

First Report of Confidential Review of Maternal Deaths, Kerala

# Why Mothers Die

Kerala  
2004-2005



Maternal Fetal Medicine Committee  
Kerala Federation of Obstetrics & Gynaecology

Edited by V P Paily

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Title:

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Maternal Deaths, Kerala 2004 & 2005  
**Why Mothers Die, Kerala 2004 -2005**

Edited by **Dr.V P Paily**

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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 are destroyed after review process is completed.

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## Preface

Maternal death is considered a double tragedy, for, in about 70% of cases, the fetus or newborn also dies perinatally. Even when the fetus survives, its future is often bleak due to the absence of a caring mother. Successful birth and a surviving progeny is a requirement for the survival of the species. In that sense, the woman who consents to get pregnant and reproduce is ensuring the continuation of the species, performing a service to mankind. If, during this process, she is made to suffer physical hurt or loss of life, it could easily be considered a human rights issue. Society and government have a responsibility to ensure the safety of pregnant women.

Unfortunately, child-bearing and child-rearing continue to be a major hazard for women all over the world. In most civilizations, women have a pivotal role in the family. A woman's death or disability has a profound influence on her spouse, their children and society at large. The gravity of the incident doubles when one realizes that most of these deaths are not only due to factors beyond her control but that most are preventable. Hence, maternal death is not merely a health issue; it is a social injustice and a violation of fundamental human rights.

Unequal distribution and utilization of resources around the world has resulted in a wide disparity in income levels and standards of living between geographic regions, resulting in a crude differentiation of the world into developed and developing countries. Just like other health indices, maternal mortality is high in these developing countries. In most regions of Africa, Latin America and Asia, maternal mortality ratios are unacceptably high. India, despite experiencing an economic renaissance currently, is one of 51 countries in the world where measures to reduce maternal deaths are painfully slow and rather ineffective. It is estimated that out of each lakh (100,000) deliveries in India, 407 women die - a figure which existed 50 years ago in the west. This is a result of nothing but blatant social negligence, in a country where the economic growth rate is around 8%.

Kerala has enjoyed health indices much better than what is prevalent in many other Indian States and in many of the countries that became independent alongside India. Accurate statistical data are not available for the period prior to India's independence but whatever data are available seem to indicate better figures for Kerala compared with the rest of India even pre-1947. Currently a girl born in Kerala has a five-fold chance of reaching her fifth birthday and is likely to live 20 years longer than her counterpart in Uttar Pradesh. Maternal mortality also has started to decline rapidly and seems to be about 50 (out of every 100,000 deliver-

ies), a figure that does not exist in any other Indian State.

Kerala's obstetricians and gynaecologists under the banner of the Kerala Federation of Obstetrics and Gynaecology (KFOG) felt it our moral responsibility to try to reduce the maternal mortality ratio in our State. The Confidential Review of Maternal Deaths (CRMD) was started with this objective. This book compiles the experience of the first two years and adds some recommendations for future practice.

This first two years' experience has revealed that there are still many avoidable maternal deaths occurring. They require concerted efforts involving the improvement of transportation, improved training of caregivers, enhancing facilities at hospitals and increasing the availability of blood and components. The community's attitude and involvement also have to change. There are still segments of Kerala society that do not receive antenatal care. We have put down some recommendations based upon the data gathered during the first two years. These recommendations will need wider discussion to evolve a plan of action. Only then will there be the desired impact on maternal mortality within the State.

Kerala's relatively low maternal mortality rates are often attributed to the higher rate of literacy within Kerala, especially among girls. Education was widely promoted by the rulers of Kerala's erstwhile princely States, and also various religious groups through the establishment of schools and colleges throughout the State. A big boost to the social fabric of Kerala society was also given by the Land Reforms Act of 1957 which brought financial security to many families in the lower strata of society. As higher education positively influenced the health-seeking behaviour of Kerala society, more and more hospitals were started within the State. Initially, these were built by the government but with increasing financial independence, private medical care became more prevalent. Today, approximately 70% of all reported deliveries within the State occur in the private sector. Parallel to these changes in the health care delivery system, there was an improvement in the maternal mortality ratio in Kerala and the current figure is believed to be below 50 deaths for every 100,000 deliveries. But before we can boast that we are a civilized society, we must reduce this to single-digit figure within the next decade, making it on par with the other health indices in Kerala. To achieve that, we have to know why mothers die in our State. This book presents the data we could gather through confidential review of maternal deaths in Kerala. There was input from many senior practicing obstetricians and allied specialists in the analysis of the cause of death and the recommendations for managing common conditions. We hope that this book will generate an awareness of the problems and pave the way for further improvement

**V P Paily**

**V Rajasekharan Nair**

## Acknowledgements

The confidential review of maternal deaths in Kerala was possible only due to the dedication and voluntary service of a large number of my obstetrician colleagues in the State. We had been advocating and planning for this review since early nineties, but it was only in 2001 that the pilot study was conducted. Many people need to be recognized and thanked for their efforts to make this possible.

The final push towards the present form of review came from the World Health Organization's South East Asia Regional Office in New Delhi. I should especially mention Dr. Munir Islam, Dr. Ardi Kaptiningsih, Dr. Mathews Mathai and Dr. Arvind Mathur of WHO for all their encouragement.

Dr. Gwyneth Lewis, National Coordinator of Confidential Enquiry into Maternal Deaths in the United Kingdom took a special interest in guiding us and developing the reporting forms. She, along with Prof. Hugh Philpott from South Africa, participated in a workshop to plan the confidential review process for Kerala at Thiruvananthapuram and gave the final go-ahead to the review.

The Maternal and Fetal Medicine Committee of the Kerala Federation of Obstetrics and Gynaecology is the nodal agency that has taken up this pioneering work. Its members have given their time and services voluntarily, and they deserve my special thanks. The colleagues who forwarded the cases to us have to be specially thanked. It is because of their co-operation that we all can learn and try to avoid further maternal deaths.

Members of the editorial board need to be thanked profusely. It would not have been easy to spare the time and effort for this book because all of them are very busy clinicians.

The large number of assessors and coordinators (zonal and district) deserve our special thanks. Few people know that they spent from their own pockets to attend the many meetings. Support from seniors like Dr. Mrs Elizabeth Iype and Dr. K Lalitha was a great inspiration. The biggest encouragement was from the non obstetrician colleagues who acted as assessors and contributed chapters. I am indebted to them.

The Government of Kerala, represented by the Secretary for Health and Family Welfare and the Director of Health Services, extended its support and issued orders in 2004 to all District Medical Officers of Health requiring them to inform



me of any maternal death that occurs. The ministry of health and family welfare, especially the directorate of health services co-operated and encouraged the conduct of this review.

Dr. Sheela Paily voluntarily handled all the background work and provided secretarial assistance since the inception of the review process. She also took the responsibility of compiling all the data and mailing the forms. Our heartfelt gratitude goes out to her for her tireless efforts. This book would not have materialised without her support.

I am most indebted to the Executive Committee Members, especially Dr. V. Rajasekharan Nair, my co-editor, Dr. Ambujam the secretary, Dr. Lola Ramachandran the treasurer and Mr. Ravi the office superintendent for their constant support. A host of other people also chipped in to make the endeavor possible. Engineer George John helped with the computer programme. The financial support was generated through workshops conducted at Jubilee Mission Hospital and Mother Hospital. The management and staff and my colleagues in the O&G departments of these hospitals and the Govt. Medical College helped in the making of this book directly or indirectly.

Mr. T.L. David of Smriti Design, Thrissur deserves my special thanks. He had to readjust his schedules and work late into night to meet the deadline.

Finally, we are very grateful to the Hon. Minister for Health and Family Welfare, Govt. of Kerala, Smt. Sreemathy Teacher for honoring and recognising us by releasing the book during the 31st All Kerala Conference of Obstetrics & Gynaecology at Kozhikode. We hope that the support of the Ministry of Health will continue for our activities aimed at improving the health and welfare of women and children of Kerala.

**Dr. V. P. Paily**

State Coordinator

Confidential Review of Maternal Deaths, Kerala

## Introduction

Maternal mortality ratio (MMR) reflects not only the standard of health care of a community but its educational and social standing. Reduction of maternal death is a high priority for any community. The millennium development goals of UN declares that the MMR should be brought down to 1/4 of the rate in 1990, by 2015. Government of India has targeted to bring down maternal death ratio to 110 by 2015. The Rural Health Mission and the eleventh five year plan aim to achieve this.

Kerala, fortunately has achieved these national goals long ago. But compared with the existing maternal death figures of the developed countries, Kerala's MMR is still about 10 times higher. Also, compared with other indices of health like life expectancy and infant mortality rate the MMR is relatively high. The fact that the developed countries have achieved MMR of single digits makes it clear that this is achievable.

Kerala has many factors that will help to achieve good health indices including low maternal mortality ratios. The people of the State are by and large health and hygiene conscious. The habit of regular bathing, many even twice a day, is only an indication of that. The family structure is such that close relatives support when one is in need of medical care. The high literacy, availability of large number of hospitals, good transport and communication facilities, all help in maintaining high standard of health. Still we cannot claim that all avoidable maternal deaths are eradicated. A concerted effort is required to achieve this. The first requirement to reduce the mortality ratio further is to know the exact causes of these maternal deaths and the circumstances surrounding them. These facts will come out only if there is anonymity for the treating team. There should be immunity from punishment, legal action and loss of one's image in the society and among colleagues. We felt that Confidential Review of Maternal deaths is the right track to follow. This book is the result of our early experiences in conducting Confidential Review of Maternal Deaths in the State of Kerala.

## ACRONYMS USED AND THEIR EXPANSIONS

ACE	Angiotensin Converting Enzyme inhibitors
AF	Atrial Fibrillation
AFE	Amniotic Fluid Embolism
AFLP	Acute Fatty Liver of Pregnancy
AFV	Amniotic Fluid Volume
AMI	Acute Myocardial Infarction
AR	Aortic Regurgitation
ARDS	Acute Respiratory Distress Syndrome
ARM	Artificial Rupture of Membranes
AS	Aortic Stenosis
ASA	American Society of Anesthesiologists
ASD	Atrial Septal Defect
BMI	Body Mass Index
BMV	Balloon Mitral Valvotomy (PTMC & BMV are same)
CEMD	Confidential Enquiry into Maternal Deaths
CRMD	Confidential Review of Maternal Deaths
CPR	Cardio Pulmonary Resuscitation
CSE	Combined Spinal Epidural
CSF	Cerebro Spinal Fluid
CT	Computerised Tomography
CTG	Cardio Toco Graph
CVP	Central Venous Pressure
DC	Direct Current
DHS	Director of Health Services
DIC	Disseminated Intravascular Coagulation
DMO	District Medical Officer
ECC	External Cardiac Compression
EF	Ejection Fraction
EFM	Electronic Fetal Monitoring
FFP	Fresh Frozen Plasma
FH	Fetal Heart
FHR	Fetal Heart Rate
FIGO	International Federation of Obstetrics & Gynaecology
FRC	Functional Residual Capacity
GA	General Anaesthesia
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
HIV	Human Immunodeficiency Virus
ICU	Intensive Care Unit
INR	International Normalised Ratio
IPPR	Intermittent Positive Pressure Respiration
KFOG	Kerala Federation of Obstetrics & Gynaecology
LMWH	Low Molecular Weight Heparin
LSCS	Lower Segment Caesarean Section
MFMC	Maternal Fetal Medicine Committee
MMR	Maternal Mortality Ratio
MODS	Multiple Organ Dysfunction Syndrome
MS	Mitral Stenosis
MV	Mitral Valve
MVP	Mitral Valve Prolapse
NIBP	Non Invasive Blood Pressure (Monitor)
NS	Normal Saline
NYHA	New York Heart Association
OT	Operation Theatre
PA	Pulmonary Artery
PDA	Patent Ductus Arteriosus
PDPH	Post Dural Puncture Headache
PIH	Pregnancy Induced Hypertension
PPCM	Peri Partum Cardio Myopathy
PPH	Post Partum Haemorrhage
PSVT	Paroxysmal Supraventricular Tachycardia
PTMC	Percutaneous Trans Mitral Commissurotomy
RHD	Rheumatic Heart Disease
RL	Ringer Lactate
ROM	Rupture Of Membranes
RUQ	Right Upper Quadrant
SEARO	South East Asia Regional Office(of WHO)
SGOT	Serum Glutamic Oxaloacetic Transaminase
SGPT	Serum Glutamic Pyruvic Transaminase
SLE	Systemic Lupus Erythematosus
TC	Total Count
UFH	Unfractionated Heparin
UK	United Kingdom
VSD	Ventricular Septal Defect
VTE	Venous Thrombo Embolism
WHO	World Health Organisation

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**Why Mothers Die**  
**Kerala - 2004-05**

Part-One

**THE SETTING**

1





## *Chapter One:*

# Background

## Why and how did we start CRMD in Kerala

### 1.1 Why did we start CRMD?

Even though Kerala enjoys relatively better maternal mortality figures than the rest of India, exact data are not available despite the fact that the registration of deaths and births has been consistently high in Kerala for a long time. Calculations of maternal mortality had to depend on sample surveys and hospital statistics obtained from medical schools. Unfortunately, these hospital statistics cannot be extrapolated to the larger community as the hospital drainage area and the populations served are not clearly demarcated. However, a sample survey in 1997 put Kerala's Maternal Mortality Ratio (MMR) as 87 maternal deaths for every 100,000 live births (87/100,000).

These maternal mortality figures are much better than those for the rest of the country, given that the all-India average is 450 maternal deaths per 100,000 live births. However, we felt that the Kerala figures were still too high and could be reduced.

Unlike the rest of the country, Kerala has many factors in its favour that can help lower its MMR even further: high female literacy, the widespread availability of transportation and communication facilities, and a public willing to seek medical aid and pay for it, are only a few. The good doctor-patient ratio also helps in ensuring quick medical aid during emergencies. Despite all these, Kerala's MMR is approximately 10 times that of developed countries. The United Kingdom, for instance has an MMR of 12 while the United States' MMR is 8.4.

The people of Kerala are aware of the health needs but many are not able to avail of those facilities due to financial reasons. Insurance coverage and social security are not widely available especially for maternity care. The impact of all these is maximum in the lower stratum of society. The poor and unemployed can't afford the private health care and unless the government support comes in, the care they get can become suboptimum. This is found to reflect in the maternity care and maternal death statistics. To put the scant resources to best use and improve

the health care provided to the pregnant women, we have to know the exact cause and circumstances of maternal deaths in our State. This book, we hope, will fulfill that vital need.

The only way to bring Kerala's MMR down to a more acceptable level is to discover the major underlying causes for maternal death in the State, then work on a communication and training program within the State to educate obstetricians on these common causes and steps to be taken to prevent them, and also to invite the attention of the State government to specific areas that require government-led remedial measures, such as policy or legislative changes.

In an attempt to discover the actual causes of maternal death in the State, we conducted a pilot study in 2001, collecting data on maternal deaths from obstetricians across the State via personal communication with each obstetrician. We collected the details of 105 maternal deaths that occurred in the State that year. Analysis of the same, as reported by the obstetricians, revealed the causes as depicted in the table 1.1

Table 1.1 Main causes of maternal death in 2001

Total reported	105
Haemorrhage	23
Hypertension	15
Jaundice	10
Heart disease	10
Amniotic fluid embolism	6
Pulmonary embolism	2
Sepsis	6
Criminal abortion	4
Anaesthetic	2

In 2002, the Kerala Federation of Obstetrics and Gynaecology (KFOG) was formed and under its auspices, the Maternal Fetal Medicine Committee (MFMC) was set up. The committee felt the urgent need to identify the factors behind this paradox of high maternal mortality figures against the background of other good health indices. Various ways and means already tried out internationally were considered but it was felt that the Confidential Enquiry into Maternal Deaths (CEMD) which is successfully practised in the United Kingdom for more than half a century was the most suitable for Kerala's setup.

The most important aspect of the U.K. system was that practicing obstetricians were given the responsibility to conduct the enquiry. The government acted only

as a facilitator. There was no punishment handed out to doctors and anonymity was ensured. There was also no threat of legal action so that frank opinions and unbiased criticisms were possible. Thus the United Kingdom's CEMD became a fact-finding mission rather than a fault-finding exercise.

## 1.2 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 U.K. CEMD - for one month in December 2003. After revising the data collection and analysis methodology based on feedback received, the actual review began from 1<sup>st</sup> January, 2004.

This book is the result of the first two years (2004, 2005) of the Kerala CRMD.



## *Chapter Two*

# How does the system (CRMD) work

## 2.1 Data collection

Various measures were introduced, starting in October 2003, to ensure the exhaustive collection of data on maternal deaths in the State.

As per an existing government order, all District Medical Officers of Health – DMO (H) – are required to inform the State Director of Health Services (DHS), of any maternal death that occurs in his or her district within 24 hours of the incident, and submit a detailed report after investigation within seven days. The DHS instructed the DMO (H) to send carbon copy of these maternal death reports to the State Coordinator at the same time as he/she submits them to the DHS.

The Government of Kerala also issued an order requiring all medical officers in the State to inform the State CRMD Coordinator whenever a maternal death occurred under his or her care.

A CRMD District Coordinator was appointed in each district to inform the State Coordinator about any maternal death within the district. This information may reach the District Coordinator from colleagues, newspaper reports, or by hearsay. Thus, through different sources, the State coordinator comes to know about maternal deaths throughout the entire State.

Unfortunately, despite the many channels for communicating deaths to the State Coordinator, a sizeable percentage of deaths are still not being reported. The reasons for these are many. Some indirect maternal deaths occur in other departments, e.g. cases where tuberculosis or a pre-existing heart disease complicated the pregnancy. Some deaths occur in super-specialty departments where patients are admitted for management of complications resulting from obstetric events e.g. a patient could die in a nephrology ward due to renal failure that developed as a consequence of post-partum hemorrhage (PPH).

Initially, there was apprehension on the part of some hospital administrators whether or not confidentiality would be maintained, making them hesitant to disclose details of maternal deaths that occurred in their hospitals. Over the years this resistance has disappeared.

Patients from districts bordering neighbouring states may have their treatment in

those states and since the deaths occur outside Kerala, they do not get reported. It is obvious that, there is an urgent need to tighten the data collection system.

