be palpable. When a trial of forceps is undertaken, it should be done in the OT with full preparations for CS. There should be no hesitation in abandoning the procedure if difficulty is encountered with the application of the blades or locking of the handles.

With any forceps or vacuum delivery, if the presenting part shows no descent with bearing down efforts and traction over three contractions, the procedure should be abandoned and a cesarean should be done.

- If episiotomy is required make it mediolateral. Even after episiotomy, delivery of the head and shoulders should be in a controlled manner so that further extension does not occur. While suturing episiotomy, bring back proper anatomical alignment. Take care of apex and any active bleeders and use thick bites of the edges. Our aim should be to avoid complete perineal tears, but at the same time number of episiotomies also should come down. In the bargain, 1st and 2nd degree tears may increase and should be accepted.
- Active management of the 3rd stage of labour helps to prevent PPH. Oxytocin 5 units as bolus or 10 units IM is the first choice. Methergin 0.2 mg IM can also be given as prophylaxis against PPH except when it is contraindicated as in cases of hypertension and heart disease. This is given at the time of delivery of anterior shoulder. If methergin is given at the time of the delivery of the anterior shoulder, the trunk of the baby should be delivered slowly, so that the uterus contracts on to the baby's body preventing an hourglass spasm. Alternatively, after the delivery of the baby, oxytocin 10 units IM or oxytocin 5units + methergin0.2mgIM can be given. Oxytocin is preferred because it is effective in 2-3 mts after injection, has minimal side effects and can be used in all women. Prostodin 125 microgm IM, misopristol 600microgm rectally are the other oxytocic agents that can be used.
- Before attempting delivery of placenta, make sure that the uterus is contracted. While pulling on the cord, the left hand should hold the body of the uterus to prevent inversion.
- A careful inspection of the vagina and cervix to exclude tears should be done. The ideal time is in the interval between delivery of fetus and placenta. Since the placenta would not have separated, bleeding will be minimal and inspection of cervix and fornices may be easier. If there is an episiotomy check its extent and whether it has involved anal sphincter and rectal wall . It is a good habit to do a rectal examination at the end of suturing to check whether any suture has passed through the rectal wall in which case it is better to remove the stitches and redo the episiotomy.
- Forgetting to remove the tampon or pack in the upper vagina after suturing the episiotomy is an all too common mistake, especially when new batch of trainees join. Guard against this.

Chapter 21

Post natal care

Sangeetha Menon

Key summary points

Majority of deaths occur in the immediate post partum period, thus emphasizing the importance of vigilance at this time. There is often a tendency for caregivers to relax once delivery is over, this is to be guarded against.

Many of the deaths are usually the result of problems arising antenatally or intrapartum like pre eclampsia, liver disease, sepsis, injudicious use of uterotonics, intrapartum dehydration etc.

Infections - especially respiratory appear to be a major contributor to deaths whether antenatal or post natal. Strict guidelines must be in place, and commencing treatment early enough should be emphasized. In addition strict asepsis in the labour room and operation theatre is essential and should go hand in hand with the judicious use of antibiotics.

Established local practices and beliefs in the postpartum period like restriction of fluid intake and bed rest, only promote venous thrombosis and must be strongly discouraged. This form of education and persuasion must start right from the antenatal period in the form of ante natal classes.

Situations requiring post partum thromboprophylaxis must be recognized, individual risk assessment done and appropriate therapy offered.

Similarly life style modification and counseling especially in cases of PCOS, HIV, GDM etc. must not be neglected.

The importance of early and exclusive breast feeds in the interests of both the mother and the baby must be encouraged. Women must be gently coaxed to come for their 6 wk post natal review and accept an effective contraceptive as well.

The post partum period known for its emotional mood swings popularly known as post partum blues must not be neglected. Adequate emotional and medical

support must be provided. This will go a long way in averting suicides in such women.

Post natal care after a near miss event must be specially looked into, as many of these women would spend a large part of their immediate post partum period in ICUs/HDUs. Keeping a look out for infections, DVT, nutrition etc. must go hand in hand with addressing patient's worries, concerns and keeping up her morale.

Post natal care in special situations

Anaemia: Blood and products are not to be used flippantly. Once Hb is 6-7 gms%, further replenishment and build up of iron stores is best achieved with oral iron. It is recommended that in areas where the prevalence of anaemia exceeds 45%, oral iron and supplements be given for 4-6mths post nataly as a routine.

Hypertension: If BP continues to be high at the 12th post partum week, the woman is to be treated as a chronic hypertensive and appropriate anti hypertensive therapy instituted.

An increasing number of women are being seen where after discharge from the hospital, they get readmitted with seizures, sometimes 10-12 days after delivery. The entity of late post partum eclampsia vs intra cranial hemorrhage must be thought of and a multidisciplinary team must be on hand to tackle such cases. Most of them would need neuroimaging as well.

Gestational diabetes: Women must be encouraged to return in 6 wks with a 75gm WHO GTT, and overt diabetes must be excluded. Life style modifications, yearly diabetes screening must be insisted upon for the pure GDM patient.

Correctable heart disease: Cardiac surgery is advised for the correctable lesions especially before embarking on the next pregnancy.

Chapter 22

Cesarean section

P K Sekharan

Introduction

In 1985, the WHO study group reported “there is no justification for any region to have cesarean section rates higher than 10–15%”.⁽¹⁾ Over the last 25 years the practice of obstetrics and the attitude of the physicians and patients have changed a lot to *accept* a rate more than 15%. Rates of 32–35% are prevalent in developed and the developing world including India.^(2,3) Cesarean section is a life saving procedure, and with improvement and safety in anaesthesia and surgical techniques, cesarean section has become a safe procedure. Has this safety influenced the physicians and public to accept cesarean section as “*an easy mode of delivery*” compared to the long hours of waiting and struggle with the uncertainty of the outcome in vaginal delivery? The reality is that the “best” outcome for both the mother and the baby is after vaginal delivery and cesarean section is associated with a great deal of maternal morbidity and even mortality.

There is growing concern about this high cesarean rates among the obstetricians, and the media is making headlines over this issue in our state. There are no official data regarding the overall incidence of cesarean section in Kerala or for the whole country. In the WHO Global Survey on Maternal and Perinatal Health, which was conducted between 2004 and 2005 in 24 regions of eight countries in Latin America and which obtained data for all women admitted for delivery in 120 randomly selected institutions, the median rate of cesarean delivery was 33%; rates of up to 51% were noted in private hospitals⁽⁴⁾.

The survey conducted by the Federation of Obstetric and Gynaecological Societies of India (FOGSI) for the year 2007, 2008 and 2009 has shown a cesarean section rate of 29% in India (793482 cesarean sections in 2718088 deliveries). During the same period, the FOGSI survey had shown that Kerala was having a cesarean section rate of 32 % in a study of nearly 400,000 deliveries. (127895 cesarean sections in 399983 deliveries, unpublished data)⁽⁵⁾. A study by Kerala Federation of Obstetrics and Gynecology in 2008 had shown a rate of 35.9% in the state (Reference—Rajasekharan Nair. Deepthy & VPPaily). More recent data

from Kerala are particularly worrying. The cesarean rates have crossed 50% in four districts and for both private and government hospitals the rates are similar (Data kindly supplied by the directorate of health services).

Why an increase in Cesarean section rate?

- Women are having fewer children and the average age of primiparas is rising
- More primary caesarean will lead to more caesarean section rate, as it is – “once a cesarean, always a cesarean” phenomenon now a days.
- After the result of Hanna’s study, almost all breech presentations are delivered by CS.⁽⁶⁾
- Incidence of forceps and vacuum delivery is on the decline.
- Incidence of induction of labour is on the increase which increases the risk of cesarean section done for failed induction.
- The incidence of hypertensive disorders of pregnancy and the IUGR are on the increase with resultant increase in section rate.⁽⁷⁾
- More cesareans are done in primigravidas and with the decline in VBAC; the total section rate is going up.
- Patients conceived after ART procedures are almost always delivered by cesarean section.
- Whether malpractice litigations are influencing the decision?
- Is there any pressure to perform cesarean section on maternal request?

Strategies to reduce cesarean section rate:

- Try to achieve more of vaginal delivery in primigravidas as she will have an easier delivery next time in most instances.
- Counselling expectant mothers to allay anxiety and fear.
- Ensure adequate labour analgesia including epidural analgesia, especially for primigravidas in labour. Availability of such procedures should be known to the public.
- Allow labour room companion, relative or friend, especially for primigravidas.
- Use partogram for labour monitoring and practice active management of labour.
- Do cesarean sections only on medical indications.
- The NICE guidelines recommend that if a patient requests for cesarean section due to fear and anxiety, give her support and care and counsel her.

If in spite of proper counselling and offer of support including labour analgesia, she is afraid of vaginal delivery, offer a planned caesarean section⁽⁸⁾. (The guideline issued by Government of Kerala is not allowing such an option). Maternal request is not on its own an indication for caesarean section, and specific reasons for the request should be explored, discussed, and recorded. Keep the necessary documents when one has to take such a decision as patient is having her rights to choose the type of treatment-vaginal delivery or caesarean section-her informed choice. NICE guidelines recommend that if the physician is not in support of "patient request caesarean section", he should refer her to another physician who does caesarean section on maternal request⁽⁸⁾.

- Follow guide lines and protocols.
- Allow VBAC in selected cases with spontaneous onset of labour, engaged head with favourable cervix, and satisfactory progress and delivery in reasonable time frame of 6-8 hours. Practice VBAC only in institutions with facilities for immediate section if need arises.
- Consult another colleague before making a decision for caesarean section. A mandatory second opinion can achieve significant reduction in caesarean section rate.^(9,10) But in most of the small centres with only one obstetrician this may not be feasible.
- Ensure availability of anaesthesiologist round the clock or refer high risk cases early to higher centre.
- Uncomplicated breech near term should be offered external cephalic version.
- Practice assisted breech delivery in selected cases, as in multipara with history of normal delivery and average size fetus with satisfactory progress of labour and delivery in 6-8 hours.
- Failed induction being an important indication for caesarean section, practice ripening of cervix including mechanical methods prior to induction to achieve more success
- HIV patients on antiretroviral therapy and whose viral load is less than 400 copies are not to be offered caesarean section for prevention of mother to child transmission⁽⁸⁾.
- Practice and propagate the safe use of forceps and vacuum for assisted vaginal delivery which will help to reduce caesarean section.
- Presence of senior consultant in the labour ward will help reassure mothers and trainees that appropriate care is given at the right time and will help to reduce section rate.

- Auditing of caesarean sections done in every institution with a view to avoid unnecessary caesarean sections is mandatory. While auditing, categorise the cesareans under Robsons criteria so that inter institutional comparisons become more meaningful.

Caesarean Section-Technique:

At least one out of three pregnant women, if not more, will be delivered by caesarean section now a days⁽⁹⁾. Since the operation is done so frequently, it is important to follow the evidence based safe procedure which will result in better outcome for both the mother and baby.

Pre-Incision Antibiotic Prophylaxis

Routine use of prophylactic antibiotics reduces the risk of post-caesarean fever and infections by over 50%.⁽¹¹⁾ Consequently, antibiotic prophylaxis is recommended for all women undergoing caesarean section.⁽¹²⁾ To prevent fetal exposure, to the antibiotics the traditional practice has been to administer antibiotics only after delivery of the infant and clamping of the umbilical cord. Recent studies of antibiotic prophylaxis for caesarean delivery suggest that prophylactic antibiotics administered prior to incision is more effective in preventing post-caesarean infection than administration after umbilical cord clamping.^(13,14) The new ACOG recommendation for antibiotic prophylaxis during cesarean delivery is to give antibiotics within one hour before the start of surgery. In the case of an emergency cesarean section, prophylaxis should be started as soon as possible⁽¹⁵⁾. The recent NICE guideline recommends prophylactic antibiotics before skin incision and states that no adverse effect on baby has been demonstrated.⁽⁸⁾ A recent joint publication by ACOG and the American Academy of Pediatrics stated that "... an antibiotic before the procedure (cesarean) has been demonstrated to be more effective than administration immediately after umbilical cord clamping."⁽¹⁶⁾ The preferred prophylactic antibiotic for cesarean section is cefazolin 1-2 g IV.

Anaesthesia

Regional anaesthesia is safer and results in less maternal and neonatal morbidity than general anaesthesia. Volume pre-loading with crystalloid or colloid will reduce the risk of hypotension occurring during cesarean section. Catheterise the bladder before giving regional anaesthesia. Give a lateral tilt by keeping a wedge under the right buttock of the patient to avoid supine hypotension(A 15° tilt of the table).

Abdominal incision

A lower transverse incision is preferred and vertical incision is used only in

special situations. There is not much difference in outcome between Pfannenstiel, "Joel-Cohen", "Misgav-Ladach" and "modified Misgav-Ladach" methods. The Joel-Cohen incision showed less postoperative febrile morbidity and analgesic requirement than the Pfannenstiel incision. Maylard incision is used only in selected cases to have more exposure. Better to use scalpel for sharp dissection, rather than an electrocautery⁽⁸⁾. The size of the incision is about 15 cm for the easy delivery of the baby. The subcutaneous tissue is opened by blunt dissection with fingers. The rectus muscle is separated bluntly, avoiding transection. The peritoneum is opened as high as possible in obstructed labour and in severe adhesions.