## 2.2 Review Process

When a maternal death occurs and the State Coordinator is informed, he sends the reporting forms (Form A and Form B, see Appendix E) to the concerned doctor.

The reporting doctor fills up the two forms and returns them to the Coordinator along with anonymised photocopies of all the case records. Since the name of the hospital, name, address and number of the patient, and names of the treating doctors and other staff, are to be concealed, this is often a difficult task. Each page has to be scrutinized and the names covered with paper or white ink before making photocopies. The reporting doctor has to do this.

Form A contains the name and other details of the patient. The State Coordinator allots a code number to the patient on the basis of various personal details. The key to decode this unique number is known only to the State and Zonal Coordinators. Once the code is allotted, it is entered in the photocopy of the case records and Form B. Thereafter, Form A is filed separately.

Form B is the reporting form wherein the treating doctor enters all the details of the patient, and his/her own impressions about the cause of death. She/ he is asked to reflect on the case and suggest if any alternative approach might have made a difference to the outcome and a maternal death averted. It is explained that this unfortunate event might possibly become a learning experience. Since anonymity is guaranteed, it is expected that the treating doctor would not hesitate to make an honest assessment.

Form B also contains epidemiological and social aspects of the case to be analysed at the central level. If there are segments of society or regions suffering disproportionately high maternal death rates, this will come to light from the data collected in this section of the form. This information can then help in the design of remedial action specifically focused on that segment of the population.

The State Coordinator scans through the case records and reporting forms and makes his own assessment as to the cause of death. The copies are then dispatched to the assessors.

Assessors are chosen from different parts of the State, in areas away from where the death occurred, to safeguard the anonymity of the individuals involved. Usually, practicing obstetricians of good repute are chosen. They generally come from the State medical colleges and general health services. Practicing doctors from government and private hospitals are also included.

There are two groups of assessors. One comprises the Zonal Coordinators and

senior professors at State medical colleges. They constitute the Executive Committee and total about 13. 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 Assessors Form (see Appendix E) 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 cardiology, nephrology, neurology, gastroenterology and psychiatry, who serve as non-obstetric 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 Executive committee. 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.

Assessors are not given any remuneration for their work. The incidental expenses involved in mailing and for travel to the quarterly meetings are borne by them. They render their time and services voluntarily and freely to this cause. Many of them acknowledge that this is also a great learning experience for them.





## *Chapter Three:* **Definitions Used**

### **3.1 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.

**Three 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.

### **3.2 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.



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Part-Two

**IN SUMMARY**

2



## *Chapter Four:*

# **Findings of the First two Years of CRMD**

## **A. THE DATA**

### **A1: Deaths reported to CRMD**

#### **A.1.(a) Causes of Death**

The main purpose of this review (enquiry) can be summarized as an attempt to find out the causes and circumstances of “why mothers die” and an offshoot from that as to what steps can be taken to avoid such deaths. Hence, it is crucial to know not just the medical cause of death but the circumstances. If we have to bring about any substantial improvement our search should go beyond the medical aspects but is not easy for CRMD. Still we have tried to get some background information which is given under the epidemiological aspects 4.A.1(b). Also, knowing the medical cause and with our knowledge of the social background, it is not difficult to point out the possible contributory factors.

The causes of maternal death are in table 4.1. We have given separately the causes reported directly to CRMD and the ones to the Director of Health Services. When the same case is reported to CRMD and DHS, it will be included only under CRMD. For analysis we have limited ourselves to deaths reported to CRMD because details are available only about them.

We cannot help lamenting on one other snag in our review process. In majority of the cases the reports to CRMD are from the centre where death occurred. Often patients reached such centres after the complications had developed. Unless the circumstances and details of management at the primary centre are available, we cannot arrive at any meaningful conclusions for further guidance.

In interpreting the cause of death it should be remembered that what is given is the primary cause even though final cause of death also may be relevant. For example if a patient had severe PIH with eclampsia and coagulation failure etc. and was saved initially but succumbed after a few days to pulmonary embolism while recovering in the ward, it can be argued that the cause of death should be pulmonary embolism. We have taken the stand that it should be under Hypertensive disorder because that was the primary cause which set the ball rolling.

Table 4. 1: Number of maternal deaths by year reported to CRMD and DHS, and their primary causes

Primary Cause of Death	2004		2005	
	CRMD	DHS	CRMD	DHS
Amn. Fluid Embolism	11	6	8	5
Anaemia	1	4	3	3
Anesthetic	4	0	0	0
Cerebral haemorrhage	2	0	1	0
Ectopic pregnancy	2	1	2	1
<b>Haemorrhage</b>	16	13	22	10.
APH	3	2	5	
PPH	12	10	14	
Pl.previa	1	(accreta)1	3	
Heart Disease	3	9	12	5
Hepatobiliary Diseases	9	1	6	1
Hypertensive Disorders	12	7	17	5
Infections (other systems)		3	2	2
Abortion	0	0	3 (MTP)	0
Sepsis	3	4	7	5
Venous thromboembolism	7	6	3	2
Lupus Syndrome	2	0	0	0
Malignancy	0	3	0	0
Accident	1	1	0	1
Suicide	2	1	0	3
Chronic renal problems	0	2	0	1
Diabetes	0	0	1	0
Homicide (burns)	0	0	1	0
PPS complication	0	0	1	0
Toxic epidermal necrolysis	0	0	1	0
ARDS	0	1	0	1
Asthma	0	1	0	2
Pneumothorax	0	0	1	0
Unknown	4	12	0	15
<b>Total</b>	<b>79</b>	<b>75</b>	<b>91</b>	<b>62</b>

### 4.A.1.(b) Epidemiological Aspects

One of the goals we set for ourselves was the collection of patient data with a view to identify whether there are particular areas or groups that suffer a disproportionately large number of deaths. Various data - like the place of antenatal care, the district where the maternal death occurred, the educational and employment status of the deceased and her husband - were collected and analyzed for this. Not surprisingly, data was often not available, as the reporting doctor did not know these details. However, whatever information was available has been compiled here so that future trends can be monitored.

### Distribution of Deaths by District

The district-wise distribution is shown in Table 4.2. The relatively low number of deaths in District 1 - Thiruvananthapuram - is due to the fact that during 2004 some hospitals in the district did not report maternal deaths to the State Coordinator of CRMD. However, deaths were reported directly to the District Medical Officer of Health. Since the beginning of 2005 however, Thiruvananthapuram has started to cooperate with the CRMD reporting system.

Another anomalous fact is the larger number of cases from Districts in the middle of the State viz Ernakulam, Thrissur, Palakkad, Malappuram and Kozhikode. This reflects the better reporting in these Districts rather than the absence of maternal deaths in the north and south of Kerala.

Table 4. 2 District wise distribution of maternal deaths

District	2004		2005		District	2004		2005	
	CRMD	DHS	CRMD	DHS		CRMD	DHS	CRMD	DHS
Thiruvananthapuram	0	7	10	3	Thrissur	12	3	11	4
Kollam	1	6	3	5	Palakkad	12	7	18	4
Pathanamthitta	2	2	2	2	Malappuram	14	12	8	6
Alleppy	4	nil	3	nil	Kozhikode	8	13	9	13
Kottayam	7	3	6	8	Wayanad	1	5	10	3
Idukki	2	2	2	nil	Kannur	3	4	nil	10
Ernakulam	9	3	8	4	Kasaragod	4	8	1	nil

### Level of hospital where Deaths Occurred

Table 4.3 shows the distribution of maternal deaths by the level of hospital where they occurred. It is not surprising that the vast majority of deaths occurred in Level 3 hospitals. This reflects the fact that most of the critically ill patients are referred to such centres.



Table 4.3 Where did the death occur (Type of Hosp)

Level of hospital	2004	2005
PHC	0	0
Level 1	1	13
Level 2	13	5
Level 3	55	72
Home	0	0
On the way	5	2
Others	2	0

Seven (7) patients died while in transit from the periphery to the major centres and were brought dead to the higher centre. This could be due to many factors – a delay in decision-making or a delay due to the lack of availability of transport. Another possible reason could be the severity of the mother's condition that necessitated her transfer in the first place but also led to her death along the way.

### Type (government or private) of Hospital where Deaths Occurred

The type of hospital, divided into government and private, where the maternal deaths occurred is shown in Table 4.4. The ratio of government to private hospitals is roughly reflective of the current patient load between the government and private sectors in the State.

Table 4.4 Where did the death occur

	2004	2005
<b>Government</b>	30	61
<b>Private sector</b>	46	28

### Level of referring hospital

Tables 4.5 and 4.6 indicate the level of hospital from where patients were referred. The data presented in these tables do not reflect any trend of significance.

Table 4. 5 Level of Hospital

Level of hospital	2004	2005
PHC	2	1
Level 1	17	38
Level 2	15	15
Level 3	1	3

Table 4. 6 If referred, was it from a Private or Govt. Hospital

	2004	2005
<b>Government</b>	15	23
<b>Private sector</b>	21	34

## Age at Death

The data in Table 4.7 shows that the vast majority (%) of the maternal deaths occurred in the age group 20 to 29 years. There is a disproportionately high representation of the older age group (above 30 years) but this cannot be confirmed until we learn the age distribution of obstetric patients in the State, which is not available. However, the fact that 47 out of the 170 deaths occurred in women who were of the age 30 years and above point to the possibility of a higher complication rate within the older age group.

Table 4.7 Age at death

Age	2004	2005
<20	5	4
20-29	52	59
30-39	22	25
40 and above	0	3

## Educational Status

Even though Kerala is a fully literate State, eight patients in the present series were reported to be illiterate. Thirty nine had only primary education (see Table 4.8). A poor educational level often goes with poor socioeconomic status and an inability to afford hospital costs. It is not surprising therefore that there is an overrepresentation of women with only primary education among the deceased.

Table 4. 8 Mother's Educational Status

Educational Status	2004	2005
<b>Nil</b>	2	6
<b>primary</b>	20	19
<b>Secondary</b>	27	29
<b>Degree and Plus</b>	5	6

## Husband's Occupation

The husband's occupation (Table 4.9) usually reflects the paying capacity of the patient. In the study group, the majority of their husbands were manual labourers, another factor reflecting the poor paying capacity and social status of the family.

**Table 4. 9**

Husband's occupation	2004	2005
Unemployed	4	0
Manual labour	36	32
Office work	6	2
Business	6	5
Executive	1	0
Teacher	1	2
Farmer	1	3
Gulf employed	4	6
Other jobs	0	9

## Mother's Occupation

Only a handful of the deceased were employed outside the home (Table 4.10). The absence of the deceased's gainful employment outside the house may also be an index of the paying capacity of the family.

**Table 4. 10 Mother's occupation**

occupation	2004	2005
House wife	58	56
Manual labour	1	4
Office work	2	2
Nurse	1	1
Student	1	4

## Religion

The ratio of the three religious groups (Hindus, Muslims, Christians)table 4.11 reflects roughly the ratio of the religious distribution in the State. It indicates that, on the basis of religion, there was no disparity in the care received.

**Table 4. 11**

Religion	2004	2005
Hindu	42	44
Muslim	24	21
Christian	13	11

## Distance from Nearest Health Facility

Table 4.12 shows the distance of the deceased's residence from the nearest health facility. For the majority (72%) a health facility was available within 10 kilometers of their home.

**Table 4.12**

Distance	2004	2005
<5KM	10	14
5 to 10	21	23
> 10 KM	12	7

## Gravidity and Parity

Most Keralites have taken to the small family norm and so it is not surprising that the vast majority of deaths belong to low gravidity and parity. (see Tables 4.13 and 4.14).

The fact that twenty eight (28) of the 170 cases were of gravida 4 or more and that twenty eight (28) were more than para 2 can be meaningfully interpreted only if we know the average gravidity and parity of obstetric patients in the State. Regarding parity an explanatory note is relevant. Unlike the pattern followed in the South African Confidential Review, we have considered the current pregnancy also in calculating the parity, if death occurred after delivery.

**Table 4.13 Gravidity**

gravida	2004	2005
G1	20	35
G2	34	24
G3	14	15
>G3	11	17

Table 4.14 Parity

Parity	2004	2005
Nullipara	8	6
Para 1	26	47
Para 2	30	20
>para 2	10	18

### Period and Type of Gestation when death occurred

The majority (101 out of 163) of patients died at a gestational age of more than 36 weeks (see Table 4.15). It was reassuring to discover that only nine (9) early pregnancy deaths occurred and only 3 primarily due to abortion. There were three early pregnancy deaths due to causes like lupus syndrome, burns etc. 124 out of 170 (73%) deaths occurred postpartum even though for many of them the problem started during the intrapartum period (Table 4.16)

Table 4.15 Period of gestation when death occurred

Period of Gestation	2004	2005
<20wks	3	4
20 wks to <30	6	5
30 - 36wks	13	23
>36wks	47	54
Ectopic	2	2
Unknown	1	3

Table 4.16 Status of pregnancy at the time of death

	2004	2005
Ectopic	2	2
Abortion	5	4
Antepartum	8	5
Intrapartum	10	10
Postpartum	54	70

### Type of Delivery

Table 4.17 shows the distribution of cases by the type of delivery. Sixty one (61) of the deliveries were by caesarean section. This figure does not imply that the cae-

sarean section was responsible for the maternal death; in most situations, the caesarean was incidental. Twenty four (24) patients died undelivered.

Table 4.17

Type of delivery	2004	2005
Undelivered	12	11
Spont.vag	25	18
Assisted vag	9	18
CS elective	8	6
CS emergency	16	32
Abortion	5	4
Ectopic	2	2

### Type and Place of Antenatal care

Table 4.18 shows that in eighteen (18) cases, the patient received no antenatal care. It is worth taking note that none of the cases had received antenatal care exclusively by midwife or nursing staff, thereby confirming the trend in Kerala society where antenatal care is almost always hospital-based. Seventy six (76) of the cases attended private hospitals, again reflecting the trend in the health care preferences in the State.

Table 4.18 Where was A/N care)

None	4	14
Midwife	0	0
Private H	41	35
Govt.H	19	33
Doctor's consult	6	13

### Neonatal outcome

Table 4.19 shows that out of the 105 known situations, 67 newborns were delivered alive and taken home, while seven died neonatally. The long-term survival of the 67 infants is not known.

Table 4.19

Neonatal outcome	2004	2005
Alive and well	33	34
Alive but NND	5	2
Stillbirth	12	19

### **4.A.2 Deaths reported only to DHS/DMO**

A substantial number of deaths were reported only to the DHS/DMO and not to the State Coordinator. This is a matter of concern as it indicates failure of the reporting system in the CRMD. The tragedy is that this happened in spite of directives from the DHS to all DMOs of Health that every maternal death should be notified to the state coordinator of CRMD.

The cause of death noted down in these reports are not as reliable as in the case of death reviewed by the CRMD. However, this gives valuable insight to the reasons for maternal deaths and their distribution in different districts of the State.

In a significant number of cases the cause of death is unknown. Of the known causes of death PPH and heart disease lead the list followed closely by Amniotic Fluid Embolism.

District wise distribution of deaths reported to DHS is given in table 4.2 and the cause of death in table 4.1. The cause of death recorded in the reports to DHS are usually the ones given by the field staff whereas the cause of death in the CRMD list are the ones arrived at by the assessors after detailed study of the case records. Obviously, there will be differences

### **4.A.3 Deaths known to have occurred but not included for want of details**

There are a few deaths which were known to the state coordinator but a report about them or details were not available.

A total of 11 deaths came under this category. They were not included in the final analysis but remained as a separate group. About three of them were from border districts and died in hospitals outside the State. In some cases the concerned doctors failed to respond in spite of many letters. Some were deaths known through paper reports but a doctor responsible could not be identified.

## 4B Overview of Confidential Review of Maternal Deaths (CRMD) for 2004 & 2005

The first two years of Confidential Review of Maternal Deaths in Kerala (CRMD) has brought out some valuable data which can form the basis for future planning and activities on the health front.

The most striking observation is the lower number of maternal deaths reported than what was expected based on sample surveys. This holds true even after allowing for potential deficiencies in our data collection methods which are discussed in another section (Chapter 2). The various causes of death identified are listed in Table 4.1. 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 directly 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 CRMD committee is conscious of the deficiencies in data collection which would have resulted in incomplete reporting. Even after addition of data collected by the Director of Health Services, there could be omissions. This points to an all-round slackness in reporting maternal deaths. The committee feels that more stringent government action is the only practical solution. There is a need to sensitize legislators and administrators in this regard and to issue strict orders for prompt reporting of all deaths. Based on the available data, there were 552,740 births and 154 deaths in 2004 and 5,748,57 births and 153 deaths in 2005. Thus Kerala's MMR works out to be 28 per 100,000 in 2004 and 27 per 100,000 in 2005. Even if we allow for an equal number of unreported cases, the MMR for the State will be only just above 50 per 1,00,000.

- Under reporting would have occurred.
- More stringent government orders are required for prompt reporting of all deaths.
- Even if an equal number of maternal deaths went unreported, the MMR will be only just above 50 per 1,00,000.

The main purpose of the CRMD was to identify why mothers die. Only when all the circumstances of death are known is a realistic analysis of remedial factors possible. 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 frequently incomplete. Often the notes from the hospital where the death oc-



curred are 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. However one has to accept that this problem exists even in developed countries where autopsy in every case of maternal death is not mandatory. Moreover, it may not be important in cases where there are enough clinical features to arrive at a diagnosis. However, where the clinician is unsure of the cause of death, a provision for autopsy should be made available. We are aware that there are many logistical problems with implementing such a policy of compulsory autopsy for every case of maternal death. In addition, even in cases where an autopsy has been conducted because of medico-legal reasons, there is no mechanism to make the findings available to the CRMD committee. This could be easily remedied if the Government issued an appropriate order.

- 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 first started
- 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.

## Haemorrhage

The most common direct cause of death still remains PPH. There were 29 deaths in 2004 and 32 in 2005 due to obstetric hemorrhage working out a percentage of 19.8. The major contributor was atonic PPH. (This is contrary to the findings of similar confidential inquiries in the United Kingdom where hemorrhage has declined in significance and ranks after thromboembolism as a cause of maternal death.) Both APH and PPH contributed to maternal deaths but PPH was the primary cause in the majority of cases. In some cases assigned to PPH, it was not possible to ascertain if trauma - like deep vaginal and cervical lacerations or even rupture of the uterus - was present.

The speed with which haemorrhage killed the mother was astonishing in some

cases. The usually quoted one or two hours, the so-called “golden hours” - were not available in some cases. This raises the suspicion that there were factors other than atonicity that caused the bleeding. Factors like unrecognized trauma or associated coagulopathy, rather than atonicity of the uterus, come to mind.

### **First-aid essential before transfer 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. Reorientation and further training of medical officers, labour room nurses and other staff seem to be urgently required to ensure that adequate first aid is given before transferring these patients.

### **Steps to arrest bleeding**

Once abnormal haemorrhage is identified, quick and definitive steps are essential to arrest the bleeding. Such steps include tamponade using packs, condom or balloon, suturing lacerations and employing various sutures for stepwise devascularisation of the uterus.

A variety of such suturing techniques are available from which the obstetrician can choose (also, known as the *cafeteria approach*). There is an urgent need for practicing obstetricians to update their skills in this regard. Regional workshops seem to be the only practical way to achieve this.

Sudden onset of coagulopathy is possible if there is amniotic fluid embolism or if the patient bleeds profusely and goes into shock. If there are extensive lacerations in a friable vagina or cervix or lower segment, such massive blood loss is possible. Once the patient goes into shock the uterus becomes atonic and the vicious cycle is perpetuated. One suspicion raised (but not substantiated by properly conducted studies) is that overuse of prostaglandins might be a contributing factor. The committee feels that starting cervical ripening with mechanical methods like foley's catheter and extra amniotic saline and following it up with prostaglandins (E1 or E2) will help to reduce the dose of prostaglandins and thereby the risk of lacerations. The dosage and interval of application of prostaglandins should be as per standard recommendations. If prostaglandin E1 is used, 25 microgrammes in the posterior fornix of the vagina at 4-to-6-hour intervals should be the standard regime.

The committee for CRMD is concerned about another practice for the acceleration of cervical dilatation that has become established in certain centres. Drugs like drotaverin, valethamide and hyoscine are being used routinely until delivery. There is no justification for their use in cases where the cervix is already soft,

effaced and 3cm or more dilated. The committee feels that these agents which are smooth muscle relaxants could cause vasodilatation and thus increase the bleeding and potential for amniotic fluid embolism. We believe that such practices should be stopped until scientific studies can establish their advisability.

Every centre where delivery occurs should have a standard protocol to deal with PPH. Ensuring that an emergency trolley with necessary supplies is available in each labour room is perhaps the first step. Steps should also be taken to arrest the bleeding which may include tamponade using balloon or packing and to prevent hypovolemia with ample IV fluids. These procedures do not seem to have been incorporated into the usual practice.

### **Ambulance service**

Even transporting patients needing emergency transfer to a higher centre was at times a problem. Often the issue was either the lack of a free ambulance service or the inability of the patient's family to pay for an ambulance. This happened more in the remote and hilly areas of the State. Even within big cities, rush hour traffic may delay transfer from one centre to the other, especially if it is not in an ambulance. There is an urgent need to streamline the patient transport system in the State. It is worth considering whether voluntary agencies can be roped in to help with this issue.

- Haemorrhage is the most common cause of maternal death.
- Patients seem to collapse very quickly once haemorrhage starts, probably due to the massive nature of bleeding.
- First aid to arrest bleeding like packing or tamponade is essential, especially before referring to higher centres.
- Ample fluids should be given to prevent hypovolemia.
- Use of prostaglandins for ripening and induction should be standardised.
- Mechanical methods of ripening should precede use of prostaglandins.
- Routine use of smooth muscle relaxants like drotaverin and valethamide, even after cervix is effaced, should be stopped.
- All obstetricians should be trained in stepwise devascularisation of uterus.

### **Hypertensive Disorders**

The second most common cause of maternal death was hypertension or its associated complications. Severe pre-eclampsia, eclampsia, and HELLP syndrome are included in this category. Altogether 41 out of 307 (13.35%) deaths were due to

hypertensive disorders in 2004 and 2005. Certain common features emerged upon review of these cases.

### **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. Unfortunately, it is not used widely in Kerala as is evident from our review. Several reasons have been quoted for this. The non-availability of the drug is the most commonly cited reason but this issue can be easily and quickly remedied if the government takes the initiative. Magnesium sulphate is a cheap drug, is stable at room temperature, and has a long shelf-life. The government's immediate attention is required to solve this issue. Another reason provided is the reluctance of non-obstetrician doctors in the treating team to use  $\text{MgSO}_4$ . Neurologists and anesthesiologists are often quoted as preferring other drugs like Phenytoin Sodium. It is imperative to convince these colleagues of the need for primarily using  $\text{MgSO}_4$ .

Even in cases where  $\text{MgSO}_4$  is used, the committee found that the dose used was inadequate or the drug was withdrawn too early. The committee came across two cases where the loading dose was not followed up with adequate subsequent doses and the patients succumbed to recurrence of fits. In the Indian medical literature, there have been many papers recommending a reduced dose of  $\text{MgSO}_4$ . In the light of the above experience, a reduction in the dose of  $\text{MgSO}_4$  should not be attempted until properly conducted studies confirm the wisdom of this approach.

### **Parenteral antihypertensives -essential**

A significant number of deaths in hypertensive disorder were due to cerebrovascular accidents. Often these problems developed after the patient had reached the hospital, which means that an opportunity for preventing this catastrophe was missed. However, most hospitals lack parenteral antihypertensives, and even after IV labetalol became available, the high cost hinders its widespread use. Even though nifedipine has been criticized for causing an acute drop in BP and myocardial ischaemia when used sublingually, it is possible to adjust the dose to avoid a sudden and drastic fall in BP and any associated complications. Our recommendation is to use it only when parenteral agents are not available and not to use more than 5mg of nifedipine at a time. Oral nifedipine has to be started along with the sublingual and further doses should be oral.

### **Timing of termination of pregnancy**

Mode and time of termination of pregnancy is the third aspect in the 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 the cervix. If conditions are favourable for vaginal delivery within a few hours, this option can be pursued. Otherwise caesarean delivery has to be done.

When severe hypertensive disorder sets in before the fetus has reached salvageable age, the obstetrician is often tempted to continue conservative management. Several factors should be considered before deciding on such a course and each case has to be considered on an individual basis. Broadly speaking, development of severe pre-eclampsia, oligamnios or growth restriction or Doppler abnormalities before 28 weeks carries very poor prognosis.

- $\text{MgSO}_4$  should be promoted as the first-line anticonvulsant in eclampsia and severe pre-eclampsia.
- Government should take steps to supply it to all government medical institutions.
- Reducing the dose of  $\text{MgSO}_4$  from the one recommended in standard books should be initiated only after properly conducted studies.
- Parenteral antihypertensives like labetalol and hydralazine should be made available and affordable in the country.
- Once eclampsia or severe pre-eclampsia have set in, further continuation of pregnancy should be allowed only after ensuring maternal safety.

### **Amniotic Fluid Embolism (AFE)**

The third most common cause of maternal death was amniotic fluid embolism. There were 30 cases of AFE in the two years. Unfortunately, this is a diagnosis made only on clinical grounds with very little autopsy evidence to support it. However, the review committee was careful to assign this as the cause of death only after excluding other possibilities and only when the clinical picture was rather typical. The typical clinical features were sudden onset of chest discomfort, a brief spell of convulsions, cyanosis, coagulation failure, and severe PPH. If the fetus was still in utero, fetal distress or death ensued. In the majority of cases, there was excess uterine activity preceding the onset of symptoms.

The large number of cases falling under this category is a matter of great concern as this was not a major cause of maternal death in the experience of the committee members prior to the nineties. The committee considered whether this increased incidence could be related to any recent change in obstetric practice. The possible changes pointed out were:

1. The increasing use of prostaglandins for induction of labour.
2. The practice of using smooth muscle relaxant agents like drotaverine and valethamide to hasten cervical dilatation.

The association between hyperstimulation and AFE was disputed earlier but is now accepted on the basis of clinical observations. The theoretical objection raised earlier was that a hypertonic myometrium will keep the vascular channels closed thus preventing the passage of amniotic fluid into the circulation. However, the association between prostaglandins and amniotic fluid embolism can be explained in another way. When prostaglandins are used, it causes softening and vasodilatation in the upper vagina, cervix, and lower segment, making these tissues vulnerable to lacerations. It is conceivable that under these circumstances, amniotic fluid gets into the circulation more easily and causes embolism.

The second aspect was the routine and irrational use of smooth muscle relaxants with the belief that it hastens dilatation of the cervix. In some of the cases we analysed, the instruction given to nursing staff was to routinely administer such drugs parenterally on half-hourly or hourly intervals till delivery. It is difficult to ascertain whether this has contributed to the increased incidence of amniotic fluid embolisms but at least it is not a practice that has any scientific support and hence is not followed by the committee members. We are constrained to recommend that such use of vasodilators on a routine basis should be stopped until a clear benefit is demonstrated on the basis of scientific studies. If at all used it should be restricted to cases where there is resistance to cervical dilatation in spite of oxytocic agents.

Prostaglandins (PGE1 & PGE2) cause cervical softening and friability, and higher doses lead to hyperstimulation. Hence, the dose used should be limited to the minimum effective one. Ordinarily PGE1 should not be used at a dose of more than 25 microgrammes every 4 hours. Once the cervix has become fully effaced prostaglandins should not be used. Instead, oxytocin as a drip should be used if further stimulation of the uterus is required.

Management of AFE is essentially that of resuscitation of an acutely collapsed patient. Therein lies the importance of always being prepared for this eventuality in the labour room. Amniotic fluid embolism can occur in any labour case without warning. A well-maintained emergency trolley, and staff who have updated their resuscitation skills are essential to save lives. Incidentally, it is worth remembering that acute collapse can occur in many other clinical scenarios in the labour room. The availability of resuscitation equipment and skilled staff will be the crucial factors that decide the outcome.

In cases of amniotic fluid embolism where the patient survives the initial collapse, secondary effects of the pathology, like coagulation failure, may develop. This needs expeditious management. Replacement of coagulation factors, restoration of circulating volume, and maintenance of cardiorespiratory functions, are the subsequent steps required in such a scenario.

The obstetrician may be called upon to take quick and decisive steps. For example, if the patient is undelivered, the need for an immediate caesarean section



has to be addressed. Similarly, if there is severe PPH, the question of a hysterectomy or other measures to arrest the haemorrhage needs to be considered. These decisions must be made based on the merits of each case.

- Amniotic fluid embolism has emerged as a major cause of maternal death.
- Possible contribution from widespread use of prostaglandins and smooth muscle relaxants like drotaverin / valethamide is suspected.
- Prompt resuscitation measures are essential to save the life after AFE; every obstetrician should be trained in emergency resuscitation.
- Every labour room should have a well-maintained emergency trolley.

## Heart Disease

The fourth most common cause of mortality in the series was heart disease – 29 out of 307 (9%). Unfortunately, many of these cases were not available for detailed analysis by our cardiologists. Valvular disease was the most common. We assume that the majority of them were sequelae to rheumatic fever. The poor outcomes in some of these cases could also be the result of poverty and backwardness, pregnancy being only an associated factor. The case of a young primigravida diagnosed to have mitral stenosis in early pregnancy is a typical example. She was advised to have surgical correction in early pregnancy but could not afford it. When she finally developed acute pulmonary edema in early labour, it was too late for any surgical correction.

The committee members and our cardiology colleagues do feel that valvular heart disease of rheumatic etiology is on the decline. However, we had 17 cases reported in 2005, the majority of which were thought to be due to acquired valvular disease.

Our cardiology colleagues have made some recommendations which are contrary to conventional thinking. Earlier teaching held that vaginal delivery after spontaneous onset of labour would provide the best outcome when there is heart disease complicating pregnancy. Our cardiology colleagues have suggested that, 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.

- Correctable valvular diseases should be treated before pregnancy.
- In cases known to deteriorate as term approaches, the option of elective cesarean delivery once the fetus becomes salvageable may be recommended.

## Venous Thromboembolism

In 18 out of 307 cases, the cause of death was assessed to be venous thromboembolism. Two main types of problems are classified under this category: one is venous thrombosis which may get dislodged and cause pulmonary embolism and quite often, instant death; the other is cerebral venous thrombosis which causes cerebral malfunction and death.

Venous thromboembolism ranks as the number one cause of maternal death in the UK. In the present series, out of the 18 cases, 13 occurred in 2004 and only 5 in 2005. Out of the 10 deaths reported to CRMD for analysis, 7 followed cesarean delivery.

In the present series seven out of ten were allotted to pulmonary embolism (all but one following caesarean). There were two other cases where final cause of death was pulmonary embolism but are allotted to hypertensive disorder because that was the primary cause. Three of the ten were assigned to cerebral venous thrombosis. With the increasing incidence of fatal pulmonary embolism, comes the question of thromboprophylaxis. However we could not identify specific risk factors except operative delivery (cesarean section). Even here it is hard to recommend thromboprophylaxis. The overall incidence of venous thromboembolism is much less in our society, so we cannot copy wholesale the recommendations of the Royal College of Obstetricians and Gynaecologists. Due to the absence of any hard evidence related to our population, we have been forced to make some recommendations on the basis of the experience of the members of the editorial board. It is emphasized that these are empirical suggestions.

1. Patients after vaginal delivery as well as caesarean section should be encouraged to ambulate at the earliest. This may mean encouraging patients after vaginal delivery to ambulate and go to toilet within 24 hours of delivery. After a caesarean section, patients should be encouraged to move their limbs and turn in bed on the day of surgery itself and to move out of bed by the next day.
2. Adequate fluid intake should be encouraged. This is specially mentioned because there seems to be a belief in the minds of many people that drinking fluids in the postpartum period will delay involutional changes and lead to bulging of the abdominal wall.
3. Low molecular weight heparin can be advised antenatally if the patient gives a history of recurrent thrombosis during or prior to present pregnancy or if there is a history of thrombophilia. They need postpartum thromboprophylaxis as well.
4. Postpartum thromboprophylaxis with low molecular weight heparin may be considered if any three of the following conditions exist.
  - a. BMI of more than 30



- b. Above the age of 35
- c. parity >4
- d. Hypertension
- e. On complete bed rest prior to delivery for at least 4 days
- f. Those who had triplets or higher order multiple pregnancy
- g. Extensive varicose veins
- h. Caesarean delivery
- i Sickle cell anemia
- j Severe blood loss or severe infection

Low molecular weight heparin at a dose of 5000 units subcutaneously once daily is recommended for thromboprophylaxis. This is to be administered once the patient is stable after delivery or within one hour, whichever is earlier. The thromboprophylaxis may be stopped once the patient is fully ambulant or after 5 days, whichever is later, except in high risk cases where it may be continued for a longer period.

5. Elastic compression stockings are known to reduce the risk of venous thromboembolism. This may be used by itself especially in patients with large varicose veins and also as supplement to the use of heparin.

- Fatal pulmonary embolism is on the increase.
- Thromboprophylaxis should be considered , especially after caesarean
- Adequate hydration and early ambulation should be encouraged.

### Anaesthetic causes

It was gratifying to note that after the 4 deaths assigned to anaesthetic causes in the first year, there were none in the following year. However continued vigilance is required from our anaesthesia colleagues while giving anaesthesia or analgesia for the obstetric patient because of the special risks in using regional or general anaesthesia for these patients. Whenever possible regional anaesthesia should be preferred over general anaesthesia for caesarean section.

- When there is no contraindication for regional anaesthesia, it should be preferred over general anaesthesia

### Liver Disease in Pregnancy

There were 17 deaths assigned to this cause. These do not include hepatic dysfunction that may result from severe preeclampsia as in HELLP syndrome. In cases of severe hepatic dysfunction, sometimes it is not possible to distinguish between etiological factors like viral hepatitis or acute fatty liver of pregnancy. While it is generally agreed that immediate termination of pregnancy is indicated

in acute fatty liver, the recommendation in cases of viral hepatitis is to leave the pregnancy alone rather than induce. What we have observed is that when hepatic dysfunction occurs in late pregnancy, e.g. viral hepatitis after 34 wks, often the patient goes into labour at the peak of this dysfunction and dies. In acute yellow atrophy - acute fatty liver of pregnancy - unless the pregnancy is terminated, the condition of the patient will continue to deteriorate. It is on these grounds that the editorial board suggests taking steps to deliver the fetus at the first sign of hepatic dysfunction occurring beyond 34 wks of pregnancy even if it is viral hepatitis. It is expected that if such a course is followed, the possibility of the patient going into labour at the peak of liver dysfunction and then bleeding to death can be reduced. While we have some anecdotal experience of good outcomes following such a policy, the editorial board admits that there is no hard evidence to make such a recommendation.

- Consider termination of pregnancy at the first sign of hepatic dysfunction beyond 34 wks. Otherwise the patient may go into labour at the peak of hepatic dysfunction. Please note that this recommendation is on the basis of clinical observation and not randomized trials.

## Miscellaneous causes

There were 107 deaths that were not assigned to categories mentioned earlier.

## Infection

In the case of 26 patients, infection of an organ system was assigned as the cause. For seven cases other organ systems were involved but in 19 cases the major involvement was of the genital tract. Only 10 cases were reported to the CRMD. It is obvious that infection still contributes in a substantial way to maternal deaths though not in the form of septic abortion.

## Anaemia

For eleven deaths, the cause was anaemia – four of them were reviewed by the CRMD committee. We were careful in assigning anaemia as the cause only when there was evidence that anaemia was the primary cause. For example, aplastic anemia non-responsive to intensive therapy was one such case. In the cases assigned to anaemia by the DHS, all the details to exclude other causes were not available.

## Ectopic pregnancies

There were six cases assigned to the category of ectopic. These were either due to late diagnosis with extensive intraperitoneal bleed or missed diagnosis. This is in contrast to the way ectopic pregnancies topped the list of direct causes of death in the UK a couple of trienniums ago. In the UK, the scenario was that of women

often living alone dying at home due to internal haemorrhage without coming to the attention of health care workers or relatives. Hopefully in the Kerala scenario, with more widespread use of ultrasound scanning, deaths from ectopic pregnancies will lessen in the future.