Dense intraperitoneal adhesions

If there is dense adhesions between the anterior abdominal wall and the anterior surface of the uterus, enter the peritoneum bluntly and as close as possible to the upper abdomen to avoid areas of dense scar tissue in the lower abdomen. Sharp dissection may also be required, and should be done cautiously under direct vision. If adhesions require extensive dissection with risk of injury to the bowel, bladder, or major blood vessels in order to expose the lower uterine segment, avoid or minimize extensive dissection, and incise the uterus at the most appropriate accessible location, including the upper segment, especially if the patient is desirous of sterilisation. Get the help of an experienced surgeon in such situations.

Uterine incision

A bladder flap may be made by opening the uterovesical fold of peritoneum and bladder is pushed down 2-3 cm to expose the lower uterine segment. In cases of obstructed and prolonged labour, the incision on the uterus should be made just at the reflection of the UV fold of peritoneum to avoid entry to the vagina. A sharp incision of two cms is made in the lower uterine segment which is then enlarged with blunt dissection with fingers or by sharp dissection with scissors. Care must be taken not to injure the baby if possible by keeping the membrane intact while incising the uterus.

Delivery of the baby

Outlet forceps or vacuum should be available in the theatre to deliver the floating head of the baby. In prolonged labour the head may be jammed in the pelvis which could be dislodged by the assistant pushing from below. Alternatively the surgeon grasps the fetal legs in the upper segment and delivers the baby as breech- "reverse breech". Delayed, rather than immediate, cord clamp-

ing results in greater neonatal haemoglobin levels and appears to be beneficial for both preterm and term infants.

After the baby is delivered, 10 units of oxytocin is added to 500 ml normal saline and infused at 10 ml/min for a few minutes to get a sustained contraction of the uterus. In addition give 5 units I V bolus or 10 units IM. Placental separation is aided by oxytocin induced uterine contraction and delivered by cord traction from a contracted uterus as manual removal may lead to excess bleeding. The oxytocin is continued as intravenous infusion of 10 units in 500 ml of normal saline 125ml/hr for 8 to 12 hours.

Uterine closure

It is not necessary to exteriorize the uterus routinely for closure, but could be of advantage if there is extension of the wound or if the uterus is flabby to massage the uterus effectively. The disadvantage of exteriorisation is the discomfort and vomiting in cases where the surgery is done under regional anaesthesia.

Given the available data, either a one layer or two layer closure techniques is within acceptable standards of medical care. Uterine rupture has been reported following vaginal delivery both in single layer and double layer closure. Suture material could be absorbable or delayed absorbable number 0 or 1. At Parkland hospital, they prefer one layer uterine closure. Placing separate suture beyond the angle on either end of the uterine incision will prevent bleeding from the angles. After placing the initial suture beyond the angle of uterine incision, a running-lock suture, which gives a more haemostatic closure, is performed with each suture penetrating the full thickness of the myometrium. The running-lock suture is continued beyond the opposite angle of uterine incision. (A non locking running stitch also may be used, provided additional stitches are taken to take care of any bleeders from the edges. Editors) Additional figure-of-eight or mattress sutures may be required for complete haemostasis. Locking suture in cases of very thin lower uterine flap may cause tear and may be avoided.

[Editor's note: To reduce the risk of bleeding from the angles, we recommend an additional safe guard. The anchoring stitch is taken lateral to the angle and a mattress stitch is then taken to include about 1cm of the edges and tied. This is done first on the right angle. The suturing then starts on the other side and is continued medially after the anchoring and mattress stitches]

It is essential to inspect the adnexae for presence of ovarian mass before closing the abdomen as it can be removed at the same time. Most of the fibroids can also be safely removed during caesarean section. Make sure the paracolic gutters and pouch of Douglas are free of any clots, meconium or amniotic fluid. Mop and instrument counts should be checked before closure of abdomen.

Closure of the abdomen

There are arguments for and against closure of the parietal peritoneum during cesarean section. Nonclosure might allow the enlarged uterus to adhere to the anterior abdominal wall or impede spontaneous closure of the peritoneum, while closure might cause a foreign body reaction to sutures and tissue damage. A systematic review of peritoneal nonclosure and adhesion formation after cesarean delivery found some evidence that nonclosure was associated with greater adhesion formation than closure of the layer or both visceral and parietal layers⁽¹⁸⁾. Another systematic review also found that nonclosure resulted in greater adhesion formation⁽¹⁹⁾. (Readers are requested to follow further evidence emerging in this regard)

The rectus muscle is not sutured unless there is severe diastasis. The rectus sheath is closed by simple running technique using #1 or #2 delayed absorbable monofilament suture (e.g., polydioxanone-PDS) taking wide tissue bites of 1cm with short stitch intervals of 1cm with non-strangulating tension on the suture. Close the subcutaneous adipose layer with interrupted delayed-absorbable sutures if the layer is 2 cm. Skin is approximated by subcuticular suture.

Classical Cesarean section

The emerging indication to do a classical caesarean section now a days is the anterior placenta praevia increta or percreta, where placenta has developed over the previous scar area and has gone through the myometrium. Attempts to remove such a placenta will lead to catastrophic haemorrhage and death of the mother. The incidence of placenta accreta is on the increase with increasing incidence of caesarean section and with increasing number of sections in the given patient. Localization of the placenta should be done by 32 -34 weeks in all previous caesarean cases. If found to have anterior placenta praevia, offer colour Doppler evaluation to diagnose morbidly adherent placenta which can be confirmed by MRI.

Placenta accreta in previous cesarean scar.

Patients with confirmed placenta accreta with evidence of penetration into and beyond myometrium should be managed with proper surgical planning to reduce the risk of massive haemorrhage leading to morbidity and mortality.

- The patient and relatives should be informed of the suspected diagnosis with risk of bleeding, need for blood transfusion and necessity for caesarean hysterectomy as a life saving procedure.(Informed consent)
- Management by multidisciplinary team with expert obstetric surgeons,

urologists, anaesthesiologists, neonatologists in a tertiary care facility with blood and component transfusion facilities.

- Antenatal corticosteroids for lung maturity.
- With history of bleeding, termination at 34 weeks after steroids.
- Asymptomatic patients, termination at 37 weeks.
- Elective surgery by the expert team.
- Adequate blood and blood products should be made available.
- In cases of placenta accreta with evidence of penetration into the myometrium and beyond, the best outcome is by elective cesarean (Classical) hysterectomy without attempt at removal of placenta. ⁽²⁰⁾
- Abdomen is opened by vertical incision.
- A classical incision is made on the upper segment of the uterus above the placenta to avoid disruption of it. Baby is delivered as breech
- We recommend hysterectomy with placenta left in situ undisturbed when there is evidence of placenta increta or percreta to reduce the risk of torrential bleeding.
- Internal iliac artery ligation is time consuming and may not be effective in all cases.
- Carefully dissect the bladder down to reach the cervix and do a near total hysterectomy. (Reaching below the level of placental extension)

Conservative management when the patient is having severe bleeding is waste of time and will lead to increased morbidity and mortality.

[Note: Management of placenta previa accreta is discussed in the chapter on obstetric hemorrhage where a new strategy is described- Editors]

Maternal mortality and cesarean section.

Confidential Review of Maternal Deaths (CRMD) for the state of Kerala is being conducted regularly by the Kerala Federation of Obstetrics and Gynaecology (KFOG) since 2004. There were 676 maternal deaths in Kerala during the period 2006-2009. Details were available in 331 maternal deaths for analysis. Of these 331 maternal deaths, in 145 cases there was an association of caesarean section (44%). Though cause of death is not directly due to caesarean section, the association of the procedure in maternal deaths is very significant. Of the 145 deaths among caesarean cases, 23 cases were following elective caesarean section and 122 cases were following emergency section. Maternal mortality is more in emergency cases compared to elective cases. In the present study there were 23 deaths in elective cases! In both elective and emergency caesarean sections, there were deaths

due to PPH, (31cases-21% of the maternal deaths among the caesarean group was due to PPH!).

Examples of suboptimal care

Example 1

Cesarean section was done for placenta accreta in a patient who had two previous sections. Patient developed severe PPH and relaparotomy and hysterectomy was done. In spite of blood transfusion and hysterectomy, patient expired in the higher centre.

Learning points

Placental localization and evidence of placenta accreta was not done in this patient who had two previous cesarean sections. Without much planning and anticipation for the bleeding, the cesarean was done at a peripheral centre and attempt at removal of adherent placenta led to severe postpartum haemorrhage. Even a hysterectomy afterwards could not control the bleeding and patient died at the higher centre.

Placental localisation and evidence of placenta accreta should be confirmed in all cases of previous cesarean section with placenta praevia. Placenta accreta with invasion to the myometrium should be managed in tertiary care centre by expert team. In cases of placenta accreta with evidence of penetration in to the myometrium, a planned classical cesarean hysterectomy without attempt to removal of the placenta will be the optimum care. Follow the principles given above for the classical cesarean section for placenta accreta. [See the strategy for managing placenta previa accreta in the chapter on PPH – Editors]

Example -2

Cesarean section was done at a peripheral centre for CPD. Patient developed hypotension postoperatively and in spite of 4 units of blood transfusion, the condition of the patient did not improve after 8 hours, was referred to higher centre. There was evidence of intra-peritoneal bleed and an emergency laparotomy showed 2 litres of blood in the peritoneal cavity and haematoma on to the left broad ligament. A subtotal hysterectomy was performed, but patient expired due to cardiac arrest. From the operation notes, it is seen that there was bleeding from the left angle of the caesarean wound which required additional suturing.

Learning points

In this case, there was failure in controlling the bleeding from the angle of the cesarean wound. Care must be taken to secure the angles of the wound and separate stitches going beyond the angles on either side are taken first and then only start the closure of the uterine wound. In case there is extension of the incision, exteriorise the uterus and then close properly. It may be prudent to do the ligation of the uterine artery or internal iliac artery in this case. There was also failure to recognize internal bleeding in this case. In spite of 4 units of blood transfusion patient was remaining in hypotension and the signs of internal bleeding were missed. There was also delay in referring the patient to the higher centre.

Example 3

Cesarean section was done in a case of previous cesarean section at 9 pm. The uterus was adherent to the anterior abdominal wall. There was difficulty in separating the adhesions. Patient had severe vaginal bleeding and had to undergo relaparotomy and hysterectomy. Patient developed intraperitoneal bleeding and bleeding from the abdominal wall. Referred to higher centre, developed irreversible shock and expired.

Learning points

A previous cesarean case should be taken up for surgery in the convenient hours other than in cases where cesarean is done to terminate the trial of vaginal delivery. When there is adhesion between the uterus and abdominal wall, get the help of experienced surgeon, use careful blunt and sharp dissection to expose any part of the uterus, incise and deliver the baby and do a sterilisation. As more and more cesareans are going to be done for previous cesareans, such cases are bound to come and one should be careful to use the correct technique.

Conclusions

Cesarean section is a life saving procedure but still we are having maternal mortality among caesarean cases. It may be the indication for which the section is done that contributes to the mortality, but role of the surgical procedure itself leading to mortality cannot be ruled out. Emergency cesarean done by junior staff at less optimal conditions may increase the mortality. In spite of hysterectomy for PPH during cesarean section, 21% of such patients were lost. Since 45% of the cases of maternal deaths analysed in CRMD are associated with cesarean section, care and proper technique should be followed in performing cesarean section.

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Chapter 23

Instrumental Vaginal Delivery

Betsy Thomas

Instrument assisted vaginal delivery is an essential component of obstetric management. Forceps and ventouse (vacuum) deliveries come under this category. Unfortunately, use of both of these instruments is on the decline. The obstetric forceps is almost abandoned.

Ideally obstetrician should be confident and competent in the use of both forceps and vacuum extractor as they definitely help to reduce cesarean delivery rates. Assisted vaginal delivery with ventouse and forceps is less morbid than cesarean delivery especially a second stage cesarean delivery. However, improper use of these instruments can lead to maternal and neonatal mortality and morbidity; hence, the importance of proper training in the use of these instruments.

Example

This 27 year old G₂P₁L₁, previous normal delivery was admitted at 40 weeks for safe confinement. She was induced with 25µg vaginal misoprost at 10 pm. At 6:30pm, the next day, cervix fully effaced and 2 cm dilated. ARM showed clear liquor. Oxytocin started at 7:30 pm and she delivered with vacuum at 8:02pm with face to pubis a 3.4 kg baby, placenta complete. 10 units oxytocin infusion started and 400µg rectal misoprost given. Vaginal pack applied as routine procedure. Pack removed at 9:30pm to shift the patient to the ward. Increased bleeding was noticed. Inj.prostodin IM given. Patient went into shock at 10pm. Severe bleeding PV present. Referred to higher centre with I V fluids and Foleys catheter (no urine). She reached the higher centre in irreversible shock. Vaginal examination revealed vaginal tear and cervical laceration extending into the lower segment. Two units of blood and six FFP given. She was declared dead after 4 hours.

Learning points

1. Pertaining to the instrumental delivery the indication for vacuum was not mentioned. Was it a hasty application as the first and second stages of labour together was only about 90 minutes. Routine packing of the vagina is not

recommended after any delivery. It can conceal the ongoing bleeding.

2. It is advisable to use a cervical inspection set to inspect cervix and vagina after any instrumental delivery by the person applying the instrument himself or herself.
3. Even after noticing PPH, cervical inspection was not done. By the time it was done in the higher centre, she was moribund.

What are the essential conditions for safe operative vaginal delivery ?

Safe operative vaginal delivery requires a careful assessment of the clinical situation, clear communication with the mother and the relatives and expertise in the chosen procedure.

What are the pre requisites for operative vaginal delivery ?

- There should be a definite indication
- Informed (preferably written) consent
- Head is < 1/5th palpable per abdomen
- Vertex presentation (also mento anterior and after coming head of breech for forceps) at spines or below
- Cervix is fully dilated and membrane ruptured
- Exact position of the head should be known
- Bladder should be empty
- Back up plan in place in case of failure to deliver
- Presence of senior obstetrician, if a junior trainee is performing delivery
- Anticipation of complication that may arise (eg: shoulder dystocia, PPH)
- Presence of personnel trained in neonatal resuscitation

Which instrument to apply ?

The operator should choose the instrument most suitable to the clinical circumstance and the level of skill. Forceps and vacuum are associated with different benefits and risks. Failed delivery is more common with vacuum. The vacuum cup should be over the flexion point (3cm in front of the posterior fontanelle). There is insufficient evidence to favour either a rapid (over 2 minutes) or a step wise increment in negative pressure with vacuum extraction. Always a cephalic application of forceps is preferred.

Generally speaking it can be stated that a forceps is to be preferred in cases with complete rotation of head, low stations and when the fetus is preterm. A vacuum extraction is to be preferred if it is a full term, vertex presentation with incomplete rotation.

What about preterm babies ?

Forceps (small sized forceps available) is safe for preterm babies. A vacuum extractor should not be used at gestation less than 34 weeks. The safety of vacuum extractor at 34-36 weeks is uncertain and should therefore be used with caution. Vacuum is safe for 36 weeks and beyond. Of the different types of cups, a rigid cup like Malmstroms is to be chosen when strong traction is expected as in occipitoposterior positions. A soft cup like the silastic will leave less mark on fetal scalp but the traction force that can be exerted is less. Hence, the chance of slippage (pop off) is more with silastic cup.

When is operative vaginal delivery likely to fail ?

- BMI > 30
- Estimated birth weight > 4kg
- Occipito posterior position
- When 1/5th or more of the head palpable per abdomen

When should operative vaginal delivery be abandoned ?

Operative vaginal delivery should be abandoned where there is no evidence of progressive descent with moderate traction during each contraction or where delivery is not imminent following 3 pulls of a correctly applied instrument by an experienced operator. The use of sequential instruments is associated with an increased risk of trauma to the baby; but may be judiciously used when indicated.

All operative vaginal delivery patients should undergo a cervical and vaginal inspection soon after delivery and should be closely monitored in postpartum period.

Analyzing the data from CRMD, it can be seen that all the seven maternal deaths have happened after vacuum and not a single death is reported after forceps. This is definitely because of the very few numbers of forceps being used by the obstetricians in our state.

Chapter 24

Obstetric care in the periphery: Role of Health workers in periphery in reducing maternal death

Dr.Lalithambica K

Many maternal deaths occur in tertiary care centres. But, going into the details of the cases, it is realized that the initiating event occurred in the periphery either in the form of commission or omission. So one of the important steps to reduce maternal mortality will be to augment the capacity of medical officers and other health care providers in the periphery, in early diagnosis and appropriate management of complications related to pregnancy and delivery and timely referral of cases.

Following are a few real situations.

Example 1.

26 year old primi brought with history of cesarean section followed by PPH, which was controlled with usual measures . But she developed status epilepticus, In spite of ventilatory support and all other measures, she died.

On detailed history it was revealed that she was a known epileptic who stopped anti epileptic drugs (AED) during pregnancy against medical advice. This lady had regular antenatal care, but this history was never known to the obstetrician.

A proper history in the antenatal period and continuation of AED could have saved that patient.

Example 2.

Primi, admitted for safe confinement. Low risk mother, 39 weeks 5 days . BP-130/90 mm Hg. At 6 am p/v Cx 75% effaced, 2 fingers. PGE2 was instilled, **Pitocin started simultaneously**. At 7 am ARM was done and pitocin continued. Every hour vaginal examination was repeated. Buscopan suppository was given (2 doses 2 hourly). She delivered at 11 am, developed severe PPH and coagulation failure from which she could not be saved.

Method of induction should be chosen carefully. It is very important that prostaglandins should not be used on a "favourable cervix". Other drugs like drotaverin and buscopan are associated with cervical lacerations and PPH.

Example 3.

Mrs X para-2, underwent LSCS at a peripheral hospital. After 8 hours she was brought in shock and DIC to a tertiary centre. She died in less than one hour.

Here LSCS was done for a low risk previous CS. Intraperitoneal bleeding was diagnosed two hours later. At that time patient was hemodynamically stable. As blood was not available, waited for 2 hours. Meanwhile intraperitoneal bleeding continued. Without adequate support or resuscitation, relaparotomy was done in the peripheral hospital itself leading to the catastrophe. *Immediate referral could have saved the patient.*

In all the above situations, there is a definite preventable element.

Antenatal Care at periphery

Educate prospective mother and their relatives

Health care providers should try to identify risk factors by a proper history, examination and appropriate investigations in early pregnancy itself. Remember, any pregnant woman can have complications which can lead to maternal morbidity and mortality.

So any centre catering to pregnant woman should have facilities for easy transport and access to a referral centre.

Educate the patient and relatives regarding warning signs: fever, headache , blurring of vision, epigastric pain, vomiting, generalized swelling of body and puffiness of face, palpitations, fatigability progressively increasing breathlessness on exertion and breathlessness even on rest, severe pain abdomen, bleeding per vagina, watery discharge per vagina, decreased urine output, decreased fetal movements.

Tubal ectopic pregnancy and scar ectopic pregnancy are two important life threatening complications in early pregnancy. The first two conditions are quite asymptomatic till they rupture and produce massive intraperitoneal bleeding. Massive hemorrhage can occur even with an abortion. As far as Kerala is concerned ultrasonogram is available even in villages, so it is ideal to have a scan in early pregnancy to rule out scar ectopic and tubal ectopic pregnancy.

Inevitable /Incomplete abortion with profuse hemorrhage should be managed in the periphery rather than referring to a higher centre because once products

are removed bleeding will stop. *So even PHCs should be provided with facility for abortion evacuation in terms of manpower and equipments.*

In the first visit itself Blood Pressure(BP) should be checked to rule out chronic hypertension and chest should be carefully auscultated to detect valvular disease like mitral stenosis. In second trimester physiological fall in BP can mask chronic hypertension.

Gestational Hypertension(GHT)

Careful BP check by a reliable person is a must. If an increase in BP is observed , for example from 110/70 to 130/85 , **though it is below the cut off for diagnosis of GHT ,it should be given importance.** When there is such an observation, the woman should be asked to come for BP check after two or three days. Do not wait for another two to four weeks. Meanwhile some of them can go for complications. Also sudden weight gains, massive pedal edema, facial puffiness, etc. should be carefully observed and evaluated for early detection of gestational hypertension and its complications. Facility for checking urine albumin, platelet count, liver enzymes etc should be available even in small centres.

One important cause of mortality associated with gestational hypertension is cerebral hemorrhage. Failure to achieve control of BP below 160 /110 is associated with this complication. So all obstetricians in periphery should be aware of timely management of hypertension. Even if patient is referred antihypertensive of appropriate dose should be given , if referral centre is far away. During transport also BP should be monitored and controlled. If BP remains around 160/120 cerebral autoregulation will be lost and cerebral hemorrhage can occur during transport.

Postpartum hypertension is a dangerous complication. It can be the manifestation of various life threatening complications. TTP/HUS and, Cortical venous sinus thrombosis are two important complications that can present with hypertension in puerperium.

Eclampsia

Give loading dose of MgSo₄ irrespective of urine output. Record the dose and time of administration and inform the higher centre by phone as well as referral letter. If convulsion occurs for the first time in puerperium, always think of other possibilities and consider the need for imaging brain and investigating for other causes, rather than blindly treating as postpartum eclampsia, though one is justified in giving magsulf.

Diabetes mellitus : All pregnant women should have a blood sugar check in early pregnancy to pick up cases of pregestational diabetes mellitus (PGDM). PGDM carries high risk of diabetic ketoacidosis which is a life threatening complication. All cases of PGDM should be referred to higher centre early.

Placenta previa in a case of previous caesarean should be considered a serious problem and all such patients should be referred sufficiently early, as they are at high risk of placenta accreta/percreta. All cases of major degree placenta previa are also better referred to higher centers.

Jaundice: viral hepatitis is common in our state. Loss of appetite, nausea and vomiting are some of the earliest symptoms. Many a times these early symptoms are ignored by the patient and/ or obstetrician and vomiting is treated symptomatically and viral hepatitis is diagnosed late with serious sequelae .

Fever: Though most cases of fever are self limiting and resolve with supportive measures, Weill's disease and H1N1 influenza are two conditions that are associated with mortality. *Most important measure to reduce mortality is early diagnosis.* Awareness among patients as well as doctors is very important for early diagnosis. Look for early symptoms and signs like sore throat ,myalgia, conjunctival congestion etc. Serology will be positive only after a few days.

Intrapartum Care

- **Avoid unnecessary interventions.** Induction of labor should be on definite indications. Use prostaglandin only in appropriate patients. It is very important that prostaglandins should not be used on a "favourable cervix". Other drugs like drotaverin and buscopan are associated with cervical lacerations and PPH.
- **Use of partogram** is a must. Proper knowledge and experience in interpretation of partogram is essential. Whenever labour progress is not satisfactory, always seek help. Either get second opinion or refer.
- **Always avoid dehydration and electrolyte imbalance** which predisposes to PPH. A dehydrated mother develops shock even with smaller blood loss.
- **Intrapartum fetal monitoring:** intermittent auscultation is enough for low risk patients. If Electronic fetal monitoring is used, sufficient knowledge and experience in interpretation is essential. Otherwise unnecessary interventions will increase cesarean section rate and thereby increase complications.

- **Instrumental delivery**, if mandatory, should be done with caution. Outlet forceps of appropriate size should be available. Active management of Third stage of labour (AMTSL) should be strictly adhered to. Make it a rule to visualize cervix and vagina after every instrumental delivery for early diagnosis of traumatic PPH. Also look at paraurethral region to rule out tear. Massive bleeding can occur unnoticed from paraurethral tear.
- So called “4th stage of labour” should be under close supervision. Patient should be closely monitored for any excess bleeding. During this period in addition to monitoring pulse and BP, the uterine fundus should be palpated to ensure that it is contracted and gently proceed to express the any clots. It will be a good practice to make sure that she passes urine before being shifted to the ward. This will help to rule out PPH as well as perineal wound hematoma.
- **Sepsis** is an important contributor to serious maternal morbidity and mortality. Any centre intended to conduct delivery should have a labour room where aseptic precautions are maintained like in an operation theatre.

The staff working in periphery should have proper training and periodic evaluation in the following aspects.

- a. aseptic precautions.
- b. early detection of PPH and assessment of degree of shock.
- c. identification of types of PPH.
- d. skills for immediate management of PPH and referral.
- e. active management of third stage of labour for prevention of PPH.
- f. treatment of shock after quick assessment of mother’s general condition.
- g. appropriate and timely referral of cases, referral only after starting IV infusion (Normal Saline) and adding 20 units of Oxytocin to it, making sure that the drip runs during transportation of the patient, applying condom tamponade to minimize bleeding during transport.
- h. referral to be made only to the hospitals having facilities for blood transfusion.
- i. Inform the medical officer on duty at the referral centre over telephone about patient’s condition and also the blood group if known.
- j. most facilities need replacement of blood, prepare the family for blood donation and the donors to accompany the patient.

Anaesthesiologist working in the periphery has an important role in reducing maternal mortality. They also should be actively involved in various teaching and training programs aimed at reducing maternal mortality.

The services expected of the peripheral health workers can be viewed in terms of the three delay model.

Delay 1: Delay in recognizing the problem by the mother or her family. (Lack of awareness of danger signs) and deciding to seek care(due to inaccessible health facility, lack of resources to pay for services/supplies and medicines).

Delay 2: Delay in reaching the health facility(due to unavailability of transport, lack of awareness of appropriate referral facility).

Delay 3: Delay in receiving treatment once a woman has arrived at the health facility (due to inadequately equipped health facility , lack of trained personnel , emergency medicines, blood etc.).

Peripheral health worker has a key role to play in reducing the delay 1&2. Timely information passed on to the receiving centre will help in reducing the third delay as well.

In Summary

Sensitizing the community and family for right decision at right time and timely referral through pre-identified transport can address the first two delays and would help women access the services available as and when required. Simultaneously, the health workers need to be technically competent and facility adequately equipped to provide services/care to the woman reaching the health facilities in the periphery. This would help in ensuring the provision of skilled attendance to all women during pregnancy and childbirth and enable us to achieve the goal .