## **Suicide**

The six cases assigned to suicide raise concern. It is true that suicide is a major contributor to maternal deaths in a developed country like the UK. The social and family structures are totally different there compared with Kerala. Yet the problem of suicide and its significant contribution in the Kerala scenario worry us. It appears that the erosion of family support as Keralites take to the nuclear family pattern might be contributing to the suicide rate among expectant mothers. Only two of the cases came to the attention of the CRMD committee.

## **Accidents**

The three deaths that resulted from accidents reflect the rapidly increasing number of automobile accidents in the State. In these cases the deceased was not driving the vehicle and use of a seat belt would not have made a difference. However, advising pregnant women not to travel in the front seat and, if they do, insisting on their wearing a seat belt suitably modified may prove beneficial in the future.

The remaining deaths belonged to various causes with low incidence

## **Unknown**

In 31 of the deaths, no cause could be assigned. Only four of them were reported to the CRMD committee. The committee put them under the “unknown” category because the case notes or other details were not available or the patient was brought dead without any accompanying clinical details. Another 25 were from the list supplied by the Director of Health Services who did not get the necessary information from the field. The remaining two were brought dead.

We cannot help but comment on some other observations made while doing the confidential review.

## **Obstetricians eager to improve their performance**

Obstetricians in charge of the deceased were eager to get feedback about the case and wanted to improve on subsequent patient management. The committee could not fulfill this desire on an individual case-by-case basis. Several factors were responsible for this but the main one was our inability to suggest an alternative line of management that would have definitely made a different outcome. Another reason was that by the time CRMD analyzed the case, several months would have elapsed and interest in the particular case would have waned. We hope that pub-

lication of the findings in this book in an anonymous way will help obstetricians learn lessons without hurting anyone's ego.

## **Violence against hospitals and staff should stop**

Every maternal death is a tragedy of multiple dimensions. It affects not just the immediate family. The caregivers especially the concerned obstetrician is badly shaken. While this is a universal truth, unfortunately in Kerala there is another practice that is widespread and causes great concern to us. This is the often violent reaction from the patient's relatives and friends towards the caregivers and institutions that looked after the deceased. Peripheral obstetricians face the brunt of this fury, often whipped up by some interested local groups. Usually this reaction is based on accusations of neglect or mismanagement by the caregivers. As a result the peripheral obstetricians are frightened and often refer the patient to higher centres even without trying the remedial measures that they are capable of. The observations made earlier regarding PPH patients being referred without trying a pack or tamponade is probably due to the fear complex generated in these circumstances.

Another related development is the change in health centres run by the government. Many of the primary health centres and community health centres where deliveries used to take place have stopped conducting deliveries.

A change in the above-mentioned public reaction is essential. If there is suspicion of mismanagement or neglect on the part of caregivers, there are enough avenues available in our system for redressal of the grievance and instant violence against care givers and hospitals should stop.

## **Patient Transportation**

Transportation of critically ill patients to higher centres is still a problem due to the absence of an organized ambulance system. Often such patients are transported in private taxis or auto-rickshaws. In many cases the cost involved may be too high for the patient's family and valuable time is lost either in collecting sufficient money or organizing a vehicle. If an organized ambulance service was available, these problems would be solved. It should be possible to enroll the help of non-governmental organizations like the Rotary or Lions Clubs. They already have an organized system of picking up accident victims. It should be much easier to organize a transport system for maternity cases. The urgent attention of the government is required in this regard to facilitate a proper ambulance service, free for maternity cases.

## **Special Support to the Tribals and the Poor**

Analysis of epidemiological data proves that a disproportionately high number of mothers who died belonged to the low socioeconomic group or tribal community

and had a low educational status. This is despite the fact that there are many arrangements made by the government to give them physical and financial assistance for treatment. These point to the need for educating the tribals and their support group to encourage effective utilization of such resources.

### **Accreditation of hospitals**

Kerala can be proud of its 98% institutional deliveries, a figure that exists in only very few areas in the world. Our people have shown their trust in institutional deliveries. It is also observed that 60 to 70% deliveries take place in the private sector. But, are the centres where deliveries take place equipped to handle normal and abnormal deliveries and possible complications round the clock? Many centres in the public as well as in the private sector lack in adequate manpower and equipments. A mechanism has to be established to ensure that minimum standards are maintained in centres where obstetric care is provided.

### **Upgrading staff knowledge**

Concepts about management of various obstetric situations that can lead to morbidity and mortality are rapidly changing. Unless the knowledge and skill of the entire staff in the labour room is upgraded, optimum results cannot be achieved. There is no compulsion or incentive to do this at present. It is mandatory that nurses and midwives are included in such programmes to upgrade their knowledge. There is an urgent need to look into these aspects before the public's trust in modern medical institutions erodes and they resort to other systems or even home deliveries. Again, we feel that this initiative has to come from the government.

### **Confidential Review – The way forward**

The CRMD committee while analyzing the circumstances of deaths, tried to see if the death was avoidable and what lessons could be learned for the future. It was found that in many cases an alternative approach would have helped to avoid the tragedy. However it would be futile or rather counterproductive to bring up such a discussion of individual cases. It would erode the confidentiality of the whole system and result in a lack of cooperation in the future. Instead we have projected typical case scenarios and suggested alternative management strategies. We think that this is the most civilized and most productive way to bring about a change and achieve the goal of maternal mortality reduction.

## *Chapter Five*

# Key Recommendations

The Confidential Review of Maternal Deaths (CRMD) brought to light the fact that there was room for improvement in the management of many of the cases of maternal deaths 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 postpartum collapse due to haemorrhage, or a collapse due to amniotic fluid or pulmonary embolism. A trolley with the required drugs, fluids, IV sets, cannula, “cut down sets,” endotracheal tubes, laryngoscopes, etc., should be readily available in every labour room. The obstetrician in charge should appoint a person (e.g. the head nurse) in the labour room to be responsible for ensuring that all items in the emergency trolley are readily available and in working order and that the drugs are within the expiry date.
2. Provide regular training and updating to obstetricians and labour room nurses. This should include training for setting up intravenous lines, intubation, and external cardiac massage. Those in service and in training should undergo this updating.
3. Establish an alarm system in labour rooms to call for help, especially in those labour rooms that have large, separate enclosures for the second stage of labour.
4. Make a concerted effort to ensure availability of blood and components to all centres where deliveries take place. This does not mean setting up full-fledged blood banks in all centres. In centres where blood and components are not available, 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.
5. Smaller centres should establish tie-ups with higher centres for accepting obstetric emergencies. If a complication needing the help of a more experienced obstetrician or anesthetist develops, it should be possible to arrange such help without delay. Whether the patient should be transported to the higher centre, or experienced personnel from such centres go to the

periphery, has to be decided on the nature of each situation but transport should always be readily available either way.

6. Develop a network of ambulance services throughout the State. Non-governmental organizations can be recruited to help in this. This facility should be free or heavily subsidized so that the service is not denied to anybody for financial reasons. The ambulance service should be available not only for transporting patients but also to bring blood and components and to transport expert teams in busy cities.
7. The government should take the lead to ensure that life-saving drugs are available in all centres. This includes magnesium sulphate and antihypertensives like labetalol and intravenous hydralazine.
8. 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 Officer of Health or a voluntary agency working with the Government's approval. 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. The Kerala Federation of Obstetrics and Gynaecology (KFOG) and the Kerala chapter of the National Neonatology Forum can be requested to help with this certification system.
9. Establish obstetric intensive care facilities especially in major centres and medical colleges. There should be ambulances with transport ventilators attached to such centres.
10. Increase the public's awareness of the need for antenatal care and early reporting to hospitals if complications develop. Conducting regular antenatal classes will help in conveying valuable information to patients and their families. Even though such classes are already conducted at present in some centres, there is a need for more to be started in others so that they are available to all antenatal women and their families.
11. Improve reporting of maternal deaths by making it mandatory. Make it the responsibility of the hospital administration who should see that the data are collected from the obstetrician gynaecologist and forwarded to the state coordinator.
12. 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 note should be given to the higher centre, even if the doctor accompanies the patient.

**Why Mothers Die**  
**Kerala - 2004-05**

Part-Three

**CAUSES AND  
SOLUTIONS**

3





## *Chapter 6*

# **Obstetric Haemorrhage**

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**On behalf of the Editorial Board**

### **Key Summary Points**

- Obstetric haemorrhage is the leading cause of maternal deaths in the years under review.
- Of the various types of haemorrhage, atonic PPH is the most significant.
- From the notes available, it is not possible to ascertain whether active management of 3<sup>rd</sup> stage is universally followed.
- It appears that in many centres, the prophylaxis against PPH is only increasing the drip rate of oxytocics sometimes with additional units added to the drip. A step to obtain sustained uterine contractions like administration of ergometrine or PGF<sub>2</sub> alpha is desirable.
- Close observation of postpartum patient for atleast 2 hours to pick up postpartum haemorrhage should be mandatory. It is desirable that the patient is shifted to her own room only after this period.
- The nurse observing the postpartum patient should palpate and ascertain whether the uterus is contracted and whether there is excessive vaginal bleeding rather than just record the pulse and BP.
- A systematic approach seems to be missing after PPH is recognized. Practicing PPH drill should be popularized.
- When patients are referred to higher centre, adequate steps to reduce bleeding until she reaches the higher centre do not seem to be taken.
- Transport facilities for transferring these patients to higher centre have to be stream lined.

### Obstetric Haemorrhage: Key Recommendations.

1. Management protocol for obstetric haemorrhage should be developed and made available in all institutions where deliveries are conducted. All members of the staff including nurses, nursing assistants and staff working in the blood bank and laboratory should rehearse the protocol like a "Fire Drill" or "PPH Drill" and should be aware of their role.
2. Women with known risk of obstetric haemorrhage should be delivered in a centre with facilities for blood transfusion, laboratory work up and surgical procedures
3. Protocol with involvement of a Physician and Anesthesiologist is crucial in the management of massive obstetric haemorrhage
4. Active management of third stage of labour must be a routine.
5. Placenta praevia accreta, especially with previous scar, should be managed in a tertiary centre by a senior consultant.
6. Initial management of PPH includes early recognition followed by prompt attention to the resuscitation and simultaneous search for the cause of the bleeding.
7. Uterine packing and the condom tamponade should be a management option before performing surgical procedures.
8. Obstetrician in training as well as those in service should be well versed with surgical steps to arrest bleeding in PPH.
9. Decision for surgical management, especially for hysterectomy should be taken at the appropriate time, and not as a last resort.
10. All labour rooms should have cervical inspection sets and a well equipped trolley with emergency drugs.
11. Clinical Audit programme should be practised in each centre.
12. All patients after delivery should be under close observation for about 2 hours during which vital signs and whether uterus is contracted or not should be recorded.
13. Since PPH is not predictable in every case, having an IV line with cannula will be a prudent step in every labour

### Summary of findings:

Of the 307 deaths reported during the years 2004 & 2005, sixty one (about 20%) were due to haemorrhage. Of the sixty one deaths 36 were due to post partum haemorrhage, 5 due to placenta praevia and 10 due to abruptio placentae. Break up of the ten deaths reported by DHS in 2005 is not known.

Since this is the first report of CRMD, we have no previous data to compare with and know the trend. Hence, institutional data, even though not representative of the whole State, is used for comparison.

Institutional statistics show that obstetric haemorrhage was the leading cause of maternal deaths, 31% for the period 1978 to 82 and 19% during 1993-97.

**Table 6.1 Maternal mortality due to obstetric haemorrhage**

Period	1978 -82*	1987-91*	1993-97*	Pilot study 2001**	2004CRMD +DHS***	2005CRMD + DHS***
No.of maternal deaths	237	121	81	105	154	153
No.of deliveries	35,267	76,276	94,200		552740	574857
Haemorrhage	75 (31%)	27(22%)	16(19%)	23(22%)	29(20%)	32(21%)
MMR	672/100,000	159/100,000	85/100,000			

\*Statistics from Calicut Medical College., \*\* Study conducted by Maternal Fetal medicine committee of KFOG. \*\*\* Figures from CRMD and DHS.

There has been a satisfactory reduction in the maternal mortality rate in the Northern part of Kerala as evidenced from the statistics of Calicut Medical College. However the incidence of death due to haemorrhage has continued to top the list, for the whole state.

**Table 6.2 Death due to haemorrhage ten years back – Govt. Medical Colleges**

Period	No. of deliveries	No .of deaths	Due to Hge	Percentage
1993-97	234690	310	53	17.09%

Analysis of maternal deaths in the teaching hospitals of Kerala for the five year period from 1993 to 1997 has shown a maternal mortality rate of 135/100,000 and obstetric haemorrhage was contributing to 17% of maternal deaths<sup>(1)</sup>.

Of the 61 deaths due to haemorrhage, we could reliably categorise only the 38 deaths reported to the CRMD, 26 of them were due to PPH, 8 were due to abruptio placenta and four due to placenta previa. Of the 26 belonging to PPH, 16 were of atonic type and 10 were of traumatic type.

Table 6.3 Deaths due to haemorrhage (CRMD)

Atonic postpartum haemorrhage	16
Traumatic postpartum haemorrhage	10
Placenta Praevia	4
Abruptio Placentae	8
Total	38

## Postpartum Haemorrhage (PPH)

### Atonic

The average parity and age of the patients with atonic pph were not significantly different compared with the age and parity of the entire deceased mothers. On almost all occasions the babies were saved.

All except one of the atonic PPH cases had vaginal delivery. All of them except two had spontaneous onset or leaking of liquor. From the case notes it is difficult to ascertain how many had an actively managed 3<sup>rd</sup> stage. However, once PPH had set in, liberal use of methergin, oxytocin and prostaglandin F2 alpha and E1 is seen. One wonders whether the opportunity of prevention was missed and treatment was started too late.

Ten of the 16 patients were referred to higher centres for further care. One of them reached there dead and another was gasping on arrival. A third was moribund. It is difficult to find out what sort of first aid was used to control the bleeding during transportation. Four of these patients had hysterectomy and one had a relaparotomy because of ongoing intraperitoneal bleeding.

Four patients had gone in for Disseminated Intravascular Coagulation (DIC) which made further management really difficult. The rapidity with which some of the patients deteriorated and ended in multiorgan dysfunction syndrome is quite alarming. This indicates the need for prompt action in controlling the bleeding and resuscitation, so that the patient does not slip into shock and its consequences.

## Learning from example

### Example 1

This 35yr old, G5 P4 was admitted with mild labour pains 10 days prior to her EDD. Blood group A negative, Hb 9.2gms. Progressed spontaneously and had vaginal delivery at 00.10hrs of an unasphyxiated baby weighing 3 kg. Placenta and membranes delivered, methergin given. About 20 mts later there was profuse bleeding. PGF<sub>2</sub> alfa, dexona, efcorlin etc were given. IV line started, 2 units of blood was given. She rapidly deteriorated and was declared dead by 1.50am.

Going through the case notes it is found that the patient received an injection of valethamate at 6cm dilatation (10.30pm)

This case brings up many practical points. The IV line was started at delivery - was it there when the bleeding started? Profuse bleeding was noted 20mts after the delivery of the fetus - there is no mention of steps taken to rule out traumatic PPH (cervical inspection) nor of any attempt to arrest the bleeding, other than oxytocic administration (eg:condom tamponade or packing). Was that valethamate indicated when she was already 6cm dilated and progressing smoothly? On the contrary, at least theoretically a vasodilator and smooth muscle relaxant like valethamate could have contributed to further bleeding.

The rapidity with which the patient deteriorated and died (just about 1 hour and 30mts)is alarming. This emphasizes the need for every one to approach PPH as a drill - PPH drill.

### Example 2

This 34 yr old G2P1 and LCB 10yrs previously was admitted 4 days prior to her EDD for safe confinement. BP130/90. On p/v cervix thick, 1 finger. 50 microgramme misoprostol was inserted into posterior fornix. Leaking per vagina started at 2.30am. Oxytocin drip started. 3am vertex at introitus. 3.17am vacuum applied and delivered baby with minimal shoulder dystocia. Baby was asphyxiated, weight 4.2 kg. Methergin 1 amp IV given.

At 3.45 am patient complained of giddiness, BP 80/70, uterus contracted well. On P/V, plenty of clots and blood in the vagina which was removed and pack given. Inj.dexona, prostodin, haemaccel, normal saline etc given. Pulse was raised. Ordered for blood but bystanders were not available. Since patient's condition was deteriorating, decided to send to a higher centre. She was sent in an ambulance accompanied by the doctor and nurse. She was gasping on reaching the casualty of the higher centre and succumbed.

The reporting doctor has put amniotic fluid embolism as the cause of death. On being asked "if you were treating this case again, what changes would you make that will help to avoid maternal death?" the response was "Elective caesarean section for a big baby".

This case reflects the difficulties and dilemmas doctors at the periphery face. It is easy to criticize obstetricians at the periphery with "wisdom of hind sight" and blame them for the maternal death. If we do so, we will be forgetting that a major chunk of the obstetric service is provided by such small centres who work round the clock, without facilities of blood banks or enough man power. We do appreciate their services. However, a few deviations from the standard practices cannot be ignored.

1. Perhaps the size of the baby (4.2Kg) was not appreciated while deciding on induction. Realisation of this and the experience of shoulder dystocia may be the reasons for suggesting elective caesarean on a subsequent occasion. But looking at the time frame (2.30am 3/5 dilated, pitocin started, 3am fully dilated, vx at introitus, 3.17am vacuum applied and delivered) one cannot help wondering whether there was a hurry burry in the management . Was the shoulder dystocia also iatrogenic?
2. Within 28 minutes of delivery patient complained of giddiness and then progressively deteriorated. Vagina was packed. Uterus was contracted. So the possibility of cervical and vaginal lacerations should have been looked for. Obviously the packing was not effective. A good cervical inspection set would have helped in proper visualization of the cervix and upper vagina.
3. Was it necessary to put prostaglandin 50 microgramme at 6.30 pm? We feel that such elective induction should be planned for the morning. Also for an unfavourable cervix, mechanical ripening using foley catheter and extra amniotic saline prior to use of misoprostol would certainly reduce the complications. As happened in this case, if PPH develops in the early hours of the morning, there will be many problems in arranging blood and other resuscitative measures.
4. The provisional diagnosis put by the peripheral doctor was amniotic fluid embolism. There is very little supportive evidence for that diagnosis. Even as a case of PPH whether it was atonic or traumatic is difficult to ascertain.