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Chapter 25

Common pitfalls in Obstetric Practice

Nirmala.C

Introduction

Child bearing is a major life passage for approximately 5.4 lakhs mothers in Kerala every year. Maternal mortality and Near-miss mortality has been used as a measure of the status of women, their access to health care and the capacity of the health care system to respond to their needs¹. A situational analysis of trends in obstetric care in Kerala has raised various concerns, the major ones being increasingly avoidable errors and pitfalls leading to mortality and immediate and long term morbidity for mothers and their newborns. Most of these pitfalls result from omissions or incorrect treatment or overuse or underuse of interventions. Many of these pitfalls are preventable if proper guidelines and checklists are adhered to. This article reviews some of the pitfalls encountered in the major areas of Obstetric care that contributes to maternal mortality and morbidity which are modifiable and preventable.

Major opportunity for Kerala in improving maternal health is that 99.4% of deliveries are institutional, attended by obstetricians and medical officers². Hence achievable and desirable changes are possible as most of the pitfalls leading to maternal mortality and near -miss situations are related to management during antepartum, intrapartum and post partum periods.

Pitfalls in effective management of labor

1. Elective induction of labor - a growing concern

Although induction of labour has been efficacious in the management of post term pregnancy and in expediting delivery when the condition of the mother or infant makes continuation of pregnancy hazardous, the recent rapid increase in inductions particularly for debatable indications is a matter of concern. Rate of elective labor induction (Induction of labor without an accepted medical or obstetrical indication *before* the spontaneous onset of labor or rupture of membranes) has dramatically increased in Kerala and this has contributed to adverse maternal and fetal outcomes. When comparing spontaneous labor, elective

inductions result in more cesarean deliveries and longer hospital stay. Elective induction doubles the cesarean delivery rate, particularly among primigravidas with unfavorable cervix. In addition, labour progression differs significantly for women with an elective induction of labour compared with women who have spontaneous onset of labour. One of the pitfalls related to induction is incorrect estimation of gestational age.

a) Estimating gestational age: Prenatal methods of estimating gestational age have a margin of ± 2 weeks. So, elective induction of labor will lead to delivery of an earlier gestational age than intended. Hence, assessment of gestational age and consideration of any potential risks to the mother or fetus are of paramount importance. Definitions of “full-term” and weeks of gestation that define safe birth are commonly misunderstood by many. No induction should be done prior to 40 weeks of gestation in the absence of medical indications. *The general consensus of 38 weeks of gestation as term pregnancy is proved to be wrong.* The delivery of infants who are born between 37 weeks to 39 weeks gestation is called early term deliveries. Induction of labor during this period denies the critical fetal development that occurs during the last weeks of pregnancy. Newborns born at 37 weeks gestation have a 7.5 fold greater rate of developing RDS versus those born at 39 to 41 weeks gestation (Tita, et al., 2009)³. Early-term newborns born at 37-38 weeks gestation also are at higher risk for transient tachypnea of the newborn, pulmonary hypertension, hospital stays greater than 5 days as well as diagnoses associated with severe morbidities or death versus newborns delivered at 39 weeks gestation⁴(Engle & Kominiarek, 2008).

Maternal problems include increased rates of cesarean, chorioamnionitis, endometritis, intrapartum sepsis and postpartum anaemia. One of the major causes of maternal mortality in Kerala is Sepsis. Induction in the setting of an unfavourable cervix especially in the nulliparous woman can result in prolonged labour, failed induction and an increased cesarean delivery rate and increased rate of sepsis.

b) Diagnosing failed induction: Diagnosing failed induction is a matter of concern. The decision to diagnose failed induction is mostly subjective and is based on individual obstetrician’s discretion. No clear-cut objective criteria are being followed. Most of the failed inductions are diagnosed in latent phase of labor. Allowing at least 12–18 hours of latent labor before diagnosing a failed induction may reduce the risk of cesarean delivery

c) Monitoring labor –use of partogram: A partogram is a graphical representation of a woman’s progress in labour, plotting the duration of labour in hours against cervical dilatation in centimetres. Use of partogram facilitates earlier recognition of dystocia, thereby optimizing the timing of appropriate intervention, such as amniotomy, oxytocin augmentation, or most importantly

cesarean section (CS). In spite of clear advantage, partogram is not used for decisions regarding various interventions. Situational analysis shows that intrapartum indications for cesareans like dystocia, failure to progress are not supported by partograms.

d) Intrapartum monitoring – interpreting CTG: Intrapartum fetal distress is often quoted as indication for caesarean section in Kerala. This can be attributed to popularity of electronic fetal monitoring. But assessment of the fetal distress during labor is a challenging task. Various indicators of interpreting CTG include baseline rate, variability, accelerations and decelerations. Interpreting and arriving at decisions as to normal CTG, suspicious CTG and pathological CTG, requires proper skills and knowledge. Poor skills in interpreting CTG leads to increased cesarean for fetal distress. Incorrect interpretation of CTG and improper training are pitfalls associated with increased rates of cesarean delivery for abnormal fetal heart rate.

[We feel that the adverse outcomes following induction mentioned above can be overcome to a large extent if a few precautions are taken.

1. Use mechanical methods of ripening of cervix prior to use of oxytocic agents.
2. Do not take up for elective induction earlier than seven days prior to EDD, that is 273 days.
3. Never commit to patient and relatives a definite time limit for delivery after elective induction.]Editors

Increasing cesarean section rates

The steep rise in cesarean delivery rates in Kerala is a matter of serious concern. Very little documented health benefit for newborns is associated with the near-doubling of cesarean deliveries over the years. Delivery by cesarean is beneficial and life saving if used for selected indications. Absolute indication for cesarean is applicable only for selected proportion of deliveries. The perceived safety of cesareans is the major pitfall contributing to the rising rate of cesarean deliveries in recent years in Kerala. Although cesarean deliveries have become dramatically safer over the past century, they are not without significant risks both to the mother and her newborn. Patients and providers need to understand these risks before embarking on this most common of surgical procedures.

a) Elective Repeat Cesarean Section without Labor: Non-medically indicated (elective) cesarean before 39 weeks gestation carry significant risks for the baby with no known benefit to the mother.

Repeat elective cesarean sections before 39 weeks gestation results in higher rates of adverse respiratory distress syndrome (RDS), mechanical ventilation, sepsis, and hypoglycemia for the newborns (Tita,, et. al., 2009). Studies have

found that delivery before 39 weeks even with confirmed fetal lung maturity was associated with increased neonatal morbidity, compared to delivery at 39 to 40 weeks. Recent studies highlight concerns that late preterm and possibly early term deliveries may increase babies' risk of brain injury and long-term neurodevelopmental abnormalities. Approximately 50% of cortical volume growth occurs between 34 and 40 weeks. At 37 weeks, the brain weighs only 80% of the weight at 40 weeks and gray matter volume increases at a rate of 1.4% per week between 36 and 40 weeks. Similarly, there is rapid growth of the cerebellum with approximately 25% of its volume developing after the late preterm period. MRI evaluation in preterm infants has shown an impairment of the cerebellar growth compared to term infants⁵.

- b) Maternal complications of cesareans:** There is considerable evidence that cesarean deliveries put women at risk for obstetric hemorrhage and infection—the most frequent causes of severe maternal morbidity and the two leading causes of maternal death in Kerala. These risks to women's health rise with each additional cesarean surgery.
- c) Re-Laparotomy after Cesarean Delivery:** Improper performance of surgery causing abdominal wall hematomas and intraperitoneal hematomas, failure to identify intraoperative bleeding and improper infection control measures leading to peritonitis are the common indications for relaparotomy following cesarean. Repeat surgical procedures lead to acute kidney injury which can lead to death
- d) Long term risks of primary cesarean** are not visible to practicing obstetricians. However, repeat cesareans, in particular, carry significant risks and complications. Unfortunately, these "future" risks of repeated cesareans are not well appreciated by either obstetricians or the patients. The most serious risk for women undergoing multiple repeat cesarean deliveries is a dramatically increased risk for life-threatening hemorrhage and morbidity due to placental implantation problems, including placenta previa and placenta accreta⁶, adhesions and subacute intestinal obstruction.

Common pitfalls leading to increased cesarean rates

- Underuse of physiologic maternal care
- Elective inductions before 40 weeks of gestation especially with unfavourable cervix.
- Pitfalls in diagnosis of labour and admissions to labour room in latent phase
- Nonuse of partogram leading to misdiagnosis of labor disorders like failure to progress and dystocia.
- Inappropriate ARM and oxytocin use in labor.
- Incorrect interpretation of CTG with misdiagnosis of fetal distress.

- Underuse of instrumental deliveries.
- Not having labor companions to provide continuous support in labor.

Common Errors Leading to Maternal Death in cesarean

- Adequate surgical hemostasis not obtained at cesarean delivery.
- Post cesarean hypotension not aggressively evaluated and treated, including transfusion and/or reoperation.

Pitfalls related to prevention and management of postpartum hemorrhage

Lack of systematic protocol for early recognition and rapid response

Post-partum hemorrhage (PPH) remains the most significant contributor to maternal mortality globally. Kerala is not an exception⁷. Relatively few institutions have created a systematic PPH protocol for early recognition and rapid response.

Active Management of the Third Stage of Labour, including the use of uterotonics, is the recommended standard of practice for all births in order to prevent and treat PPH. The Cochrane Review of four randomized controlled trials examining expectant versus active management concluded that active management reduced risks of the following: 1) maternal blood loss; 2) postpartum hemorrhage exceeding 500 mL; and 3) prolonged third stage labor. In spite of the evidence on the effectiveness of active management in preventing PPH, active management protocols are not being followed routinely in many of the hospitals.

Failure of early recognition is a major pitfall in diagnosing PPH. This is due to

a. Improper monitoring of immediate post partum period

Four hours following delivery is a crucial period. Clear protocols for monitoring this period have to be rigorously employed for early detection of PPH. Failure to monitor is the major pitfall leading to maternal mortality and morbidity

b. Underestimating true blood loss

Objective measures for blood loss is not advocated in most centres. Clinicians typically underestimate true blood loss. Subjective measurement tends to underestimate real loss leading to delay in instituting prompt treatment.

c. Using Vital signs as indicators of blood loss

Another pitfall is using vital signs as indicators of blood loss. Hemodynamic instability shows massive loss and resuscitative measure may not be helpful at this stage.

PPH-related morbidities: include massive transfusions, secondary surgical

procedures, ICU admissions and fertility loss. Most of these morbidities are preventable by early detection and prompt treatment.

Blood transfusion: The need for transfusion is a marker for identification and assessment of maternal hemorrhage which is an indicator of poor management of PPH.

Placenta previa accreta—common pitfalls in managing is lack of appropriate evaluation and/or referral for treatment of possible placenta accreta in women with one or more uterine incisions and a placenta previa _

Can be avoided by the following steps

- Screen all women with prior cesarean birth for placenta previa with ultrasound.
- Screen all women with placenta previa for accreta first with ultrasound, then with MRI if ultrasound results are suspicious or inconclusive.
- Prepare a multi-disciplinary approach for delivery, including a plan for emergency surgery prior to scheduled delivery. Planning should include primary OB surgeon, Blood Bank, perinatologist, anesthesiologist, /experienced pelvic surgeon, labor & delivery nurses, operating room personnel, nursery and pediatric teams.
- Consider early delivery (32-36 weeks) before labor and after pretreatment with betamethasone for fetal lung maturity.
- Strongly consider hysterectomy (without removal of placenta) if no further children desired.
- Ensure immediate availability of 4-6 units of PRBC, FFP, and platelets

Pre-eclampsia. Common Errors Leading to Maternal Death

Failure to manage pre-eclampsia can result in death or serious injury to both mother and baby.

- Delay in diagnosis particularly assessing the severity. IUGR, a sign of severity of preeclampsia is often missed in early stages.
- Delay in referral to higher centres and referring in moribund stage.
- Delay in clinical decision to terminate pregnancy.
- No adherence to protocols for imminent eclampsia and Eclampsia, especially with regard to anticonvulsants.
- Respiratory symptoms in the setting of preeclampsia not aggressively evaluated and treated.
- Improper fluid management

- Improper control of hypertension
- Substandard critical care.

Amniotic fluid embolism—a preventable tragedy?

Experts agree that this condition is not preventable. But awareness regarding risk factors may be helpful in averting amniotic fluid embolism. Induction of labor is a major risk factor for amniotic fluid embolism. Careful selection of cases and judicious use of methods of induction may help. Oxytocin has been implicated as a risk factor⁸. Injudicious use of oxytocin for labor induction and augmentation in labor may be a cause for increasing death due to amniotic fluid embolism. Use of multiple methods for induction and causing tumultuous uterine contraction should be avoided, very careful monitoring of women undergoing induction of labor is to be ensured..

Infection prevention and inappropriate antibiotic use

Lack of proper infection prevention strategies contribute to increasing sepsis as a cause of maternal mortality and morbidity. Infection is the most common serious complication of cesarean delivery. The most important way to reduce hospital infection is for doctors and other medical staff to regularly wash their hands, especially in between examining patients. Appropriate prophylactic antibiotic received within one hour prior to surgical incision in cesarean section reduces the risk of morbidity to the mother and decreases the overall cost of care by avoiding the expense of treating postoperative infections. Other pitfalls include failure in early detection of chorioamnionitis, and failure to screen and treat urinary tract infections. Failure to follow recommended regimen of antibiotic prophylaxis can lead to sepsis and antibiotic resistance leading to multiorgan failure and death due to sepsis.

Recommended regime for antibiotic use in cesarean— is a single dose of cefazolin 1-2 g intravenously within 60 minutes prior to incision. (Strength of evidence for prophylaxis = A). For patients allergic to penicillins or cephalosporins alternative agents may include clindamycin 600 mg plus gentamicin 1.5 mg/kg, ciprofloxacin 400 mg, levofloxacin 400 mg⁹ or aztreonam 1g.

Preventing death due to thrombo-embolism

Failure to advocate proper DVT prophylaxis is a major pitfall in preventing deaths due to embolism.

Conclusions: The above list of pitfalls is only selected few commonly seen in obstetric practice in our state. A critical look at our own day to day practice will help us pick up many more such pitfalls which lead to severe morbidity or even

mortality. Regular audit and protocol based management are the only way to overcome these problems.

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Chapter 26

Blood and Blood Components' Use

Suseela Innah

Blood has been viewed as something special. It has been created with magic qualities of healing properties. People believed blood determined the qualities of an individual and that such quality could be transferred in the blood.

Transfusion therapy in obstetrics affects the pregnant woman, the developing fetus and the neonate. Although various hematological diseases can predispose a pregnant woman to the need for transfusion, even a healthy pregnant woman at term can experience significant blood loss that also requires transfusion. Further various hematological disorders may require that transfusions be given to the developing fetus during pregnancy or to the neonates after delivery.

Blood Component Therapy

Blood component therapy is an integral part of the management of postpartum hemorrhage. The assessment as to when to begin blood product replacement is tailored to the clinical assessment, including the cause of blood loss, the base line hemoglobin and hematocrit, the patient's age, the amount of estimated blood loss, and the rapidity with which the blood loss will be controlled.

Blood and Blood components used in transfusion

A. Whole Blood

- Volume: 350ml / 450ml
- Proportion of anticoagulant (CPD-A1) – 14ml / 100ml Blood
- Hb approximately – 12 g/dl
- HCT – 35-45%
- No functional platelet
- No labile coagulation factors (V and XIII)
- Unit of issue : One donation referred as one 'unit' or 'pack'

- Storage : 2 °C - 6 °C is approved Blood bank refrigerator filter with temperature chart and alarm. Transfusion should be started within 30min of removal from refrigerator.
- *Indications:*
 - Red cell replacement in acute blood loss with hypovolemia
 - Exchange transfusion
 - Patient needing red cell transfer when red cell concentrates or suspension is not available
- *Contraindication :*
 - Risk of volume overload in patients with
 - (a).Chronic anemia
 - (b). Incipient cardiac failure
- *Administration:*
 - Must be ABO & Rh D compatible with the recipient
 - Never add medication to a unit of blood
 - Complete one unit of transfusion within 4 hours of commencement.

B. Red Cell Concentrates ('packed red cells', 'plasma-reduced blood')

- *Description:*
 - 150-200 ml red cells from which most of the plasma has been removed. Hemoglobin approximately 20 g/100 ml (not less than 45 g per unit). Hematocrit 55%-75%.
- *Unit of issue:* 1 donation
- *Infection risk:* Same as whole blood
- *Storage:* Same as whole blood
- **Indications:**
 - Replacement of red cells in anemic patients.
 - Use with crystalloid replacement fluids or colloid solution in acute blood loss.
- **Administration:**
 - Same as whole blood.
 - To improve transfusion flow, normal saline (50-100ml) may be added using a Y-pattern infusion .

C. Red Cell Suspension

- **Description:**
150-200 ml red cells with minimal residual plasma to which +/-100ml normal saline, adenine, glucose, mannitol solution (SAG-M) or an equivalent red cell nutrient solution has been added. Hemoglobin approximately 15 g/100 ml (not less than 45 g per unit).Hematocrit 50%-70%.
- **Unit of issue:** 1 donation
- **Infection risk:** Same as whole blood
- **Storage:** Same as whole blood
- **Indications:** Same as red cell concentrate
- **Contraindications:**
Not advised for exchange transfusion of neonates. The additives solution may be replaced with plasma, 45% albumin or an isotonic crystalloid solution, such as normal saline .
- **Administration:** Same as whole blood. Better flow rates are achieved than with red cell concentrate or whole blood

D. Leucocyte Depleted Red Cells

- **Description:**
 - A red cell suspension or concentrate containing $<5 \times 10^6$ white cells per pack, prepared by filtration through a leukocyte-depleting filter.
 - Hemoglobin concentration and Hematocrit depend on whether the product is whole blood, red cell concentrate or red cell suspension.
 - Leukocyte depletion significantly reduces the risk of transmission of cytomegalovirus (CMV).
- **Unit of issue:** 1 donation
- **Infection risk:** Same as whole blood for all other transfusion –transmissible infections.
- **Storage:** Same as whole blood
- **Indications:**
Minimize white cell immunization in patients receiving repeated transfusions but, to achieve this, all blood components given to the patient must be leukocyte-depleted.
Reduces risk of CMV transmission.
Patients who have experienced two or more previous febrile reactions to red cell transfusion.

- Note: **Will not prevent graft versus host disease.** For this purpose blood components should be irradiated where facilities are available (radiation dose: 25-30Gy).

E. Platelet Concentrates (Prepared from whole blood donation)

- *Description:* Single donor unit in a volume of 50-60 ml of plasma should contain:
 - At least 5.5×10^9 platelets/bag
 - $<1.2 \times 10^9$ red cells/bag
 - $<0.12 \times 10^9$ leucocytes/bag
- *Unit of issue:* May be supplied as either,
 - Single donor unit: platelet prepared from one donation.
 - Pooled unit: platelets prepared from 4 to 6 donor units 'pooled' into one pack to contain an adult dose of at least 240×10^9 platelets.
- *Infection risk:*
 - Same as whole blood, but a normal adult dose involves between 4 and 6 donor exposures.
 - Bacterial contamination affects about 1% of pooled units.
- *Storage:*
 - Up to 5 days at 20°C to 24°C (with agitation) unless collected in specialized platelet packs. **Do not store at 2°C to 6°C**
 - Longer storage increases the risk of bacterial proliferation and septicemia in the recipient.
- *Indications:* Treatment of bleeding due to
 - Thrombocytopenia
 - Platelet function defects
 - Prevention of bleeding due to thrombocytopenia, such as in bone marrow failure.
- *Contraindications:*

Not generally indicated for prophylaxis of bleeding in surgical patients, unless known to have significant pre-operative platelet deficiency.

Not indicated in:

- Idiopathic autoimmune thrombocytopenic purpura (ITP)
- Thrombotic thrombocytopenic purpura (TTP)
- Untreated disseminated intravascular coagulation (DIC)

- Thrombocytopenia associated with septicemia until treatment has commenced or in cases of hypersplenism.
- *Dosage:*
 - 1 unit of platelet concentrate /10kg body weight: in a 60 or 70 kg adult, 4-6 single donor units containing at least 240×10^9 platelet should raise the platelet count by $20-40/\text{mm}^3$.
 - Increment will be less if there is:
 - Splenomegaly
 - Disseminated intravascular coagulation
 - Septicaemia
- *Administration:*
 - After pooling, platelet concentrate should be infused as soon as possible, generally within 4 hours, because of the risk of bacterial proliferation.
 - **Must not be refrigerated before infusion** as this reduces platelet function.
 - 4-6 units of platelet concentrate (which may be supplied pooled) should be infused through a fresh standard blood administration set.
 - Special platelet infusion sets are not required.
 - Should be infused over a period of about 30 minutes.
 - Do not give platelet concentrate prepared from RhD positive donors to an RhD negative female with child-bearing potential.
 - Give platelet concentrates that are ABO compatible, whenever possible
- *Complications:*
 - Febrile, non-hemolytic and allergic urticarial reactions are not uncommon, especially, in patients receiving multiple transfusions.

Platelets Transfusion in Pregnancy

Conditions associated with low platelet count in pregnancy include:-

1. Benign Thrombocytopenia of Pregnancy
2. ITP and Pregnancy
3. Thrombocytopenia associated with hypertension in pregnancy
4. HELLP
5. Thrombotic Thrombocytopenic Purpura and Hemolytic Uremic syndrome
6. Alloimmune Thrombocytopenia

Management:-

1. In BTP Platelet count is repeated each trimester and neonatal platelet count is done. Usually patient does not require any transfusion.
2. In ITP and Pregnancy steroids are given if platelet count is less than 50,000/mm³
3. In HELLP syndrome platelets should be available for transfusion if platelet count is low (20,000 - 50,000/mm³)

F. Platelet Concentrates (collected by plateletpheresis)

- *Description:*
 - Volume 150-300 ml
 - Platelet content 150-500 x 10⁹, equivalent to 3-10 single donations
 - Platelet content, volume of plasma and leukocyte contamination depends on the collection procedure
- *Unit of issue:* 1 pack containing platelet concentrates collected by a cell separator device from a single donor
- Infection risk: Same as whole blood
- *Storage:* Up to 72hours at 20°C to 24°C (with agitation), unless collected in specialized platelets packs validated for longer storage periods; **do not store at 2°C and 6°C**
- *Indications:*
 - Generally equivalent to the same dose of platelet concentrate prepared from whole blood.
 - If a specially typed, compatible donor is required for the patient, several doses may be obtained from the selected donor.

G. Fresh Frozen Plasma

- *Description:*
 - Pack containing the plasma separated from one whole blood donation within 6 hours of collection and then rapidly frozen to -25°C or colder.
 - Contains normal plasma levels of stable clotting factors, albumin and immunoglobulin.
 - Factor VIII level at least 70% of normal fresh plasma level.
- *Unit of issue:*
 - Usual volume of pack is 200-300 ml
 - Smaller volume packs may be available for children

● *Infection risk:*

- If treated, same as whole blood
- Very low risk if treated with methylene blue / ultraviolet light inactivation (see virus 'inactivated' plasma)

● *Storage:*

- At -25°C or colder for up to 1 year
- Before use, should be thawed in the blood bank in water which is between 30°C to 37°C. Higher temperature will destroy clotting factors and proteins. Once thawed, can be stored in refrigerator at +2°C to +6 °C for upto 12 hours.

● *Indications:*

Replacement of multiple coagulation factor deficiencies: e.g.

- Liver disease
- Warfarin (anticoagulant) over dose
- Depletion of coagulation factors in patients receiving large volume transfusions.

Disseminated intravascular coagulation (DIC)

Thrombotic thrombocytopenic purpura (TTP)

● *Precautions:*

- Acute allergic reactions are not uncommon, especially with rapid infusions.
- Severe life threatening anaphylactic reactions occasionally occur.
- Hypovolaemia alone is not an indication for use.

● *Dosage:* Initial dose of 15 ml/kg

● *Administration:*

- Must normally be ABO compatible to avoid risk of haemolysis in recipient.
- **Need not be Rh compatible.**
- No compatibility testing required.
- Infuse using a standard blood administration set as soon as possible. after thawing. Labile coagulation factors rapidly degrade; use within 6 hours of thawing.

Fresh Frozen Plasma is used for massive obstetric hemorrhage and for patients who have demonstrated a factor deficiency or developed DIC. The usual starting dose of FFP is two units. **AB plasma can be used for all blood groups.**

H. Cryoprecipitate

- *Description:*
 - Prepared from fresh frozen plasma by collecting the precipitate formed during controlled thawing at +4°C and resuspending it in 10-20ml plasma. Contains about half of the factor VIII and fibrinogen in the donated whole bloods: e.g. Factor VIII: 80-100iu/bag; fibrinogen: 150-300 mg/bag.
- *Unit of issue:* Usually supplied as a single donor bag or a bag of 6 or more single donor units that have been pooled.
- *Infection risk:* As for plasma, but a normal adult dose involves at least 6 donor exposures
- *Storage:* At – 25°C or colder for up to 1 year
- *Indications:*

As an alternative to factor VIII concentrate in the treatment of inherited deficiencies of:

 - von Willebrand Factor (von Willebrand's disease)
 - Factor VIII (hemophilia A)
 - Factor XIII

As a source of fibrinogen in acquired coagulopathies: e.g. disseminated intravascular coagulation (DIC)
- *Administration:*
 - If possible, use ABO- compatible product.
 - No compatibility testing required.
 - After thawing, infuse as soon as possible through a standard blood administration set.
 - Must be infused within 6 hours of thawing.

Massive Transfusion

Massive blood loss is arbitrarily defined as the loss of one blood volume within a 24-hour period although other, more convenient, definitions include 50% blood volume loss within 3 hours or a rate of loss of 150mL/mt. This degree of blood loss may be associated with significant coagulation abnormalities. Thrombocytopenia can occur reasonably quickly and usually results from dilution, but increased consumption of platelets may also occur. The use of plasma - reduced red cell concentrates can result in significant dilution of coagulation factors.

The management of ongoing bleeding requires both clinical and laboratory input. The inability of standard laboratory tests to keep pace with the clinical picture is well recognized; nonetheless, patients receiving massive transfusion should have routine test of haemostasis performed early in order to define previous abnormalities.

Whilst there is no clear evidence to support transfusion triggers, guidelines do exist in order to prevent the indiscriminate use of component therapy. The British committee for standards in hematology recommends the following:

- Maintain Hb >8 g/dl
- Keep platelets >75 x 10⁹ /L – microvascular bleeding and general oozing from wounds or venepuncture sites are particularly likely when the platelet count falls below 50 x 10⁹ /L.
- Maintain PT & APTT <1.5 x mean control – administer FFP 12-15 ml/kg. Anticipate needs for replacement after 1-1.5 x blood volume replacement.
- Maintain fibrinogen >1.0 g/L – if not corrected by FFP, give two pooled pack of cryoprecipitate or fibrinogen concentrate.
- The need for on going haemostatic treatment should be guided by the patient's clinical response and results of repeated laboratory tests.