### Example 3

This 30yr old G4 with previous 3 normal deliveries had her regular antenatal care from a peripheral hospital. At 30 weeks and 33 wks she had APH. Ultrasound scan had confirmed placenta previa partially covering the os. She was again admitted at 35 wks with bleeding p/v. It was also managed conservatively with plans for elective cs at 36 wks. However she got into labour at 35 wks itself and had the baby spontaneously delivered at 7.05 pm. Third stage was uneventful. Prophylactic PGF2 and oxytocin were given. Fresh bleeding was noted at 8.10pm followed by severe atonic PPH not responding to oxytocics, bimanual compression and blood replacement. She was taken for hysterectomy at 9.30 pm under general anesthesia. Total hysterectomy was done. Diffuse bleeding noted at the time of closure. She developed acute hypotension and tachycardia. She received 6 units of fresh blood and other fluids. She was shifted to a higher centre with endotracheal tube and ambu bag ventilation and accompanied by the anesthetist.

She reached the higher centre at 3.35am . She was extremely pale with heart rate 134/mt, with free fluid in the peritoneal cavity, only 50ml blood

stained urine in urobag. As the condition did not improve with blood, plasma and platelets, relaparotomy was done. Peritoneal cavity was filled with fluid blood. Hysterectomy stumps were inspected. Generalised ooze was noted especially from bladder base. After surgery general condition and urine output improved but the DIC continued and she expired, 22 hours after admission to the higher centre.

We cannot blame the peripheral hospital or the higher centre for any laxity in the management. The peripheral centre decided on hysterectomy and did it in about 2 hours. They managed to give 6 units of blood in the middle of the night in the course of about 5 hours. The higher centre had a team of specialists including an anaesthesiologist who is an intensivist also to look after her. A relaparotomy was done and 13 units of whole blood, 16 units of Fresh frozen plasma and 13 units of platelets were given. In spite of all these, the patient was lost. However, if we look through the case notes there are many learning points for future management.

1. On 18/1 itself it is written that cervix is 2 finger loose with placental tissue felt. This was her 3<sup>rd</sup> admission and she was already 35 wks. Usually placenta previa with recurrent bouts of bleeding has each subsequent bout heavier and heavier. Since the natural barrier to infection viz closed os, is not there, ascending infection also sets in. This baby was already more than 2 Kg. One wonders whether elective delivery at that point should have been considered
2. Immediately after delivery everything seemed to be under control but within one hour bleeding set in, not responding to oxytocics, bimanual compression etc. It is quite possible that the bleeding was from the lower segment to start with. Subsequently the uterus would have gone flabby and coagulation failure set in all because she went into shock. Bimanual compression will not be very effective in such situations. A Bakri balloon or condom tamponade will be more effective in applying uniform pressure against the open sinuses of the placental bed.
3. The decision to do total hysterectomy was questioned by some of the reviewers. That was on the assumption that it was atonic pph. For reasons described in the previous para we feel that the decision to remove the cervix was correct.
4. On relaparotomy, there is no report of the stumps bleeding. We congratulate the primary surgeon for prompt decision on hysterectomy and hemostasis from the stumps. However, the description in the case notes do not indicate whether the hysterectomy was done the way an obstetric hysterectomy should be done. The special aspects of an obstetric hysterectomy are the following –

Put clamps and cut the upper pedicle, push down the bladder, and put clamps to the uterines safe guarding the ureters; then only start tying the stumps. All pedicles especially vascular ones should be double ligated.



5. One of the reviewers suggested in a similar context, rather than relaparotomy, a tube drain and correction of coagulopathy will be better. The suggestion was to use commercially available recombinant Factor 7. It can be considered as one option. However, there will always be the nagging fear of whether we are missing a bleeder inside and so the decision to do a relaparotomy is understandable.
6. Transporting a critically ill patient on ventilator to a higher centre is a nightmare. In this case the peripheral centre did the best possible by continuing ambu ventilation and the anesthesiologist accompanying the patient and handing over to the higher centre. This highlights the need for availability of ambulances with transport ventilators.

#### Example 4

This 30 year old G3P1L1 was admitted with leaking 2days after EDD at 5.15am. She was already on "alphadopa" half tablet twice daily.. Seen by consultant and found to have partially effaced cervix, 2 finger dilated. Tramadol, diazepam2mg and drotaverin 1amp I V given. Pitocin was started at full dilatation. Vacuum delivery with episiotomy at 6.38 am. Baby 3.4 Kg. On suturing episiotomy bleeding more than normal, vaginal varices noted. BP fall at 7.45am. Inj prostodin, efcorlin etc. Bleeding time and clotting time normal. Took to theatre for exploration. No cervical tear, cavity empty. Subtotal hysterectomy done. She went into anuria. One bottle of B negative blood given. Declared dead at 11.15am.

This is another example of rapid deterioration due to PPH, again highlighting the importance of early recognition and prompt action. The records suggest that she had excess bleeding before the fall in BP and consultants came into action. Thereafter decision for intervention was taken fairly promptly but by then patient had already gone into shock with multiorgan failure. This is another reminder of the importance of the PPH drill. Delay /difficulty to procure blood was only one of the contributing factors.

One cannot help wondering what the need for that drotaverin was and whether that also contributed to the excess bleeding and final outcome.

#### Traumatic PPH

There were 10 deaths assigned to this category. Their average age was 28.8 and gravidity 3.3 and parity 2.6. One of them had vacuum delivery, another one vacuum delivery completed with forceps, two had caesarean section. One patient had rupture uterus diagnosed prior to delivery and hence had laparotomy and hysterectomy. The others had vaginal delivery. It is difficult to say how many of these patients had induction. As per the notes almost all those ending in vaginal delivery had spontaneous onset except one where after failure of pitocin drip some "tablet" was put in the vagina following which contraction started.

All these patients were referred to a higher centre. Two of them died on the way and two others reached gasping and could not be salvaged. Others lived for a variable duration of few hours to few days.

Four patients had relaparotomy at the higher centre and continued to live from 2 days to 12 days. Their course after relaparotomy was towards MODS .

## Learning from Examples

### Example 1

This 32 yr old primi had spontaneous delivery at 6.20am . She was immediately referred to the higher centre because of bleeding. She reached the higher centre in shock with altered sensorium, was resuscitated and taken to the theatre for suturing vaginal lacerations under anesthesia. By noon her condition was still critical, not responding to stimuli but BP was 180/120 and SPO2 80%. US Scan showed minimal free fluid in the peritoneal cavity. She was taken to the theatre again and resuturing done. But her condition remained critical, multiorgan dysfunction developed and she succumbed after 4 days.

## Learning Points

1. It is understandable why the doctor at the subcentre referred her to a higher centre. But, was anything done to arrest the bleeding? It is not clear from the notes but most likely nothing was done. Nor is there any information whether an I V line was started or the higher centre was alerted about the case coming. All these make a big difference in the outcome.
2. Most likely she reached the higher centre in a nonsalvageable state but the anaesthetists notes prior to suturing the lacerations indicate a conscious patient, drowsy, and responding to commands with a BP of 110 systolic. But it is to be assumed that the suturing was not satisfactorily done. She subsequently had another attempt at suturing, presumably by a senior member. The second attempt was indicated because her condition did not improve and there was minimal fluid in the peritoneal cavity. But there was no mention of a laparotomy. Probably there were multiple irregular lacerations with anatomy distorted by the first attempt at suturing. In such cases it is very difficult to achieve haemostasis and restore normal anatomy. One often forgets that the internal iliac supplies the descending cervical and vaginal arteries and that internal iliac artery ligation can reduce the vaginal bleeding.

### Example 2

This 35 year old G3P3 had delivery at local hospital of a stillborn baby. She developed PPH and hence was referred to the district hospital and from there to the

medical college. She was admitted there with features of shock and DIC. Examination revealed a cervical laceration extending upwards. Concurrent with resuscitation, laparotomy was done. Haematoma was noted in the lower segment. Total hysterectomy was done.. Six hours later she had relaparotomy for further internal bleeding. There was bleeding from the stumps which was arrested. She received 10 units of blood, 2 units plasma and 1 unit of platelets. Subsequently renal function deteriorated, right lobar pneumonia developed, shifted to ICU, dialysis done. Patient expired on day 6 from multi organ dysfunction syndrome.

### Learning Points

1. Diagnosing a cervical laceration would not have been difficult if only a proper cervical inspection set was used.
2. Referring to the district hospital where she did not get admitted and then being taken to the medical college meant loss of valuable time. If only there was an attempt to contact the higher centre and brief them about the patient being referred, this would not have happened.
3. The patient who was in shock on arrival had to have two laparotomies in the course of 6 hours. This certainly would have contributed to her subsequent deterioration.
4. While doing surgery on a patient with low blood pressure it is possible to miss some bleeders which may get activated when blood pressure rises. It is mandatory to leave a drain in such situations. It is not clear whether a drain was kept after the first laparotomy

### Example 3

This 28yr old gavida 2 had a vacuum delivery at a peripheral hospital. Soon after delivery she had bleeding per vagina and abdominal distension. She underwent subtotal hysterectomy. During that surgery she had cardiac arrest, was resuscitated but the bleeding persisted and hence was referred to the higher centre . There, ultrasound scan showed large pelvic haematoma. Radiological investigation showed feeder vessels and hence bilateral uterine artery embolisation was done. But bleeding continued. Four hours later relaparotomy was done and bleeding from ovarian vessels was identified and ligated. Haemostasis was incomplete even after that and hence closed leaving a pack behind to be removed through vagina. Her condition continued to be critical. There was ischaemic change of the left lower limb. Four days later she had profuse bleeding. There was multi organ dysfunction and she expired on 19<sup>th</sup>.

### Learning Points

It should be admitted that there was aggressive approach in the management of

this patient from the 1<sup>st</sup> and the 2<sup>nd</sup> centres. Modern techniques like embolisation was employed. Still she continued to bleed from another pedicle, re emphasizing the need to secure the pedicles in such laparotomies. This patient shows one of the complications of embolisation – occlusion of unintended vessels.

#### Example 4

This 28yr old gravida 4 with EDD on 7<sup>th</sup> was admitted on 29<sup>th</sup> of the previous month at 7.45am with labour pains. Cx was 2 finger loose. Pitocin drip 2.5 units in 5% dextrose and inj Valethamate 1 amp IV hourly was started. At 11.50am she had a bout of bleeding which stopped abruptly. Cx was 4/5 dilated. Vacuum was applied which brought the head down to the perineum. Delivery was completed with forceps. Methergin was given. Placenta delivered. She continued to have some bleeding. IV fluids, prostodin and IV Revicee etc were given. She was referred to a higher centre but died on the way.

#### Learning Points

1. There are many deviations from the standard practice in this patient. One example is putting oxytocin 2.5units in 500ml of 5% dextrose. Our recommendation is that a standard dose of 5 units in 500ml be followed and that instead of 5% dextrose an electrolyte solution like normal saline or ringer lactate be used.
2. There was no compelling reason to apply ventouse at 4/5 dilatation of the cervix, that too with head at a high station only to require the forceps to complete the delivery. Sequential use of instruments have serious consequences for the mother and the fetus.
3. We cannot support the hourly intravenous administration of Valethamate till delivery as we fear that it can increase the risk of PPH and possibly amniotic fluid embolism.
4. No attempt seems to have been made to ascertain the cause of bleeding. Applying instruments before full dilatation and sequential use of ventouse and forceps are known to increase the risk of genital tract trauma. No attempt seems to have been made to check for this, nor was any first aid like packing or tamponade employed before referring the patient.
5. The lack of communication between the periphery and higher centre is again worrying. A reference letter, eventhough essential, is not a substitute for direct contact between the referring and receiving doctors

Unfortunately postpartum haemorrhage continues to be one of the leading causes of maternal mortality in our state. Of the 26 deaths due to postpartum haemorrhage, 16 were atonic and ten traumatic type.

## Placenta previa

There were four cases assigned to this group. Three of the four had previous caesarean section. It is well known that when placenta previa occur in previous caesarean the chance of morbid adhesion and invasion of bladder is high.

Management of major degree of placenta previa is a challenge especially if the placenta is morbidly adherent or invading the bladder. The whole team should be mentally and physically prepared to tackle the problem. We recommend the following

- Atleast 4 units of blood should be crossmatched and kept ready and more should be readily available if the need arises.
- Urologist should be on standby unless the obstetrician feels confident in tackling urological injury
- The anaesthesiologist should be aware of the gravity of the situation
- Two 16 G IV lines and a CVP line should be in place.
- Abdomen should be opened by vertical incision
- Open the uterus by upper segment vertical incision away from placenta.
- Once fetus is delivered and cord cut short, uterus may be exteriorized.
- If placenta is morbidly adherent and there is no bleeding, there is the option of leaving the placenta in situ after tying the cord close to placenta. It is worth remembering that secondary PPH, infection etc are possible. Methotrexate to hasten placental necrosis and absorption is now questioned as the drug may not reach the placenta unlike in a case of trophoblastic disease where the lesion is vascular.
- If the decision is to remove the placenta or do hysterectomy – we recommend ligation of the internal iliac on both sides.
- Tackle the cornual pedicles first and tie the uterines as much as possible.
- Once the bladder is separated severe bleeding will occur. Anticipate this, separate the bladder, apply the double clamps on sides and across the cervix /vagina and remove the uterus. Ligate the stumps. Bleeding from bladder side may require bulk stitches.
- Uterosacral also may contain active bleeders and will require special care.
- If bladder is entered, see the ureteric orifice and if necessary stent it. Remove any invading placental tissue from bladder and attain haemostasis before closing rent in layers.
- Leave one or more drains.

## Abruptio Placenta

There were 8 cases assigned to this cause of maternal death. It is heartening to note that abruptio placenta which used to equal or exceed PPH as a cause of death has come down in incidence. This could be due to the increased intake of folic acid, improved nutritional status and reducing parity of the obstetric patients.

Once abruption starts it can progress rapidly and kill both mother and baby. Prompt delivery is the only way to save these lives. DIC and renal failure are natural sequelae. Concealed abruption is a deceiver when the placenta is posterior. The tense tender uterus that usually raises suspicion of the condition will also be missing. Awareness of this possibility is essential for all obstetricians faced with women in late pregnancy complaining of vague symptoms and unexplained fetal distress.

Time interval from onset of abruption to delivery is the critical factor that decides outcome. If cervix is unfavourable and vaginal delivery is not imminent, one should not hesitate to do caesarean. This applies to cases of even fetal death and unfavourable cervix

## Learning from Example

### Case 1

This 25 yr old, gravida 2 at 37 wks of pregnancy was admitted with complaints of backache. She was given symptomatic treatment, improved and discharged the next day. A day later she came back with severe breathlessness, abdominal pain and blood stained discharge pv. Cyanosis was present. Suspected amniotic fluid embolism or abruptio placentae. LSCS was done under general anesthesia. Uterus was full of clots, fetus was dead. Cardiac arrest occurred and she died inspite of attempted resuscitation.

### Learning Points

Obviously on the first day itself the abruption would have started. It is unfortunate that the diagnosis was missed with grave consequences. "Only what is in mind, the eyes see".

### Case 2

28yr old G3 P2 admitted to a tertiary centre in a critical state with DIC, shock etc due to abruptio placentae. Cervix was 1 cm long, 2cm dilated. Considering the delay for vaginal delivery decided for immediate caesarean section. During intubation cardiac arrest occurred, resuscitated, and caesarean completed. Atonic pph – subtotal hysterectomy. Coagulation problem got corrected with blood and blood products. Next day DIC reappeared, renal function deteriorated, ventilatory care continued. Peritoneal dialysis



was started. She seemed to improve further. Ultrasound showed abdominal wall haematoma and free fluid in peritoneal cavity. After about 36 hrs, due to some ventilator malfunction she had to be changed to ambu ventilation. In about 4 hours she expired.

### Learning Points

1. This patient was referred from Taluk hospital to District hospital to Medical College. A more direct referral to Medical College would have avoided some delay.
2. The decision to do caesarean section and later hysterectomy in the presence of DIC and shock may be questioned. It was really a difficult situation. Waiting to get the general condition improved also could be questioned. So we cannot blame the decision taken to proceed surgically. However, an alternative strategy could have been to put scalp traction and oxytocin after ARM while getting the patient resuscitated with blood and blood products. In any case not more than one or two hours should have been spent on these.
3. Even when surgery was planned, a low vertical midline may be preferable to a transverse incision in the presence of coagulation failure. There will be less chance of subsequent haematoma as less vessels are cut.
4. It is really unfortunate that after all those heroic attempts finally the patient was lost due to nonobstetric cause. The root cause of such tragedies have to be looked into.

### Management of postpartum haemorrhage:

Postpartum hemorrhage (PPH) is an obstetrical emergency that can follow vaginal or cesarean delivery. It is a major cause of maternal morbidity and mortality, with sequelae such as shock, renal failure, acute respiratory distress syndrome, coagulopathy, and Sheehan's syndrome. The incidence is approximately 3 to 5 percent of births. WHO estimates that there are 14 million obstetric haemorrhages a year, and postpartum haemorrhage is the single most common cause of maternal death worldwide<sup>(2)</sup> In September, 2003, at its triennial meeting in Santiago, Chile, the International Federation of Obstetrics and Gynecology (FIGO) made postpartum haemorrhage its top priority<sup>(3)</sup>.

### Prevention:

FIGO and the International Confederation of Midwives recommend active management during the third stage of labour as the optimum way to reduce postpartum haemorrhage. <sup>(3)</sup> Active management of third stage include-

1. Early oxytocic therapy (with the delivery of the anterior shoulder or shortly after the delivery of the baby.) Oxytocin 10 units IM or IV or methergin 0.2

- mg IM or IV (avoid methergin if the mother is hypertensive)
- 2. Uterine massage
- 3. Early cord clamping and placental delivery by controlled cord traction :  
(Early cord clamping is now removed from the recommendations.)