Recombinant F VIIa

In recent years, recombinant FVIIa (NovoSeven) has been used for patients with uncontrollable, life-threatening hemorrhage. This product was originally developed for use in hemophilia patients with inhibitors to FVIII or FIX. In the setting of massive blood loss, the evidence for rVIIa use is limited and anecdotal but is increasing. A recent systematic review concluded that the application of rVIIa in severe bleeding is promising and relatively safe with a 1-2% incidence of thrombotic complications. Due to its expense, its use is generally limited to 'rescue' therapy for massively transfused patients with persistent bleeding despite appropriate blood component transfusion, haemostatic and pharmacological measures and surgical intervention. A dosing schedule of 300µg/kg followed by further doses of 100µg/kg at 1 and 3 hours as required is suggested – as the half-life is relatively short, repeat dose may be needed to decrease bleeding significantly.

Before the infusion of Recombinant Factor VIIa the following blood parameters should be corrected:-

1. Platelet Count > 75x10³
2. Fibrinogen>100mg/dl

3. PT & APTT – normal or near normal.

The mainly listed indications for Factor VIIa are the following:-

1. Factor VIII or Factor IX inhibitors
2. Factor VII deficiency
3. Glanzmanns Thrombasthenia

The decision to use this product should generally be made in consultation with a hematologist/ transfusionist.

Disseminated Intravascular Coagulation

In disseminated intravascular coagulation (DIC), the coagulation and fibrinolytic systems are both activated, leading to deficiencies of the coagulation factors, fibrinogen and platelets.

Laboratory Findings

DIC is characterized by:

- Reduced coagulation factors(so all coagulation tests are prolonged)
- Reduced platelet count (Thrombocytopenia)
- Prolonged activated partial thromboplastin time (APTT)
- Prolonged prothrombin time (PT)
- Prolonged thrombin time: particularly helpful in establishing presence or absence of DIC
- Decreased fibrinogen concentration
- Breakdown of products of fibrinogen: fibrin degradation products (FDPs)
- Fragmented red cells on the blood film.

Management

Rapid treatment of removal of the underlying condition is imperative.

If DIC is suspected, do not delay treatment while waiting for the results of coagulation tests.

Treat the cause and use blood products to help control hemorrhage.

Transfusion support should be given to help control bleeding until the underlying cause has been dealt with and to maintain an adequate platelet count and coagulation factor levels.

1. Monitor:

- Activated partial thromboplastin time
- Prothrombin time
- Thrombin time
- Platelet count
- Fibrinogen.

2. Identify and treat or remove the cause of DIC.

3. Give supportive care:

- Fluids
- Vasopressor agents
- Renal, cardiac or ventilatory assistance.

Transfusion

1. If the PT or APTT is prolonged and the patient is bleeding:
 - Replace red cell losses with PRBC <5days old or the fresh whole blood.
 - Give fresh frozen plasma as these contain labile coagulation factors: 1pack/ 15kg body weight (4-5packs in adults)
 - Repeat FFP according to the clinical response.
2. If fibrinogen is low or the APTT or thrombin time is prolonged, also give cryoprecipitate (To supply fibrinogen and factor VIII): (8-10 packs in adults).
3. If the platelet count is less than $50 \times 10^9/L$ and the patient is bleeding, also give platelet concentrates: 4-6 packs (adults).
4. The use of heparin is not recommended in bleeding patients with DIC.

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Chapter 27

Fluid resuscitation in collapse/ shock

P M Jayaraj & Vasanthi Jayaraj

Shock is a physiologic state characterized by a significant reduction of systemic tissue perfusion, resulting in decreased oxygen delivery to the tissues. Prolonged oxygen deprivation may lead to sequential cell death, end-organ damage, multi-system organ failure, and death.

Table 27.1 Pathophysiology and hemodynamic profile of shock states

Physiologic variable	Preload	Pump function	Afterload	Tissue perfusion
Clinical measurement	Pulmonary capillary wedge pressure	Cardiac output	Systemic vascular resistance	Mixed venous oxygen saturation
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Distributive	↓ or ↔	↑	↓	↑

Shock begins with an inciting event. This produces a systemic circulatory abnormality that may progress through several stages: preshock, shock, and end-organ dysfunction. The progression can culminate in irreversible end-organ damage and death. Cardinal features include hypotension, oliguria, cool and clammy skin, abnormal mental status, and metabolic acidosis.

The differential diagnosis for the cause of shock is narrowed by determining the type of shock. Hypovolemic shock may be hemorrhage induced or fluid loss induced. Cardiogenic shock may be due to cardiomyopathy, arrhythmia, mechanical abnormality (eg, valvular dysfunction), or extracardiac abnormality (eg, pulmonary embolism). Distributive shock has many causes, such as septic shock, systemic inflammatory response syndrome, and anaphylaxis.

When a patient is suspected of having shock, diagnostic evaluation should occur at the same time as resuscitation. Resuscitative efforts should NOT be delayed for history, physical examination, laboratory testing, or imaging.

The cause of shock can usually be determined or narrowed to a few possibilities using the data acquired from the medical history, physical examination, and laboratory evaluation.

Mortality due to shock is high.

Management

Introduction

Rapid volume repletion is indicated in patients with severe hypovolemia or hypovolemic shock. Delayed therapy can lead to ischemic injury and possibly to irreversible shock and multiorgan system failure. Three issues generally need to be considered in this setting: the rate of fluid replacement; the type of fluid infused; and the role for buffer therapy in patients with concurrent lactic acidosis. Vasopressors (eg, norepinephrine) generally should not be administered, until enough fluid has been infused since they do not correct the primary problem and tend to further reduce tissue perfusion.

Rate of Fluid repletion

It is not possible to precisely predict the total fluid deficit in a given patient with hypovolemic shock, particularly if fluid loss continues (eg: persistent bleeding or third space sequestration). In general, at least one to two liters of isotonic saline are initially given as rapidly as possible in an attempt to restore tissue perfusion.

Early correction of the volume deficit is essential in hypovolemic shock to prevent the decline in tissue perfusion from becoming irreversible. Irreversible shock is associated with loss of vascular tone, a drop in systemic vascular resistance, pooling of blood in the capillaries and tissues, and an impaired response to vasoactive medications.

Fluid repletion should continue at the initial rapid rate as long as the systemic blood pressure remains low. Clinical signs, including blood pressure, urine output, mental status, and peripheral perfusion, are often adequate to guide resuscitation. The development of peripheral edema is often due to acute dilutional hypoalbuminemia and should not be used as a marker for adequate fluid resuscitation or fluid overload.

A central venous catheter should be considered in patients who fail to respond promptly to initial fluid resuscitation. In such patients, monitoring the central venous pressure can help direct therapy. There is little added value from measurement of the pulmonary capillary wedge pressure, unless the patient has underlying cardiopulmonary disease.

Choice of replacement fluid

The choice of replacement fluid depends in part upon the type of fluid that has been lost. As an obvious example, blood components are indicated in patients who are bleeding. In this situation, the hematocrit should not be raised above 35 percent because a further increase is not necessary for oxygen transport and may increase blood viscosity, potentially leading to stasis in the already compromised capillary circulation.

Colloid versus crystalloid

Both isotonic saline solutions and colloid-containing solutions have been used to replace the extracellular fluid deficit. Concern has been raised about risks associated with the use of the synthetic colloid hydroxyethyl starch. Highly substituted starch solutions have been associated with coagulopathy, while hyperoncotic starch solutions have been associated with increased risk of acute kidney injury.

Saline solutions are equally effective in expanding the plasma volume, although 1.5 to 3 times as much saline must be given because of its extravascular distribution. This is not deleterious, however, since fluid loss also leads to an interstitial fluid deficit that is repaired by saline administration.

Buffer Therapy

Patients with marked hypoperfusion may develop lactic acidosis, leading to a reduction in extracellular pH below 7.10. Sodium bicarbonate can be added to the replacement fluid in this setting, in an attempt to correct both the acidemia and the volume deficit. However, the efficacy of alkali therapy in lactic acidosis is uncertain and its use remains controversial

Large volume resuscitation using normal saline may be associated with the development of hyperchloremic metabolic acidosis. This has led to suggestions that physiologically-buffered fluids (eg, lactated-Ringers solution) be used instead of isotonic saline for large volume resuscitation. However, benefit of this approach has not been demonstrated.

Optimal monitoring of acid-base status may require measurement of mixed venous as well as arterial blood gases.

The protocol for Blood & Blood products transfusion

Two units of packed red blood cells (PRBC) be transfused if hemodynamics fail to improve after the administration of 2 to 3 liters of crystalloids. Further transfusions are given based upon the patient's response to the initial transfusion.

Transfusion of clotting factors and platelets

Treatment of hemorrhage with IV crystalloid and PRBCs increases the risk of coagulopathy from dilution of platelets and clotting factors, and possibly hypothermia. Prevention of coagulopathy with early transfusion of plasma and platelets is critical in the patient with severe hemorrhage. Give one unit of fresh frozen plasma (FFP) for every unit of PRBCs. (four units of FFP are given once four units of PRBCs are given). Give six units of platelets once six units of PRBCs have been transfused

Control hypothermia

Rapid infusion of blood from refrigerator and intravenous fluids at room temperature can lower the core temperature. Hypothermia may impair cardiac output and oxygen delivery to tissues and also can lead to coagulopathy. Hence ideally blood should be run through warming devices and body kept warm with blankets etc.

Monitoring

Hemoglobin:

- Transfuse two units PRBCs if hemoglobin falls below 8 g/dl for patients without risk for acute coronary syndrome (ACS).
- Below 10 g/dL for patients at risk for ACS

Platelets:

- Transfuse six units if level falls below 50,000/microL

Prothrombin Time (INR)

- Transfuse 2 units of FFP if INR rises above 2

Fibrinogen:

- Transfuse 10 units of cryoprecipitate if the fibrinogen level falls below 100 mg/dL

Goals

- **Blood pressure:** maintain MAP above 65 mmHg
- **Heart rate:** maintain between 60 and 100 beats per minute
- **Oxygen saturation:** maintain above 94 percent
- **Urine output:** maintain above 0.5 mL/kg/hour
- **Central venous pressure:** maintain between 8 and 12 mmHg
- **Lactate and base deficit:** Monitor serum lactate and serum bicarbonate every four hours to ensure end-organ perfusion is adequate or improving with resuscitation.
- **Mixed central venous oxygen saturation:** Monitor every four hours to ensure end-organ perfusion is adequate or improving with resuscitation; goal is to maintain above 70 percent.

- **Blood gases**

Arterial Vs Mixed venous - Mixed venous blood gas sample is more appropriate :Arterial values may not reflect the acid-base status at the tissue level in the settings of circulatory failure or cardiopulmonary resuscitation in which the cardiac output is markedly reduced. Maintenance of ventilation allows the blood delivered to the lungs to be normally cleared of CO₂, resulting in a normal or even reduced PCO₂.

In contrast, the mixed venous PCO₂ may be markedly elevated because of decreased systemic and pulmonary blood flow.

Finally a word on the dreaded complication of ARF in the background of shock :

- ARF is common in the setting of Hypotension and shock

Generally kidneys are very resourceful organs, and can withstand significant degree of insult. There is considerable haemodynamic adjustments to maintain the blood supply to the renal cortex at the expense of the medulla, hence the medulla suffers .And this insult to medulla results essentially as acute tubular necrosis (ATN) and rarely, in case of severe and prolonged shock acute cortical necrosis (ACN).

- If properly managed ATN is completely reversible

The basic dictum for reno protection is to maintain **adequate intra vascular volume**. This would be a systolic BP of 90 mm or MAP of 65mm, and avoid nephrotoxic drugs like NSAIDs and aminoglycosides.

Even now lot of ICUs continue to use dopamine (even after BP has stabilized)

hoping that low dose dopamine will improve renal perfusion. This has been proved wrong in a number of well conducted randomized control trials(RCTs) and has been, many a time, proved to have deleterious effects.

So, no renal dose dopamine please!

Chapter 28

Severe Acute Maternal Morbidity(SAMM)

K Ambujam, Lola Ramachandran

The analysis of maternal deaths has long been used as one of the criteria for evaluating women's health and quality of obstetric care. Maternal death is a devastating outcome for the woman's family and society. But this is only the "tip of the iceberg". By evaluating only maternal deaths, other major problems in obstetric care may be overlooked. The continuum of maternal health may be considered as progressing from normal pregnancy through morbidity, severe morbidity and near miss to the final outcome, death. Morbidity is an important outcome which affects many women. In some cases death would have been preventable but survival will be as a cripple, ending as a burden to the family and society.

In recent years the number of maternal deaths in many centres has come down so much that it is no longer meaningful to audit only maternal deaths. Review of morbidity and avoided maternal deaths therefore become more relevant and practical. Also, since an averted maternal death is often a happy ending. There is likelihood of better cooperation from all concerned. For all these reason monitoring maternal morbidity is becoming more and more popular way of auditing maternal health care.

Severe maternal morbidity also known as "Near Miss" is defined as "A very ill pregnant or recently delivered woman who would have died had it not been that luck and good care was on her side". Scrutiny of such cases may be useful for several reasons: larger numbers of cases could permit more simultaneous reporting; lessons to be learned from the management of cases who survived may be at least as useful as from those who died; Severe morbidity case could also be seen as controls for deaths.

Although incidence and evaluation of obstetric complications is presented as a relatively easy alternative to maternal deaths, difficulties remain in their definition and identification. The threshold of severity is easy to define for some conditions e.g. rupture uterus, but may not be straight forward for others eg. vaginal bleeding. An added difficulty is that the threshold above which an adverse obstetric event becomes life threatening may be context specific. Some diagnosis of

severe obstetric complications may be particularly dependent on physician’s judgement. Due to all these difficulties in definition, three approaches have been proposed for defining life threatening obstetric complications. These approaches include definitions based on (a) management (b) clinical signs and symptoms (c) organ systems

(a) Management based definitions

Management criteria most commonly used is admission to ICU, regardless of the medical reason for the admission. Other examples are emergency obstetric hysterectomy, cesarean section, blood transfusion, prolonged hospitalization, anesthetic accidents

(b) Definitions based on clinical signs and symptoms

These are generally built around obstetric diagnosis or complications and tend to focus on the major causes of maternal mortality such as hemorrhage, hypertensive disorders, and sepsis.

(c) Organ system based definitions

A woman with organ failure or organ dysfunction during or within six weeks after pregnancy is very likely to die if she does not receive adequate care. For example a hemorrhage might become life threatening if the bleeding leads to vascular renal or coagulation dysfunction. Infection on the other hand might become life threatening if the woman shows signs of respiratory, immunological or cerebral dysfunction.

Table 28.1 Criteria for inclusion under severe morbidity

No	Category	Definition
1.	Major obstetric haemorrhage	Estimated blood loss >2500ml or transfused 5 or more units of blood or received treatment for coagulopathy (FFP, cryoprecipitate, platelets). Includes ectopic pregnancy meeting these criteria
2.	Eclampsia	Seizure associated with antepartum, intrapartum or post partum symptoms and signs of pre eclampsia
3.	Renal or liver dysfunction	Acute onset of biochemical disturbances, AST/ ALT >200U/l
4.	Cardiac arrest	No detectable major pulse
5.	Pulmonary oedema	Clinically diagnosed pulmonary oedema associated with acute breathlessness and O ₂

		saturation <95%, requiring O ₂ diuretics or ventilation
6.	Acute respiratory dysfunction	Requiring intubation or ventilation for >60 minutes(not including duration of GA)
7.	Coma	Including diabetic coma. Unconscious >12hours
8.	Cerebrovascular event	Stroke, cerebral/cerebellar hemorrhage or infarction, subarachnoid hemorrhage, dural venous sinus thrombosis
9.	Status epilepticus	Unremitting seizures in a patient with known epilepsy
10.	Anaphylactic shock	An allergic reaction resulting in collapse with severe hypotension, difficulty in breathing and swelling/rash
11.	Septicaemic shock	Shock(systolic BP<80mmHg)in association with infection. No other cause for decreased BP. Pulse of 120/min or more
12.	Anesthetic problem	Aspiration, failed intubation, high spinal or epidural
13.	Massive pulmonary embolism	Increased respiratory rate(>20/min), tachycardia, hypotension. Diagnosed as high probability on V/Q scan or positive spiral chest CT scan. Treated by heparin, thrombolysis or embolectomy
14.	Intensive or coronary care admission(CCU)	Unit equipped to ventilate adults. Admission for one of the above problems or for any other reason. Include admission to CCUs

(Source: Saving Mothers' lives 2003- 05. CEMACH)

Studies estimating the incidence of severe maternal morbidity have used different definitions and ways of definition. It was Waterstone et al and Mantel who had laid down criteria to identify women with severe obstetric morbidity. But they excluded those conditions that are difficult to diagnose accurately the examples being pulmonary and amniotic fluid embolism. The World Health Organization (WHO) developed the maternal near miss approach, a tool to uniformly identify near miss cases and evaluate quality of care provided to women presenting severe complications. WHO defines severe obstetric morbidity as a woman presenting with any of the following life threatening conditions and surviving a complication that occurred during pregnancy, child birth or within 42 days of termination of pregnancy.

Clinical Criteria

- Acute cyanosis
- Breathing rate >40 or <6
- Oliguria unresponsive to fluids or diuretics
- Loss of consciousness for >12 hrs
- Unconscious, no pulse/heart beat
- Jaundice concomitantly with preeclampsia
- Gaspings
- Shock
- Coagulation disorders
- Cerebrovascular accident
- Total paralysis

Laboratory criteria

- Oxygen saturation <90% for >60mts
- Acute thrombocytopenia (<50000)
- Creatinine >3.5mg/dl, Bilirubin >6mg/dl,
- Presence of glucose and ketoacidosis in urine
- Lactate >5 PaO₂/FiO₂ <200, pH <7.1

Management Criteria

- Use of continuous vasoactive drug
- Dialysis for treatment of acute kidney failure
- Puerperal hysterectomy due to infection or hemorrhage
- Cardiopulmonary resuscitation
- Transfusion >5 units of red blood cell concentrate
- Intubation and ventilation for a period of >60 minutes unrelated to anesthesia.

The Scottish confidential audit of severe maternal morbidity has been going on since 2001 and their findings of 2003 - 05 were published in Saving Mothers' Lives December 2007. For their audit, in order to bring uniformity to the cases studied, 14 conditions were identified. Cases with any one of these conditions will be included (see table 28.1) under severe maternal morbidity. Minimal data on each case is collected and organised centrally and used to calculate the level of severe maternal morbidity events in the region. This is a model that can be tried in any region.

Common causes of severe maternal morbidity in our state are, hemorrhage, hypertensive disorders and sepsis. Severe anemia also contributes to severe obstetric morbidity. Cesarean section increases the risk of morbidity. Another factor is that severe maternal morbidity is directly linked with poverty. Severe maternal morbidity is measurable and may be a more meaningful way to measure improvements in health care, mainly in view of the larger number of cases and because the woman herself is able to provide information on the event and on the difficulties she had to face. Therefore study of maternal near miss cases has been suggested as a practical alternative to the surveillance of maternal morbidity and mortality.

Practical aspects of Near miss audit

As of now, a state or national level near miss audit is not practical in our country. This type of audit is best performed at the institutional level. The responsibility is on the head of the department to conduct the audit and use it as a tool to improve the care given.

The ultimate aim should be to pick up the cases before they reach the level of severe morbidity. A useful tool to monitor vital signs and take action at the appropriate time is the colour coded early warning chart developed by Aberdeen Maternity Hospital(See CEMACH website www.cemach.org.uk).

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Chapter 29

Emergency Obstetric Care & Basic Life Support

M Bindu, Neetha George

Review of maternal death records revealed that on many occasions appropriate steps were not taken at the initial stages when the problems were reversible.

Typical scenarios are

- Not taking steps to stop bleeding, e.g: condom tamponade for atonic PPH
- Packing for traumatic PPH
- Not bringing down blood pressure in severe pre eclampsia so that the patient ends up with cerebral bleed.
- Not giving adrenaline in anaphylactic shock
- Not providing cardiac compression even after cardiac arrest.
- Not providing ventilatory support in respiratory arrest.

The list is very long. In almost all the cases, the reviewers felt that an alternative approach could have made a difference in the outcome and even the death could have been averted.

These problems are not unique to our set up. Globally, the need for emergency obstetric care and provision of basic life support at the beginning of an obstetric emergency is recognized. Managing obstetric emergencies and Trauma (MOET) course in the UK and Acute Life Support in Obstetrics (ALSO) course in America and Australia were started to remedy this problem.

The CRMD committee had urged the need for such training in the first edition of “Why Mothers Die”. The Kerala Federation of Obstetrics and Gynecology (KFOG) has realized the importance of this step and started a training programme (EMOCALS). The trainers training of this project and a few training sessions are already completed. A committee has been formed under KFOG to oversee this training programme and six regional units have been formed to organize the

training sessions (Thiruvananthapuram, Kottayam, Cochin, Thrissur, Kozhikode and Kannur). Participants in the training sessions will be awarded certificates which will be valid for five years. The topics to be covered in this two day sessions are:

- Medical and surgical management of PPH- including sessions on internal iliac artery ligation, common iliac artery clamps, etc
- Hypertension and eclampsia management
- Cerebrovascular emergencies
- Malpresentations
- Inversion uterus
- Twins
- Instrumental delivery
- Emergencies during CS
- Obstructed labour
- Ist trimester complications
- Cord prolapse
- Adherent placenta and its management
- Jaundice in pregnancy
- Amniotic fluid embolism and pulmonary embolism
- CTG interpretation – latest guidelines
- Blood and Blood products ,Recombinant Factor VIIa; DIC
- Acute collapse in labour
- Cardiopulmonary failure and resuscitation
- Monitors; How to interpret ABG; ECG - Basics
- Respiratory support and ventilatory care
- Neonatal resuscitation
- Fluid and electrolyte imbalance
- Sepsis bundles and antibiotic policy
- Diabetic keto aciodsis

Work stations (with models and manikins)

PPH management	Shoulder dystocia
CTG	Venous access- central and peripheral
Cardioversion and cardiac compression	Monitors
Airway management	Respiratory support

Venous cut down

Instrumental delivery

Emergency trolley

Neonatal resuscitation

MVA, ECG

ABG interpretation

It is hoped that with such training the obstetricians, especially those in the periphery will be equipped to give the proper first aid which in turn will reduce maternal mortality. We are aware that it is not enough to train the obstetrician or medical officer. The nurses and nursing assistants also have to be trained. KFOG has already shown its willingness to help in this respect but the prime move has to come from the authorities concerned and the hospital management.

Annexures

Useful investigations

Betsy Thomas

COAGULATION PROFILE

Hemoglobin (g/dL)	10.5–15.0
Platelet ($\times 10^9/L$)	146–429
Bleeding Time (minutes)	2 – 6
Clotting Time (minutes)	8 – 20
Partial thromboplastin time, activated (aPTT) (sec)	22.6–35.0
Prothrombin time (PT) (sec)	9.6–12.9
International normalized ratio (INR)	0.80–1.09
D-Dimer (ng/ml)	< 500
Fibrinogen (mg/dL)	301–696

ELECTROLYTES

Sodium (mEq/L)	130–148
Potassium (mEq/L)	3.3–5.1
Chloride (mEq/L)	97–109
Bicarbonate (mEq/L)	22–26

LIVER FUNCTION TESTS

Bilirubin (mg/dl)	0.2 – 0.8
Alanine transaminase(SGPT) (ALT)(U/L)	2–25
Aspartate transaminase (SGOT)(AST) (U/L)	4–32
Lactate dehydrogenase (U/L)	82–524
Alkaline phosphatase (U/L)*	38–229

*Alkaline phosphatase will normally be elevated in pregnancy (doubled) due to the contribution from the placenta.

RENAL FUNCTION TESTS

24–hr protein excretion (mg/24 hr)	46–185
Serum Uric acid (mg/dL)	3.1–6.3
Serum Creatinine (mg/dL)	0.4–0.9
Urea (mg/dL)	15 - 40

PIH INVESTIGATIONS

Haemogram including Platelets

RFT including Uric acid

LFT including LDH

HELLP SYNDROME CRITERIA

Serum Bilirubin (mg/dL) > 1.2

Presence of *schistocytes* in peripheral smear

SGOT (U/L) > 72

LDH (U/L) > 600

Platelet Count < 1 lakh / mm³

BLOOD GAS ANALYSIS

pH = 7.36 - 7.44

Bicarbonate (HCO₃⁻) (mEq/L) = 22-26

PCO₂ (mm Hg) = 36-44

PO₂ (mm Hg) = 92-107

Drugs: Useful dosages and preparations

Betsy Thomas

Antihypertensives

NIFEDIPINE: Widely available and cheap. It is effective in severe and acute hypertension. The dose is 10-20 mg TID or QID upto a maximum of 160-200 mg/day in divided doses. Sublingual Nifedipine is not advisable due to sudden hypotension, if at all being used not more than 5 mg at a time.

LABETALOL: More expensive. It is available as tablets and IV preparation. The drug is given IV 20 mg initial dose, doubled every 10 minutes, until the desired BP is achieved or a cumulative dose of 300mg is reached. When used orally it is given at 100mg twice daily, increased up to 400-800 mg /day in divided doses.

HYDRALAZINE: Intravenously it is given in 5-10 mg doses that are repeated at 20-30 minute intervals until the desired BP level is achieved . Orally it is given at 40-200 mg in 2 divided doses.

METHYLDOPA: It is the most widely studied drug used in hypertensive disorders of pregnancy . Orally it is given 2-4 g daily in 3-4 divided doses. It is not the drug of choice when we desire rapid control of blood pressure and in the postpartum period.

Drugs in Acute Hypertensive Crisis

Aim: To bring blood pressure down to 140 systolic and 90 diastolic

LABETALOL I.V

- 20 mg IV , double every 10 minutes till desired BP fall is achieved.
- Maximum not more than 220 mg in one hour.