The use of ergometrine and oxytocin as part of the routine active management of the third stage of labour appears to be associated with a small but statistically significant reduction in the risk of PPH when compared to oxytocin alone for blood loss of 500 ml or more. A statistically significant difference was observed in the incidence of maternal side effects, including elevation of diastolic blood pressure, vomiting and nausea, associated with ergometrine and oxytocin use compared to use of oxytocin alone<sup>(4)</sup>.

It should be noted that both oxytocin and ergometrine have to be kept in the refrigerator to keep its potency, but ergometrine deteriorates faster than oxytocin.

## Prostaglandins

- 15(S)15 Methyl PGF<sub>2α</sub> 125µgm I/M .

## Misoprostol:

- Rectal Misoprostol 600µgm per rectum.

Neither intramuscular prostaglandins nor misoprostol are preferable to conventional injectable uterotonics as part of the active management of the third stage of labour especially for low-risk women. Routine prophylactic oxytocin is recommended in all patients at the delivery of the anterior shoulder.

## Management of postpartum haemorrhage - The PPH drill

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

## Call for help

- Call experienced obstetricians
- Alert Anaesthetic Consultant
- Alert blood transfusion service
- Alert Theatre staff
- Call more staff ,Nursing / paramedics



## Resuscitation

- I V access (16 G cannula x 2)
- head down tilt
- oxygen by mask at 8 litres / min
- Transfuse blood as soon as possible.

Until blood is available, infuse in turn (as rapidly as required):

- crystalloid (eg Normal saline) maximum 2 litres
- colloid (e.g.Starch Gelofusine, Haemaccel, human albumin 4.5%) maximum 1.5 litres

## Monitoring and Investigation

- 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

## 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.

### Exclude the four 'T's of PPH :

1. Tone - Atonic uterus
2. Tissue- Retained products (placenta, membranes, clots)
3. Trauma- Vaginal/cervical lacerations or haematoma, 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 methergin and oxytocin (Methergin.0.2 mg and oxytocin 5 units i/v as bolus injection).Oxytocin infusion 20units in 500 ml of normal saline – 30 drops /minute
- “Rub up the fundus” to stimulate contractions
- Carboprost 0.25mg IM.
- Ensure bladder is empty (Foley 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 hydrostatic condom tamponade or packing)

If conservative measures fail to control haemorrhage, initiate surgical methods to arrest bleeding **SOONER RATHER THAN LATER**

- At laparotomy, direct intramyometrial injection of Carboprost 0.5mg
- B-Lynch or Hayman’s sutures
- Bilateral ligation of uterine arteries and anastomotic branch of the ovarian artery.
- Bilateral ligation of internal iliac arteries.
- Hysterectomy (Resort to hysterectomy **SOONER RATHER THAN LATER**)

Conservative measures may be tried while arranging for surgical methods.

- Bimanual compression of the uterus.
- Uterine packing.
- Hydrostatic condom tamponade.

## **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 i/v line with 18-16 G cannula 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 PPH should be transferred to tertiary care centers early after giving repeat dose of methergin, and pitocin i/v with a drip containing 20 units of pitocin, Inj..Prostaglandin i/m, and misoprostol 600µgm per rectum with out delay.
- Patient can develop shock and may die within 2 hrs of onset of postpartum haemorrhage.
- 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 the last resort..
- PPH is a leading cause of maternal mortality and many such deaths are preventable.

### **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 ante flex to control the blood flow temporarily.

### **Uterine packing**

Uterine packing can be effective in controlling PPH if performed correctly.<sup>(5)</sup> After holding the cervix with ring forceps,thick rolles 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 haemorrhage.**

Hydrostatic balloon tamponade using a catheter fitted with a condom to control massive postpartum haemorrhage was first reported from Daka, Bangladesh,<sup>(6)</sup> In an observational study during 2000 to 2001,23 women with postpartum haemorrhage due to uterine atony with uncontrolled bleeding despite adminis-

tration of uterotonics had the condom catheter placed and the bleeding was reported to have stopped within fifteen minutes. Pitocin i/v drip was continued and condom catheter was removed after 24 to 48 hours. No further intervention was necessary.

### Steps :

Keep the bladder empty by Foley's catheter.

Insert a sterile catheter into the condom, preferable to use two, one inside the other, and tie it tight at the neck of the condom.

Insert the condom along with the catheter into the uterine cavity, pack the cervix and vagina to prevent the condom from slipping down.

Connect the catheter to a saline set and fill the condom with 300 to 500 ml of saline.

Maintain the uterine contraction by continuous pitocin drip.

Condom is kept in place for 24 hours.

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## Chapter Seven

# Hypertensive Disorders of Pregnancy

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On behalf of the Editorial Board.

### Pre-eclampsia and Eclampsia: Key Summary Points.

- Eclampsia is the most common predisposing cause for death among the hypertensive diseases of pregnancy.
- The largest single cause of death is intracranial hemorrhage (10 cases) – reflecting a failure of effective antihypertensive treatment.
- Delay in referral to a better facility occurred in four cases and could have contributed to the mortality.
- Multiorgan dysfunction led to the death of seven cases.

### Pre-eclampsia and eclampsia : Key recommendations.

- All pregnant women should have regular blood pressure recording during their antenatal visits. They should be educated on the warning symptoms of severe disease such as headache and epigastric discomfort.
- Clear written protocols should be followed in all institutions. Urine examination for albumin and blood examination for liver function tests should be considered in all cases of pre eclampsia and repeated at suitable intervals to assess the progress of the disease.
- Treatment of hypertension even when the blood pressure is > 140 mm of systolic and > 90 mm of diastolic seems to be a desirable option in our set up. This is contrary to the conventional teaching.
- The dose of antihypertensive drugs should be titrated to get adequate blood pressure control. Single agent treatment may not be sufficient in most situations.

- Alphasol may be used as the first line drug for long term control of hypertension. However, if liver dysfunction is seen, alphasol has to be stopped. Hydralazine and labetalol are recommended as the second line drugs. Nifedipine may be used to bring down blood pressure in acute hypertensive crisis if parenteral preparations of labetalol and hydralazine are not available. The committee is aware of the fact that use of nifedipine is discouraged in some countries. However in centres where parenteral agents like labetalol or hydralazine are not available sublingual nifedipine may be used but the dose should not exceed 5mg.
- Magnesium sulphate is the anticonvulsant of choice in most cases of eclampsia. However the obstetrician should employ antihypertensives in all these cases to bring down blood pressure to safer levels to avoid cerebrovascular catastrophe.

### Pre eclampsia and Eclampsia - Learning Points

- Early onset pre-eclampsia poses serious threats to the mother as well as the fetus. Thrombophilia should be excluded in such cases.
- The chance of persistently elevated systolic blood pressure leading to intracranial hemorrhage should be clearly recognized in all cases of pre eclampsia and more aggressive management of hypertension is required.
- The service of senior obstetrician should be available when severe cases of pre eclampsia and eclampsia is managed. Multi disciplinary approach also is necessary in most cases.
- Magnesium sulphate is the anticonvulsant of choice in cases of eclampsia and severe pre eclampsia.
- Invasive monitoring and C.T evaluation are necessary in some cases.
- Clearly laid down protocols should be enforced to prevent sub optimal management and adverse outcome.

### Summary

There are 41 cases of eclampsia/ pre eclampsia noticed in the present series. These include cases reported to CRMD and the DHS of which 29 cases were reported to CRMD. Out of this, sixteen cases are due to eclampsia, ten cases due to severe pre eclampsia, and three cases due to HELLP syndrome. In four cases the details were not available for analysis. As this is the first confidential review we are unable to make a comparison with previous figures. However (in the light of available data from institutions) the committee feels that there is a decrease in the number of deaths from hypertensive disorders.

Table 7.1 Causes of maternal death -a comparison with previous years

Cause of maternal death	*Institutional data 93-97 ( n=310 )	**2001 Pilot study ( n= 105 )	2004 CRMD ( n= 154 )	2005 CRMD ( n=153)
Hemorrhage	53	23	29	32
Hypertension	73 (23.5 % )	15 (14 % )	19 (12.3 % )	22 (14.3%)
Hepatobiliary	51	10	10	7
Heart disease	30	10	12	17
Sepsis	27	6	7	12
Anemia	8	-	5	6
Abortions	13	4	0	3
Amniotic fluid embolism	-	6	17	13
Venous thromboembolism	-	2	13	5
Anaesthetic causes	4	2	4	0

\* Based on statistics from 5 Medical Colleges in Kerala

\*\* Study conducted by Maternal fetal medicine committee of KFOG.

### General discussion:

Majority of the deaths are from eclampsia (55 %) and the remaining due to severe pre eclampsia and or HELLP resulting in Multiorgan dysfunction. The findings in 29 cases whose details are made available for scrutiny are summarized in the following table.



Table 7.2 Summary of cases

Sl no	Diagnosis	Age	Gravida	Period of gestation	Pathological cause of death
1	Eclampsia	23	3	36 wks	HELLP, MODS
2	Severe Pre eclampsia	28	2	31+wks	Intracranial hemorrhage
3	Severe pre eclampsia	32	2	30 wks	Intracranial hemorrhage
4	Eclampsia	24	3	39 wks	Missed diagnosis,
5	Eclampsia	20	1	30wks	Eclampsia,\$
6	Eclampsia	35	3	37wks	Eclampsia*
7	Eclampsia	36	1	34wks	Eclampsia*
8	Eclampsia	26	1	38wks	Intracranial hemorrhage @
9	Eclampsia	23	1	40wks	ARDS
10	Severe Pre eclampsia	19	1	31wks	Pul.embolism
11	HTN-Superimposed pre eclampsia	36	3	25	Pul.embolism
12	Eclampsia	32	3	39wks	Intracranial hemorrhage.
13	Severe Pre eclampsia	25	1	38wks	DIC
14	HELLP	28	1	38wks	PPH, Hysterectomy, DIC
15	Eclampsia	32	3	37wks	Intracranial haemorrhage.*
16	Eclampsia	28	1	40wks	Aspiration pneumonia, Sepsis
17	HELLP	28	2	38wks	DIC, MOD
18	Severe Pre eclampsia	30	3	36wks	ARDS
19	Severe pre eclampsia	28	2	31wks	Intracranial haemorrhage.*
20	HELLP	32	1	39wks	MOD*
21	Eclampsia	28	2	41wks	Intracranial haemorrhage
22	Eclampsia	30	1	36wks	Intracranial haemorrhage
23	Severe Pre eclampsia	20	1	35wks	MOD
24	Severe Pre eclampsia	34	2	33wks	MOD
25	Severe Pre eclampsia	28	5	28wks	Abruption, DIC
26	Eclampsia	19	1	37wks	MOD
27	Eclampsia	23	2	31wks	Intracranial haemorrhage
28	Eclampsia	21	1	39wks	Intracranial haemorrhage *
29	Eclampsia	24	2	39wks	HELLP, MOD

\* Suboptimal care might have contributed to the adverse outcome.

\$ Social factors – lack of resources to take the patient to a higher centre, and non availability of any voluntary help

@ LSCS postmortem.

### Pre eclampsia:

In ten cases, severe pre eclampsia led to the death of the women. In three of these, intracranial hemorrhage was documented by C.T studies, and in two other cases,

a diagnosis of pulmonary embolism was made based on the sequence of events prior to death. DIC and multiorgan dysfunction led to the death in other cases.

### **HELLP:**

HELLP syndrome was the contributory cause of death in three cases. In the first case, a case of eclampsia, when she was admitted to the tertiary care centre, she had three convulsions and was jaundiced. Uterus was 28 wks size, FHS absent. All the biochemical parameters were elevated. Following delivery she went in for DIC, multiorgan dysfunction and death.

The second case had hepatic failure following a caesarean section. This woman was normotensive before delivery. The possibility of other causes of hepatic failure is not completely ruled out in this. The third case was a severe pre eclampsia patient.

### **Delay in delivery:**

In two cases delay in delivery of the fetus could have contributed to the fatal outcome. In the first case the patient with eclampsia was seen in two hospitals where emergency obstetric service was available. But without proper first aid measures the patient was sent to a third hospital where she reached in a moribund state and resuscitative measures failed. She died undelivered.

In the other case, it was pre eclampsia, and the severe hypertension was treated from 19 wks with three drugs. Induction at 25 wks was successful, but the mother soon died of pulmonary embolism. This woman had hypertension in her previous pregnancies also and the chance of prolongation of pregnancy from 19 weeks with three antihypertensives was unlikely. An earlier termination would have made a difference. A second opinion from a senior obstetrician may be sought in such situations.

### **Suboptimal management:**

Suboptimal care of minor degree can be identified in five cases. Inadequate doses of magnesium sulphate, choice of epsolin as the first line drug for treatment, and inadequate control of the hypertension are the points noted in these cases. No major degree of substandard care is identified in any of the cases.

### **Eclampsia:**

The majority of deaths (16/29) in the hypertensive pregnancy occurred due to eclampsia. In seven cases the death was due to intracranial bleed, and in three, multiorgan dysfunction was contributory factor. In some cases no clearly proven cause could be identified.

The management of eclampsia has gone through cardinal changes in India. Ever since the classical work of Dr.M.K.K.Menon, for a long time lytic cocktail formed the mainstay of treatment and obstetricians tried to procure a vaginal delivery at any cost. However the scenario has changed considerably and the following points can be highlighted in relation to eclampsia management.

- Nursing in a dark isolated room is not ideal. Treating the patient in an intensive care setting is more useful.
- Multi organ dysfunction is the final cause of death and hence a multidisciplinary approach is associated with the best outcome.
- Magnesium sulphate (preferably the modified low dose regime) as given in the appendix is more suitable to the Indian setting than the conventional Pritchard's regime. Phenytoin, diazepam etc are inferior anticonvulsants in eclampsia.
- Effective and prompt antihypertensive therapy should be initiated without delay so that the blood pressure is brought down to 150- 140 systolic and 100-90 diastolic. This is necessary to reduce the mortality from intracranial problems.
- The concept of intercurrent eclampsia should not be entertained in the modern obstetric practice. Termination of pregnancy is desirable in all cases of eclampsia.
- Caesarean section under general anaesthesia may be the best strategy if the fetus has a good chance of survival and the cervix is unfavourable. Generally speaking procedures like cervical ripening, mechanical or medical, may be discouraged with a viable fetus. Augmentation may be considered only if the delivery can be anticipated without much delay.
- Continued convulsions in spite of anticonvulsant therapy should raise the suspicion of intracranial haemorrhage and a CT may be considered. Additional doses of anticonvulsants in such patients without a neurological diagnosis can do more harm.

In view of the above recommendations, eclampsia cases should be referred to a suitable medical facility. Intramuscular magnesium sulphate is a good option during transit period.

## **Learning Lessons from examples:**

### **Case I – “A Question of mistaken identity”**

A 28 year old second gravida was referred to a tertiary level hospital with 30 weeks + 6 days pregnancy. She had a caesarean section in her previous pregnancy. She was seen by the doctor in the peripheral center, noticed edema

all over the body, a blood pressure of 150/100 and casts in the urine. She was referred to the higher center with a diagnosis of Acute Nephritis complicating pregnancy. She was started on Alphadopa and injection penicillin before referral. At the tertiary center, the patient was seen by the resident and a nephrology opinion was sought. Nephritis was ruled out. BP was >160/100 at that time. There was severe IUGR. On day 2, Alphadopa was continued at 250 mg 6<sup>th</sup> hrly and BP came down to 140/80. A dose of (2500.I.U) of Low molecular weight heparin and 100mg aspirin was started. On day 4 the BP went up to 180/110 and the patient had headache and vomiting, became drowsy and developed hemiparesis. Magnesium sulphate therapy was initiated at this point. Physician was consulted who opined that it could be an intracranial haemorrhage in view of the raised BP and heparin administration. Patient was switched over to eptoin, and dexamethasone and a CT examination revealed Basal Ganglionic and intraventricular haemorrhage. On day 5 there was further deterioration and a caesarean section was planned. Nimodipine and sodium nitroprusside was also started. Condition remained critical and the patient expired on day 6.

Several issues may have to be raised in this case. The referring physician has recorded the blood pressure and initiated antihypertensive treatment; he/she made an erroneous clinical diagnosis of acute nephritis and referred the patient to a higher center. The resident also was carried away by the diagnosis. The antihypertensive therapy was inadequate to prevent the intracranial bleed. The indication for the low molecular weight heparin in the tertiary facility is not clearly documented and seems unnecessary. It might have even contributed to the intracranial haemorrhage. Even though post operative thromboprophylaxis is clearly indicated in PIH, its timing in this case is debatable. Obstetrician obviously did not have a clear plan regarding termination of pregnancy which was considered only when the patient became moribund.

It is necessary to have clear written protocols for management of pre eclampsia and it should be available in all hospitals where pregnant women are looked after. General practitioners should be given adequate awareness regarding importance of recognizing pre eclamptic features in pregnant women. Mistaken diagnosis at the periphery coupled with inadequate investigations and poor management of hypertension culminated in a mortality.

## **Case II - "All pains are not labour pains" – a case of suboptimal care**

26 year old primigravida with 38 completed weeks of gestation was rushed to the casualty of a tertiary level setting in the early morning hours with pain abdomen of one hour duration. Her BP was 170/100 and had pedal edema. Uterus was term with unengaged head. She had at least four antenatal visits to the hospital. After the preliminary examination she was observed for labour pains. At +2 hrs she developed tonic-clonic convulsions

and was diagnosed as eclampsia. She was given 5 mg of diazepam I.V and 200 mg of epsolin I.V. BP was 200/120 at that time, patient was disoriented but conscious. At + 3 hrs, magnesium sulphate regime was initiated and a vaginal examination was done. Cervix was 25 % effaced, vertex was at -3 station and PGE2 induction was planned. 5mg nicardia was given sublingually and blood was collected for PIH investigations. At + 4 hrs, the B.P was 180/130, the fetal heart was present. At +5 hrs patient became unconscious, uterine contractions were absent, but FHS still present. A C.T scan revealed frontal lobe haematoma with intraventricular bleed and acute hydrocephalus. Her condition deteriorated steadily, FHS absent by +7hrs. Blood examination revealed a platelet count of 98,000 and elevated liver enzymes. At + 9 hrs patient was declared dead. A postmortem caesarean section was done at request.

Eventhough the patient died about 9 hrs after admission analysis of this particular case reveals several lacunae existing in our system, even in teaching hospitals. A series of suboptimal care situations can be identified on closer look.

- Pain abdomen in primigravida with a BP of 170/100 was interpreted as labour pain and the duty resident did not think of the possibility of impending eclampsia.
- The blood pressure continued to be at dangerous levels. The antihypertensive therapy was not initiated at the appropriate time or with appropriate agents in adequate doses. In short, no efforts were made to reduce the occurrence of intracranial bleed.
- The agents selected as anticonvulsants were inferior to magnesium sulphate. Eventhough, mag.sulphate was started at a later time, the combination of these agents, especially diazepam could have had an adverse effect on the outcome.
- Decision for cervical ripening was inappropriate for the situation as delivery could be expected only after several hours. A caesarean section would have been a better option with the findings.
- Even after involving consultants, no proper effort was taken to bring down blood pressure.
- An optic fundus examination was a routine clinical examination in previous years. This was not done here also. Probably if it was done features of raised intracranial tension would have been evident. Substituting CT for a simple clinical examination is an undesirable trend in our attitude. Neither the Obstetrician nor the physician made a fundus examination in this case.

Once again we believe that this case highlights the importance of written protocols in the labour rooms. It is even more significant in conditions like eclampsia

where the obstetrician has to act promptly and in an aggressive manner failing which we lose the mother as well as the baby as exemplified in this case. The reluctance on the part of doctors to use magnesium sulphate for the treatment of eclampsia should be addressed by all concerned.

### Case 3 “A case of missed opportunities”

This 30 year old primi at 36 wks was admitted to a level 1 hospital at 12 pm with right sided upper abdominal pain since 10am after taking puttu and meat curry. BP was 140/90, edema ++, tenderness + in the right hypochondrium. Inj.cyclopam and ranitidine were given and symptoms subsided. Tab alphasopa 250mg bid was started. By 5.50 pm she c/o headache and vertigo, BP 200/140. Nicardia 10mg sublingual and Injection eptoin 100mg IV given. She developed eclamptic fits after 10mts .Inj Magnesium sulphate 4gm IV slowly given. Inj. Diazepam 10mg in 500 ml of normal saline was started IV and patient was referred to a tertiary centre.

She was admitted at the tertiary centre at 8.30 pm, BP 200/130, Cx was 2cm long, and 1.5 cm dilated, medium consistency, Vx at -2, PGE1 25microgm put in post fornix. Nicardia started and emdopa 500 q8h was started. She developed convulsions soon after admission. MgSo4 regimen was started (4gm IV & 4gm IM). LFT results arrived after about 3 hours which showed bilirubin 4.6mg and SGOT 376, Alk. phosphatase 230, platelets one lakh. While being prepared for caesarean section, she developed cardiac arrest. Intubated within about 10 minutes. Caesarean was done late without need for anesthesia as she was nonresponsive and delivered a dead fetus. Pt was on ventilator and died after 6 days.

### Learning Points

**There are several learning points from this case.**

It is to be appreciated that the doctor at the primary hospital admitted the patient and started her on an antihypertensive . Also, when she developed convulsions, Mgso4 was started eventhough it was at a suboptimal dose. A detailed note regarding the treatment given was sent to the higher centre. However, the all too common mistake of not recognizing the importance of epigastric pain and hypochondrial tenderness was repeated here also. If only it was taken as a warning sign of impending eclampsia, she would have received more aggressive anti-convulsant treatment.

At the primary hospital, even after she had convulsion the anticonvulsants used were not of adequate quantity or type. Diazepam is not advisable as a primary anticonvulsant especially when Mg So4 was available.



Even after she reached the higher centre, the aggressive approach one would expect in the management of such a patient was missing. An attempt seems to have been made to achieve vaginal delivery. MgSo<sub>4</sub> was added at the recommended dose but even that seems to have been delayed. It is not clear whether it was given after the patient had further convulsions. One should appreciate that the administration of diazepam from the periphery would have influenced the decision at the higher centre. Polypharmacy always creates confusion.

The administration of antihypertensives also deviated from what is recommended. The first dose given at the periphery was 10mg sublingual. We would not recommend more than 5mg sublingually at one dose. Even after she reached the higher centre, already having had convulsions the antihypertensives given initially were alphadopa and nifedipine. Alphadopa is too slow a drug to be of use in such an emergency. One would have expected the use of labetalol intravenously (parenteral hydralazine not being available).

After the patient developed cardiac arrest, intubation was delayed by about 10mts. One can suspect many possible causes for such a delay like nonavailability of necessary equipments (laryngoscope and endotracheal tube) or trained personnel, again something needing improvement in most of the centres. The importance of an emergency trolley with necessary equipments and drugs and ability of staff (doctors & nurses) in resuscitative measures cannot be over emphasized.

The liver function results seem to have convinced the team at the tertiary centre to go for immediate caesarean section. But by then it was too late as she developed cardiac arrest possibly as a sequel to intracranial haemorrhage.

### **The following lessons are taught by this unfortunate event**

- The possibility of impending eclampsia should be suspected when a patient in the third trimester complains of epigastric pain or has hypochondrial tenderness.
- In such patients blood pressure should be rechecked at more frequent intervals even if the initial level is not very high.
- When high BP is noted or eclampsia is revealed, aggressive approach to control hypertension is required, which will need parenteral drugs.
- Polypharmacy to control convulsions (mixing eptoin, diazepam and Mgso<sub>4</sub>) is dangerous as it will not attain therapeutic level of any of the drugs and confuse further drug administration.
- Once eclampsia has developed (and even in impending eclampsia) delivery should be prompt and most of the time by caesarean section. Ripening of cervix and induction may be justified only in a very rare situation.

- Resuscitation of an acutely collapsed patient will be required in many clinical situations in obstetrics –PPH, amniotic fluid embolism etc. Hence the readiness in terms of equipments and training of staff should be insisted on.
- A word of appreciation is also called for here. The primary doctor sent a detailed report to the higher centre. It is not clear whether tertiary centre was alerted on the phone also. The tertiary centre is also to be commented for documenting all the events in detail with time and an honest reporting of the events to the CRMD

### **Governmental Decisions – Recommendations from CRMD Committee**

- Magnesium sulphate may be made available in all primary, secondary and tertiary care centres to promote its wide spread use for treatment of eclampsia, for transit periods and for use in severe pre eclampsia cases.
- Free transport directly to a tertiary care may be provided for Eclampsia cases.
- C.T examination may be done free of cost for eclampsia patients (Private institutions also can be motivated to do the same on humanitarian grounds)
- When drug manufacturing licenses are issued to companies, insist that each company manufactures at least one life saving drug like magnesium sulphate, hydralazine, labetalol etc. This policy will ensure liberal supply of such drugs.

## **Annexure A**

### **Guidelines for management of Pre eclampsia:**

#### **Diagnosis:**

#### **Pre eclampsia**

Hypertension (>140/90 mmHg) with Proteinuria (> 0.3g/day)

#### **Severe Pre eclampsia:**

Any one of the following

Severe Hypertension (Systolic >160 and diastolic > 110 mmhg) or

MAP > 125 mmHg.

Headache, epigastric pain, visual disturbances

Massive Proteinuria (>5gm/24 hrs)

Oliguria (<400 ml /24 hrs)



Platelet count < 100,000  
Elevated SGOT & SGPT (>twice normal values)  
Fetal Growth restriction

\* Beware of atypical presentations rarely

### **Maternal Observations in Severe Pre eclampsia:**

Blood pressure recording every 30 mts.  
Indwelling Foley and urine output chart.  
Blood counts, urea, electrolytes, Liver enzymes – daily

### **Fetal Assessment:**

Growth assessment scans.  
Liquor volume.  
Umbilical artery Doppler  
C.T.G

### **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.

## **Annexure B**

### **Management of Eclamptic seizures:**

- 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.
- Maintenance infusion of 1-2 gms/hr, IV
- Recurrent seizures – think of intracranial bleed; do CT before giving Diazepam or other drugs.
- Continue magnesium sulphate for 24 hrs after last seizure.
- Monitor urine output, tendon reflexes. Stop if urine < 30ml/hr, Resp < 14/Mt and SpO<sub>2</sub> < 95 %

## Magnesium Sulphate Regime

### Eclampsia

0 hrs	4 gm (20%) slow I.V. over 5 mts + 4 gm deep IM with 1 ml 1% xylocaine
0+2 hrs	4 gm IM or slow I.V.
0+6 hrs	4 gm IM or slow I.V.

Continue 4<sup>th</sup> hourly, till 24hrs after delivery or last seizure.

If syringe pump or infusion pump is available, give I.V. infusion at the rate of 1 gram per hour, after loading dose.

### Impending Eclampsia

Loading dose 4 gm IM and 4 gm I.V. as at 0 hr for Eclampsia.

Continue 4 gm deep IM 4 hourly or 1gm/hour IV.

### Monitoring

Before every injection, check:

- Patellar reflex
- Respiratory rate
- Urine output

### Antidote

10 ml of 10% Calcium Gluconate.

### Preparations of MgSO<sub>4</sub>

50% solution	8 ml is 4 gm
25% solution	16 ml is 4gm
20% solution	20 ml is 4 gm

### Administration

**IM injections** : Should be at gluteal region, Use 50% solution.

**IV injection** : Give slowly, taking at least 5 mts for 4 grams

**IV infusion** : Dilute before I.V. Administration : MgSO<sub>4</sub> can be given as IV infusion using microdrip set or syringe pump or infusion.



## Chapter 8

# Amniotic Fluid Embolism

**Dr.Presannakumari**

**Dr.P K Shyamala Devi**

**Dr.V P Paily**

On behalf of the Editorial Board

### Key Summary Points

- Amniotic fluid embolism emerged as one of the leading causes of maternal deaths in Kerala.
- There were 30 deaths assigned to AFE out of a total of 307 deaths in the two years. 19 of them were reported to the committee.
- The diagnosis was made on clinical grounds. Some of the cases did not have all the features to clinch the diagnosis of AFE and other possible causes of acute collapse could not be ruled out.
- A stringent analysis shows that for 10 cases the diagnosis of AFE will be the most appropriate. This chapter deals with only those 10.
- Once the signs and symptoms of AFE started, deterioration was rapid leaving very little time for rescue.
- Hyperstimulation of uterus was a common association of AFE. Use of prostaglandin (E<sup>1</sup> and E<sup>2</sup> ) was noted in 7 of 10 cases
- Use of smooth muscle relaxants with oxytocic agents seems to increase the risk of AFE. Five of the 10 cases had such combination of drugs used.
- The symptom triad that was looked for to make a diagnosis was convulsion, coagulation problems and chest discomfort rapidly leading to collapse and cardio respiratory arrest.

### Key Recommendations

As AFE is unpredictable and deteriorates rapidly once it sets in, strategies for prevention are difficult; however the following recommendations are made on the basis of our observations and literature review. Circumstantial evidence strongly

implicates oxytocic agents like prostaglandins to lead to hyperstimulation. Combined use of prostaglandins and smooth muscle relaxants increases the risk further. Hence, that practice should be stopped until properly conducted studies prove otherwise. Though the course of the disease is rapid and involving multiple systems leading to rapid deterioration, early recognition and aggressive attempts at resuscitation could make a difference to the outcome. Obstetricians, and labour room staff have to be trained for this and facilities for resuscitation and basic life support should be 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, ambu bag etc. The head nurse in charge of labour room may be the ideal choice. A mechanism should be evolved in each hospital for quickly summoning the help of other specialists like anesthesiologist, physician and cardiologist in cases of acute collapse. The availability of blood and blood products becomes crucial in AFE as many of them 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 the dose and route of administration of emergency drugs should be displayed in every labour room.

## Incidence

This being the first confidential review, we could compare it only with the available data on maternal deaths collected between 1993 and 1997 by five government medical colleges within Kerala, and an earlier CRMD pilot study conducted throughout the state in 2001.

Table 8.1

Cause of maternal death	*Institutional data93-97 ( n=310 )	**2001 Pilot study ( n= 105)	2004 CRMD+DHS ( n= 154 )	2005 CRMD+DHS ( n=153)
Hemorrhage	53	23	29	32
Hypertension	73 (23.5 % )	15(14 % )	19(12.3 % )	22(14.3%)
Hepatobiliary	51	10	10	7
Heart disease	30	10	12	17
Sepsis	27	6	7	12
Anemia	8	-	5	6
Abortions	13	4	0	3
Amniotic fluid embolism	-	6	17	13
Venous thromboembolism	-	2	13	5
Anaesthetic causes	4	2	4	0

\* Based on statistics from 5 Medical Colleges in Kerala

\*\* Study conducted by Maternal fetal medicine committee of KFOG.

AFE was not listed 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 17 out of 154 and in 2005 there were 13 out of 153. These are too limited data to comment on any trends. The committee is aware that the number of cases where AFE was cited as the diagnosis was much more than what is finally put down by the CRMD. Out of these 30 cases for the two years, 11 cases were accepted without critically looking at the details as the case notes were not available to the committee. Even among the 19 finally assigned by the committee to this group, there can be difference of opinion. Finally on the basis of more stringent criteria, the editors assigned 10 cases to this category (see table 8.2). Further analysis will be limited to these 10 cases.

**Table 8.2 Summary of Cases**

No	Age	Gr	gest- ation	Clinical features	Induction	Time - Event to death	Mode of Delivery
1	29	2	40	Convulsions,, dyspnoea	Cerviprime,Epidosyn, ARM	4hr 30'	outlet forceps
2	24	1	40	Convulsions, BP fall, Cardiac arrest	Cytotec sublingual, ARM,Epidosyn	1hr 30'	undelivered
3	29	2	40	Restless, cyanosed, hypotension, cardiac arrest	Misoprost, ARM,Drotin	4hrs 30'	outlet forceps
4	28	3	38	Convulsions, cardiac arrest, DIC	Oxytocin, ARM	16 days	LSCS
5	33	5	38	Chest discomfort, cough, fits,DIC	PGE1-multiple doses	4-5 hrs	Vacuum extraction forceps
6	31	2	40	Chest discomfort, fits, cyanosis	Cerviprime, epidosyn, ARM		
7	26	2	40	Convulsions, cyanosis, cardiac arrest, DIC	Misoprost, ARM, Pitocin	1hr 20'	undelivered
8	30	2	39	Convulsions, cyanosis, unconscious	Spontaneous labour	30'	Undelivered
9	29	2	39	Convulsions, cardiac arrest	Misoprost,	2 hrs	Undelivered
10	30	3	39	DIC, while doing Caesarean section	cs for fetal distress	4 hrs	LSCS

## General Observations

### Parity and age

Six of the ten patients were gravida 2 and most of them were in the age group 20-

30. These only reflect the general features of obstetric cases and do not seem to indicate any trends.

### Spontaneous or Induced labour

Five of ten had labour induction with PGE1 (all of them at a dose of 50 microgm, 4 intravaginally and one sublingually). Two others had PGE2 for the same purpose. One patient received oxytocin stimulation after initial use of PGE2. Thus 8 of the ten patients had oxytocic agent of one type or another.

The dose of vaginal PG of 50 micro gm is more than what is recommended.

Since the denominator, that is, the number of obstetric patients who received PGE1 for induction, is not known, it is not possible to comment on the incidence of this complication among users of PGE1. Nor is it possible to comment whether the higher dose of 50 microgm has been responsible. However prudence demands a few general guidelines regarding induction.

1. The dose of PGE1 should be minimum. Higher dose is required while trying to induce labour with firm long cervix. Prior ripening with mechanical methods like Foleys catheter and extra amniotic saline instillation may help to reduce the dose of PGE1. Once the cervix has become fully 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 tendency for hyperstimulation, it can be immediately arrested by stopping the oxytocin drip.
2. Four of the patients had smooth muscle relaxant agents like valethamide and drotaverin used in combination with prostaglandins (two with PGE2 and two with PGE1). The use of these agents was presumably for enhancing cervical dilatation. These drugs were started after the cervix was effaced and at least 4cm dilated. Traditional clinical observation is that once this stage is reached cervical dilatation progresses smoothly and rapidly unless there is disproportion. Addition of these smooth muscle relaxants (which are vasodilators as well) seem to be not only unnecessary but possibly very harmful. There are two possible consequences we are concerned about:
  1. The ease with which amniotic fluid can enter into circulation through these dilated vessels, should a tear occur in the vagina or cervix.
  2. The risk of excess bleeding from cervix or vagina.

We feel that the higher incidence of AFE atleast to some extent, was the result of this practice. Clinical features like status of membranes and mode of delivery are noted in table 8.2

**Clinical presentation**

Amniotic Fluid Embolism classically presents as the sudden onset of dyspnoea and hypotension followed shortly by cardio respiratory arrest and coagulation failure if she survives the initial hypoxic event. They usually develop any of the premonitory signs and symptoms like respiratory difficulty, cyanosis, chest pain, convulsion, abnormal behaviour, hypotension etc. A sudden change in the patient’s behaviour may be an early feature of the onset of hypoxia and a toxic confusional state.

In the 10 cases we have assigned to AFE, the following symptoms or signs were observed.