NIFEDIPINE

- Oral 10-30 mg every 4-6 hours
- Sublingual- not more than 5 mg

(Use only if parenteral antihypertensives not available)

HYDRALAZINE

- 5 mg IV bolus (slowly), repeated every 20-30 minutes

NIMODIPINE

- 60 mg every 4-6 hours orally

NITROGLYCERINE

- 25 mg in 500ml saline. Give 30ml per hour (8 drops/minute)

SODIUM NITROPRUSSIDE

- 50 mg in 500ml saline. Give 15ml per hour (4 drops/minute)

Drugs in Acute Vascular Collapse

- **DOPAMINE**

To give 5 microgram/kg/minute for a 50kg patient, add 200 mg to 500ml normal saline. Give 37.5 ml/hr (10 drops/minute). Titrate.

- **DOBUTAMINE**

To give 5 microgram/kg/minute in a 50kg patient, add 250 mg to 500ml of normal saline. Give 30 ml/hr (8 drops/minute). Titrate.

- **ADRENALINE**

Available as 1mg/ml. Give IV bolus 1ml every 3-5 minutes.

If no IV line is available, dilute 1mg in 10ml saline and give endotracheally.

- **NORADRENALINE**

Add 4 mg to 500ml of 5% dextrose or dextrose saline. Give 75ml/hour (20 drops/minute).

Titrate to get BP of 90 systolic.

Other Drugs

- **ATROPINE**

One ampoule (0.6 mg) give IV bolus. Repeat every 5 minutes till desired effect is achieved. Maximum 5 doses.

- **HYDROCORTISONE**

One ampoule (100 mg) given IV.

DRUGS IN PPH

Prevention

At the delivery of the anterior shoulder, one of the following drugs can be given:

- Oxytocin 10 units IM
- Oxytocin 5 units slow IV bolus
- Methyl Ergometrine 0.2 mg slow IV
- Carboprost (PGF₂) 125 microgm IM (Rule out history of asthma)
- Syntometrine IM (Oxytocin 5 units + Ergometrine 0.5 mg)

Out of these the best oxytocic is oxytocin. The concern about methyl ergometrine is trapped placenta, blood pressure fluctuations and vomiting. All the above drugs except methyl ergometrine may be given, at the latest, immediately after the baby is delivered.

Treatment

- Oxytocin infusion 5 – 30 units in 500 ml Saline at a rate that controls uterine atony.
- Methyl Ergometrine 0.2 mg IV every 30 minutes upto a maximum of five doses. (contraindicated in women with Hypertension)
- Carboprost (Prostodin) 250 øg IM every 15 minutes upto a maximum of 8 doses. (contraindicated in women with Asthma)
- Rectal Misoprost 600 – 1000 øg

Out of these drugs oxytocin, methyl ergometrine and carboprost should be stored in the refrigerator as they are photosensitive and thermolabile and lose the potency within 3 – 6 months.

SEPSIS

Irrational use of antibiotics is showing results in the emerging antibiotic resistant pathogenic organisms. There is no evidence to support routine use of antibiotics in normal delivery. A single dose of first generation cephalosporin, cephazolin 1-2gm IV half hour before surgery after test dose is the prophylactic antibiotic of choice in routine cesarean section. The test dose should be given through the same route as a small dose with all precautions.

Treatment of uterine sepsis

Initial Antibiotics may be any of the following :

- IV Ampicillin – Sulbactam + I.V Gentamicin 5 mg / kg in 2- 3 divided doses + I.V Metronidazole 500 mg 8 hourly.

OR

- IV Piperacillin – Tazobactam 4.75 g 6 hourly + IV Gentamicin 5 mg / kg in 2- 3 divided doses

OR

- IV Cefaperazone – Sulbactam 1.5 g 8-12 hourly + IV Metronidazole 500 mg 8 hourly

OR

- IV Clindamycin 900 mg 8 hourly + IV Gentamicin 5 mg / kg in 2- 3 divided doses
- Gentamicin has to be monitored with RFT. Antibiotics may be later changed based on culture sensitivity reports and patient response.

Index

- 4 T's of PPH 82
- Abruption 75
- Acute kidney injury 168 ,202
- Acute tubular necrosis 170
- Admission test 217
- AFLP 57 , 121,132,137,175
- ALSO 277
- Aminoglycosides 174, 201
- Amniotic fluid embolism 54,97
- Ampicillin 200
- AMTSL 80
- Anaphylactoid junction 101
- Anaphylaxis 212
- Antenatal care 215
- Antenatal class 216
- Anticardiolipin antibody 145
- Antithrombin deficiency 149
- Aorta caval occlusion syndrome 131
- APLA 165,170,178
- Arrhythmias 110
- Arterio vnous malformation 158
- ASD 111,112
- Atonic PPH 71
- Atrial fibrillation 119
- B- Lynch sutures 83
- Blood components 253
- BMV 110
- Burns 212
- Carbapenam 201
- Cardio cerebral resuscitation117
- Cefazolin 202
- Cephalosporins 201
- Cerebral venous thrombosis 161
- Cesarean section 223
- Classical cesarean 229
- Classification of hemorrhagic shock 171
- Coarctation of aorta 106
- Colloid 267
- Common iliac artery clamp 84
- Condom tamponade 83
- CRMD
- Cryoprecipitate 260
- Crystalloid 267
- CT venogram 162
- CTG 247
- Dalteparin 151
- D-dimer 162
- Dialysis 204
- DIC 262
- Drotaverine 51,97,240
- Eclampsia 91
- Ectopic pregnancy 207
- Eisenmenger's syndrome 105,106
- EMOCALS 52,212,277
- End stage renal disease 170
- Endocarditis prophylaxis 118
- Enoxaparin 151
- Episiotomy 219
- Extra amniotic saline 51
- Factor V Leiden mutation 144
- FAST test 157
- FFP 258
- Fluid challenge 172
- Forceps 235
 - Form D 30
 - Form A 29
 - Form B 29
 - Form C 30
- Fosphenytoin 154,161
- Fourth stage of labour 243
- Functional residual capacity131
- Gestational Diabetes 222,242
- Guillain Barre syndrome 147

- H1N1 Pneumonia 56, 181
 Haymann's sutures 83
 HDU 54
 HELLP syndrome 57 , 90 ,130 , 175
 Hepatitis A virus 129
 Hepatitis B virus 129
 Hepatitis E virus 129
 HICC 206
 HIV 214
 HOCM 118
 HUS-TTP syndrome 166,167,175
 Hydralazine 95
 Hyoscine 51,97,240
 Hyperhomocysteinemia 145
 Hyperkalemia 295
 Intracranial hemorrhage 158
 Jaundice 242
 Joel Cohen method 227
 Labetalol 95
 Lactate 269
 LCHAD 133
 Leucocyte depleted red cells 255
 Liver neoplasm 126
 LMWH 119
 Lower segment PPH 51
 Lupus anticoagulant 145
 Lupus nephritis 176
 Magnesium sulphate 53,95
 Manual vacuum aspirator 210,213
 Massive transfusion 261
 Maternal death 32
 MDG - 5 67
 Mendelso n's syndrome 131
 Methergine 80,82,219
 Methotrexate 208
 minor 33
 Misgav Ladach method 227
 Misoprost 82,219
 MODS 197
 MODS 141
 MOET 277
 Monobactam 201
 MR venogram 162
 MRSA 202
 MTHFR mutation 145
 MTP 213
 NASG 50
 Near miss 271
 Near miss 60
 NSAID 174
 NYHA classificaton 116
 Orthopnea 119
 Osceltamivir 181
 Oxytocin 80,82,219
 Packed red cells 254
 Partoram 242,246
 Peripartum cardiomyopathy 105,106
 PGE1 51
 PGE2 71
 Phenobarbitone 161
 Placenta previa accreta 77, 229
 Plasmapheresis 175
 Platelet concentrates 256
 PND 119
 Post natal care 221
 Postpartum cerebral angiopathy 164
 Postpartum collapse 212
 PPH 50,71
 PPH drill 68,81
 PRAKI 165
 PRAKI 57
 Pre eclampsia 91
 Preconceptional counselling 215
 Premarital counselling 215
 Primary pulmonary hypertension
 105, 106
 Prosthetic valve 113
 Protein C deficiency 144
 Protein S deficiency 144
 Prothrombin gene mutation 144
 PSVT 110
 RBBB 114
 Recombinant Factor VII a 261
 Recombinant activated protein C 199
 Red cell suspension 255

Renal cortical necrosis 170
Renal replacement therapy 179
Reversible cerebral vasoconstriction
syndrome 163
RIFLE criteria 170
RPLE syndrome 163
Rupture liver 126
SEARO 27
Seizure 159
Sepsis 170,172,191,198
Sepsis bundles 55
Sepsis management 198
Septic abortion 209
Septic shock 197
Severe acute maternal morbidity
207 , 271
Shock 265
Sickle cell crisis 214
Sildenafil 112
SIRS 197
SLE 167,176
Sodium valproate 161
Stroke 156
Subarachnoid hemorrhage 158
 Suboptimal care- major 33
Suicide 211
Surviving sepsis campaign 55
Syntometrine 80
Tetralogy of Fallot 106
Traumatic PPH 74
Trial of forceps 219
Unfractionated Heparin 119
Vacuum 235
Valethamate 51,97
Venous thrombo embolism 143
Virchow's triad 143
VRE 202
Whole blood 253

Appendix

Appendix A

List of Executive Committee Members 2006 -2009

Chairman : Dr. V P Paily
Secretary : Dr. K.Ambujam
Joint Secretary : Dr.Betsy Thomas
Treasurer : Dr. Lola Ramachandran
Coordinator : Dr.Sheela Paily

Dr.Ajitha Kumari
Dr.Mrs Elizabeth Iype
Dr.T Narayanan
Dr. V Rajasekharan Nair
Dr.C Nirmala
Dr. P K Sekharan
Dr. P K Syamala Devi
Dr. NS Sreedevi
Dr.Sareena Gilvaz
Dr. K Lalitha
Dr.Uma Devi

Appendix B

List of Obstetrician Assessors

Dr.Vasanthi Jayaraj	Dr.K Lalitha
Dr.Kunjamma Roy	Dr.Jayasree Thankachi
Dr.Sheela Shenoy	Dr.D Radhamoni
Dr.Presannakumari	Dr.Rani Santhakumari
Dr.Pramod Roy John	Dr.CP Vijayan
Dr.Hema Warriar	Dr.T N Rajalakshmy
Dr..Lakshmi Ammal	Dr.Sathi
Dr.Girija Gurudas	Dr.Seetha
Dr.Suseela Bai	Dr.Lalithambika
Dr.Hariprasad	Dr.Sumangala Devi
Dr.Deepthy M	Dr.Ajith
Dr.Manjula	Dr.P V Jose

Dr.A V Ramachandran
Dr.Sangeetha Menon
Dr.G Rajalakshmy
Dr.Alice Jose
Dr.Lakshmi S
Dr.Rasheeda Beegum
Dr.Jayanthi Raghavan
Dr.V K Chellamma

Dr. Jyothi Ramesh Chandran
Dr. K Sulochana
Dr.Sheela Balakrishnan

Appendix C

List of Non Obstetrician Assessors

Dr.Viswanath, Dr.P P Mohanan, Dr.Geevar Zachariah, Dr.K.Venugopal
Dr.Praveen, Dr.Jayaraj, Dr.Unnikrishnan, Dr.Gilvaz, Dr.Thomas Iype
Dr.Kuruvilla, Dr.Sanoj, Dr. Mathew Thomas, Dr.A Vimala, Dr.Vijaya Devi,
Padmasree Dr.Vijayaraghavan, Dr.Narendranath,
Dr.Thomas Mathew, Dr.C Gokulan

Appendix D

List of District Coordinators

Dr.Pushpa Bhat, Dr.Krishnakumari, Dr.Hariprasad, Dr.NS Sreedevi, Dr.Hema Warriier
Dr.Kunjumoideen, Dr.Ambujam, Dr.Gracy Thomas, Dr.Kunjamma Roy
Dr.M J Koshy, Dr Rani Santhakumari, Dr.Sathya Babu
Dr.Omana Madhusudhanan, Dr.D Radhamoni, Dr.Beenakumari,
Dr.P Surendran

About the first edition

Hugh Philpott: Many thanks for the copy of your Confidential Review into Maternal Deaths in Kerala. It arrived last evening and I read it through in one reading! It is excellent and underlines the incredible commitment of yourself and the people of Kerala.

Mathews Mathai: Congratulations to you and the team!!! I am happy to note that Kerala has achieved another “first” in maternal health. Will share this document with my colleagues.

Hema Divakar: Congratulations! and thank you for the fine piece of work.

Evita Fernandez: I read with great interest at a rapid pace. Thank you for All the effort put in by you and every member of your team. The information you have given will certainly help us and more importantly, I pray will induce the rest of the country to do likewise.

Lakshmi Seshadri: Great work. I did have a look at the data earlier but looking at the completed document makes a difference. There are tricky areas to tackle. I am glad you have added the case discussions and solutions. We need to come up with our own protocols for some situations- VTE, Amniotic fluid embolism. Can everyone handle IV hydralazine even if available? These are questions to be addressed.

The work is exemplary team work, should be an example for all states. Congrats once again.

Gita Arjun: Excellent work, beautifully presented.



Maternal Fetal Medicine Committee
Kerala Federation of Obstetrics & Gynaecology