Table 8.3

Convulsions	8
Chest discomfort / dyspnoea	3
Cyanosis	4
DIC	4

**Diagnosis**

Ideally diagnosis should be on the basis of clinical findings and confirmed by demonstration of fetal squames and lanugo hair in the pulmonary vasculature. This is possible at 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 justification to the argument that the syndrome of amniotic fluid embolism is not just due to the mechanical effect of blockage of pulmonary vasculature by the fetal elements but more importantly due to anaphylactic reaction to the fluid or fetal elements. This is the justification for naming this condition an “anaphylactoid reaction of pregnancy”. The argument, therefore, is that this condition should be managed like other anaphylactic reactions, e.g. anaphylaxis to penicillin. In most of the cases only a clinical diagnosis may be all that is possible until new laboratory tests become available. Already there are suggestions that measurement of complement activated AFE or the fetal antigen Sialyl Tn may help to diagnose the condition. Until then the cluster of symptoms that should be looked for are



1. Acute chest discomfort with dyspnoea, cyanosis or respiratory arrest
2. Acute hypotension or cardiac arrest
3. Coagulopathy (with lab evidence or on clinical features of DIC)

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

## Learning from example

### Case 1

This patient G2P1L1 with previous delivery 10 years earlier was induced one day after EDD with PGE1 50 microgm at 5 am. Second dose of 50 microgm at 9 am. She had two more doses of 25 microgm each at 2.30 and 6.30pm. Had drotaverin 1 amp IV at 7pm. Had tumultuous contractions and later the contractions became feeble. At 8.30pm vacuum delivery tried but failed due to excess caput. With forceps, delivery was accomplished. Baby alive ,3.6kg, cried after 2 minutes. Soon after delivery patient became restless , cyanosed, hypotensive and developed cardiac arrest. She was referred to a higher centre where laparotomy was done. At the primary hospital itself rupture, haematoma etc were ruled out. Patient died at the higher centre by 12.15am (about 3 hours later). No autopsy was done.

### Learning Points

Multiple and high doses of misoprostol and one dose of intravenous drotaverin were used in a second gravida. The last dose of PGE1 and the drotaverin when the cervix was thin would have contributed to the tumultuous contractions and subsequently the possible AFE.

### Case 2

2<sup>nd</sup> gravida was admitted 4 days before her EDD. PGE2 induction at 8.10 pm. Progressed in labour. At 10.45 , 3 finger dilatation, ARM, valethamate; at 11am pitocin drip started. 11.45 am strong contractions. Thereafter respiratory distress, cyanosis. Intubated and ventilated. Pt improved but pulled out tube herself. Oxygen given with face mask. Delivered with forceps. Transferred to higher centre where she died shortly.

### Learning Points

- Starting elective induction in the evening with prostaglandins is not advisable.

- After ARM , pitocin should be delayed.
- With 3 finger dilated cervix, the use of valethamate is not desirable.
- After the patient initially improved with intubation and ventilation, assisted ventilation should have been continued.

### Case 3

This G3P3 underwent caesarean section in a peripheral hospital for fetal distress. During surgery they noted abnormal bleeding from the wound and other puncture sites. Blood was not clotting. Hence she was referred to a higher centre where she reached in shock. In spite of resuscitative measures she succumbed within a few hours.

### Learning Points

We do not have enough details to clinch a diagnosis but development of coagulation failure without any apparent cause was what made us put this case under AFE. It is possible for AFE to develop during caesarean section and other symptoms like respiratory distress, convulsions etc. will not be apparent.

#### Key Learning points

- AFE is not a rare entity.
- It is usually preceded by premonitory signs and symptoms like respiratory distress, cyanosis, restlessness, altered behavior, and convulsions before collapse and haemorrhage occurs.
- Woman with symptoms resembling AFE should be transferred to an intensive care unit at the earliest.
- Multidisciplinary management is mandatory.
- Management is mainly supportive, maintaining adequate oxygenation, correcting cardiovascular collapse, volume replacement with isotonic solutions and inotropic agents.
- Prompt and appropriate management of DIC and PPH is essential
- Once collapse sets in, try to deliver the patient at the earliest. Even perimortem caesarean section is advised within 5 minutes of cardiac arrest.



## Chapter 9

# Anaesthetic Causes

**Dr.A.K.Unnikrishnan**  
On behalf of the editorial board

### Anaesthetic 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 anaesthesiologist 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 anaesthetic 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 oesophageal 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 hypoxaemia easily and thus compromises both the mother and the fetus.
- The potential for adverse effect on the fetus when anaesthetic 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 specialised attention from the anaesthesiologist.

### **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:

- Whether anaesthesia will result in congenital defects in the fetus, and
- 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 anaesthetic 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 anaesthetics 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 anaesthetics and surgery during pregnancy, especially during the period of organogenesis. With the current state of knowledge, it can be categorically stated that no anaesthetic or drug routinely used during anaesthesia can be implicated to be teratogenic.

### **Abortion & Preterm Labour:**

An increased incidence of abortion and preterm labour has been documented when surgery is performed in pregnant patients. There is no evidence that any particular type of anaesthetic technique or drug influences the risk for these com-

plications. Second trimester procedures and those not involving manipulation of uterus carry minimum risk.

### **Maternal safety:**

Surgery and anaesthesia in pregnant patients mandates extreme caution to avoid acid aspiration syndrome (6 weeks onwards) and aorto-caval occlusion syndrome (from 20 weeks). The possibility of deep vein thromboses (DVT) and pulmonary thromboembolism in the post-operative period should be kept in mind.

### **Regional Anaesthesia In Obstetrics**

Regional anaesthetic techniques have become very popular and are widely used in obstetric practices all over the world. Spinal (subarachnoid block), epidural, and Combined Spinal Epidural (CSE) are the techniques used in obstetric patients. The favourable effects of regional techniques on the placenta and on the fetus make them the techniques of choice in most obstetric situations. In contrast, general anaesthesia necessitates polypharmacy and is associated with relatively poor fetal outcome. However regional anaesthesia can cause serious complications, some of them peculiar to the obstetric population.

### **Aorto-caval occlusion syndrome:**

Regional anaesthesia produces peripheral vasodilatation due to sympathetic block. This results in reduced venous return which in turn reduces cardiac output. This coupled with pressure of the gravid uterus on the aorta and inferior vena cava results in an alarming fall in blood pressure and even cardiac arrest. These complications can develop dramatically within minutes of instituting a spinal anaesthetic. Close monitoring and anticipation, uterine displacement with a wedge, preloading, and prompt management of hypotension with ephedrine 10-15 mg, should avert the crisis.

### **Post-Dural Puncture Headache (PDPH):**

This is a peculiar type of headache resulting from dural puncture and the incidence of PDPH is higher in obstetrical patients than in others. The reason for the headache is low CSF pressure caused by leakage through the dural puncture site. This is generally a difficult problem to treat and hence prevention by using very fine needles and employing good technique is to be adopted. Once PDPH occurs, it is treated with absolute bed rest, good oral hydration, and the use of simple analgesics.

### **General Anaesthesia For Obstetrics:**

At present, the use of general anaesthesia in obstetric practice is limited because of the less than excellent outcomes for both the mother and the fetus. However

there are situations where general anaesthesia becomes necessary. The common indications for general anaesthesia in obstetrics include:

- Caesarean for fetal distress where there is no time to employ regionals
- Peripartum haemorrhage – Regionals are contraindicated here because of intravascular volume deficit
- Regionals contraindicated because of coagulation defects, local infection, etc.
- Situations where a relaxed uterus is required.eg: CS for transverse lie to facilitate intraoperative version.

General anaesthesia in obstetrics is associated with certain complications:

**Failed Intubation:** Laryngoscopy and intubation are relatively difficult in obstetric patients because of large breasts, airway oedema, and obesity. Delay in intubation can lead to severe hypoxaemia and compromise fetal safety. Vomiting and aspiration can occur after repeated attempts at intubation.

**Acid Aspiration Syndrome (Mendelson's Syndrome):** Obstetric patients are especially prone to this complication because of factors like increased intragastric pressure, incompetent lower esophageal sphincter, and increased acidity of gastric contents. Commonly employed prophylactic measures include –

- Routine use of H<sub>2</sub> receptor blockers and antacids
- Rapid sequence induction to limit the time from loss of consciousness to sealing of the airway
- Employing cricoid pressure (Sellick's maneuver) to prevent regurgitation of stomach contents

**Awareness during general anaesthesia:** This is a bizarre situation where patients remain awake but paralysed during general anaesthesia. This is a consequence of employing a very light plane of anaesthesia during a caesarean section to avoid fetal depression and uterine hypotonia. This problem is circumvented by using volatile anaesthetic, either halothane 0.5% or Isoflurane 1.0% in 50% O<sub>2</sub> & N<sub>2</sub>O till the baby is delivered and then using I/V narcotics.

## Obstetric Emergencies and Anaesthesia

Most obstetric emergencies fall into two broad categories: those that potentially compromise the fetus, and those causing maternal haemorrhage (pre-, intra-or post-partum).

Fetal distress or non-reassuring FHR is frequently an indication for emergency operative intervention. Such situations will necessitate recourse to general anaesthesia unless an epidural catheter is already in situ introduced earlier for labour

analgesia, in which case extension of epidural anaesthesia can be considered. Haemorrhagic emergencies almost always demand general anaesthesia. Regionals are associated with sympathetic block and consequent hypotension which will be disastrous in a hypovolaemic patient. Acid aspiration prophylaxis and rapid sequence induction are mandatory in this situation. Large I/V access (16G cannula) at (at least) two sites must be established for rapid volume repletion. Ruptured uterus, ruptured ectopic pregnancy, massive peripartum haemorrhage are situations where timely intervention is necessary to save lives. Resuscitation and definitive treatment (arrest of bleeding) should go side by side. It would be foolish to wait for establishing cardiovascular stability prior to operative intervention when the patient is actively bleeding inside.

### **Anaesthetic Management in Pre Eclampsia:**

Pre-eclampsia is a pregnancy-specific syndrome of poorly understood etiology. Eclampsia is the most dramatic manifestation of the severe pre-eclamptic state. Pre-eclampsia is associated with increased maternal and fetal morbidity and mortality.

Recognition of the pathophysiology allows the anaesthesiologist to make rational choices of anaesthetic techniques and drugs. Polypharmacy involved in the management of the condition and the involvement of all major systems poses real challenges. Anaesthesiologists can offer assistance in the management by providing labour analgesia, optimization of cardiovascular and respiratory function, control of fluid balance, and control of convulsions when they occur.

There is overwhelming evidence for the safety and utility of regional anaesthesia in these patients. They have a contracted intravascular volume and particular attention should be given to expansion of I/V volume before any regionals are employed. Epidural analgesia for labour helps in avoiding wide fluctuations in blood pressure in response to pain. Coagulation profile should be checked before instituting epidurals. These patients should ideally be managed in a critical care environment with extensive clinical monitoring for good maternal and fetal outcome.

### **Labour Analgesia**

There is no doubt that labour results in severe pain for most women. Good clinical practice dictates that the doctor has a responsibility to relieve pain whenever and wherever it is encountered. Different methods have been adopted over the centuries for this purpose.

The ideal labour analgesic technique should dramatically relieve the pain of labour safely, while allowing the mother to participate in the process. It should have minimum effect on the fetus and the progress of labour. The currently employed methods are continuous epidural, combined spinal epidural (CSE), spintal opioids, and patient controlled epidural. These are highly effective and relatively safe.



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.

## MATERNAL MORTALITY – ANAESTHESIA RELATED DEATHS

### Case Reviews:

Anaesthesia is an important cause of morbidity and mortality in pregnant women. As far as causes of maternal mortality are concerned, anaesthesia will rank behind haemorrhage, PIH, embolism, infection & cardiomyopathy. A disturbing factor noted is that, in spite of the great strides made in the field of modern Anaesthesiology, the incidence of anaesthesia related deaths amongst pregnant patients have not shown an appreciable decline.

In the year 2004, out of 79 maternal deaths reported to CRMD, 4 cases were directly or indirectly attributable to anaesthesia. These 4 cases are reviewed here. It is gratifying to note that there were no cases attributed to anesthesia in 2005.

### Case I:

29 year old 3<sup>rd</sup> gravida with 2 previous abortions had cervical encrclage at 12 weeks. She had mild hypertension for which she was on methyl dopa. At 20 weeks she had bleeding P.V for 3 days followed by abortion. On suspicion of incomplete evacuation she was posted for manual removal of

placenta. Her BP at that time was normal. Patient was obese with short neck and hence the Anaesthesiologist opted for endotracheal GA. After aspiration prophylaxis she was induced with propofol & intubated with cuffed endotracheal tube. On induction there was a fall in SpO<sub>2</sub> which improved after sometime. The surgery lasted for 30 mts. At recovery her respiration was inadequate and she developed acute pulmonary oedema. Immediate ventilation with 100% Oxygen, diuretics etc failed to improve her condition and she progressed to severe hypoxaemia, hypotension & cardiac arrest. She was resuscitated with external cardiac massage / adrenaline / debrillation. She was shifted to ICU once spontaneous sinus rhythm returned but remained hypotensive & died 6 hrs later.

This would appear to be a case of Mendelsson's syndrome. Aspiration prophylaxis regime is not 100% protection against aspiration. The reason noted by the Anaesthesiologist for opting for GA (obesity & short neck, both of which can lead to difficult laryngoscopy & intubation) is strange. A regional would have been a better choice in this case. The anaesthetic record in this case was very sketchy- no serial reading of SpO<sub>2</sub>, no time of cardiac arrest, no time of return of spontaneous cardiac activity, no notes by Anaesthesiologist after the patient was shifted to ICU. The overall impression one would get is that the overall care was suboptimal.

#### Case II:

37 year old woman G2P1L1, previous LSCS, Pre eclampsia in previous & present pregnancy. Present pregnancy is twins. Hypertension controlled with medication. Posted for elective caesarean. Patient refused regional and hence GA was administered. No aspiration prophylaxis given. Balanced endotracheal anaesthesia was used. Intraoperative period uneventful. After extubation patient became dyspnoeic, reintubated. O<sub>2</sub> saturation decreased to 80% and acute pulmonary oedema was noted. IPPR with 100% O<sub>2</sub> and diuretics were administered. Within 20 mts patient was shifted to a higher centre 27 Km away where she was put on ventilator but died 2 hours later.

Acid aspiration syndrome and AFE are probable causes of the course of events which ultimately resulted in death. Failure to use aspiration prophylaxis would have contributed to the death.

The decision to shift this patient to a place 27 kms away probably was not a wise one. The basic management of pulmonary oedema (effective IPPR with 100% O<sub>2</sub>, diuretics, morphia) could have been done in the first hospital itself. During the 45 mts it took to reach the higher centre the patient was not optimally ventilated. The records provided are rather haphazard. Finally, the failure of anaesthesiologist to accompany the patient during the shifting would reveal lack of concern for his patient.

**Case: III**

25 year old G2P1 previous LSCS. LCB 1 ¼ years. She was posted for elective LSCS with Sterilisation under spinal. Delivered a 3 Kg baby, Apgar 10 at 1& 5mts. After 5mts fall in B.P followed by cardiac arrest. CPR instituted which resulted in spontaneous cardiac activity after 10 – 15 mts. Surgery completed and when spontaneous ventilation returned extubated. Patient failed to regain consciousness- hence, reintubated and ventilated. 8 hours later she was shifted to a higher centre which took 2 hours. At arrival in the second hospital patient found to be in cardiac arrest. Resuscitated with ECC + Defibrillation. She remained comatose in spite of all efforts and died 2 days later.

The details, given in the records are very sketchy but a number of points are worth commenting on. There is no record of preanaesthetic evaluation. The anaesthesia record is very haphazard and would give an impression of a very casual attitude and suboptimal management. Inj.Bupivacaine Heavy 10 mg administered spinally is the normal dose for LSCS. But this dose given to a short person or injected during uterine contraction can result in dangerously high block. The treatment of hypotension would seem to be incorrect. No mention of I.V. fluids/ vasopressors is seen in the record. The 15 mts gap needed to get a spontaneous circulation is enough to produce irrecoverable brain damage. It is quite unfortunate that the implication of spinal anaesthesia is poorly understood which result in such tragedies.

**Case IV :**

30 yrs old patient G3P2, 2 previous LSCS done under G/A. Antenatal period uneventful. Preanaesthetic evaluation revealed no abnormality. ASA grade I. Aspiration prophylaxis followed by GA with endotracheal balanced anaesthesia was administered. Intraoperative period was uneventful. After extubation patient was found to be desaturating, hence reintubated. She was hypoxic ( $\text{SpO}_2 < 90\%$ ) and 20 mts later developed acute pulmonary oedema and severe hypotension. Diuretics/ Inotropes and IPPR resulted in marginal improvement in  $\text{SpO}_2$  & B.P. After 2 ½ hrs patient was shifted to another hospital and 24 hours later she expired. The clinical course of patient in the second hospital is not known.

AFE, Mendelssons or Cardiomyopathy could have been the problems in this patient. The anaesthetic records do not show the  $\text{SpO}_2$  in the intra-operative period, so we do not know whether the problem had started during the surgery itself. Again, as in the previous cases, the hurry shown in shifting patient to another hospital is unhealthy and would definitely affect the chance of ultimate patient survival.

## OBSERVATIONS AND RECOMMENDATIONS:

The following comments and recommendations are based on the 4 cases reviewed and also on other cases of maternal death due to different causes.

1. Obstetric surgical patient is different from other surgical patients. Because of altered physiology associated with pregnancy, she is at increased risk for serious complications like aspiration, haemorrhage, embolism, aorto-caval occlusion etc. As such she demands special care from the anaesthesiologist. Obstetric anaesthesia should be handled by experienced anaesthesiologist and not simply relegated to trainees or junior staff.
2. Proper preoperative evaluation must be done in each and every case. For this, the anaesthesiologist must be involved sufficiently early, especially when problems already exist or are anticipated. Good and timely interdepartmental communication is the key to safe management.
3. Acid aspiration prophylaxis must be a routine in all obstetric surgical patients – even when the patient has been fasting and on IV fluids or when the anaesthetic planned is regional.
4. Regional anaesthesia – spinal and epidural blocks – are serious business in obstetric patients. Unfortunately, many anaesthesiologists and most obstetricians do not view them seriously. Regional anaesthesia can produce an almost total body chemical sympathectomy leading to severe hypotension and bradycardia. These coupled with a possible aortocaval occlusion can turn into an acute circulatory arrest. These changes can occur suddenly and without warning. Constant vigilance and timely action only can save the patient.
5. The modern electronic patient monitors (for E.C.G, NIBP, SPO<sub>2</sub>, EtCO<sub>2</sub> etc) are all a great help in patient management. But, unfortunately, there is a tendency on the part of anaesthesiologist to rely too much on these gadgets and move away from the head end of the patient. This must be stopped. It must be clearly understood that monitors only indicate patient parameters. When something goes wrong in an anaesthetized patient, we need a qualified and knowledgeable person to act promptly on the information provided by the monitor. The ultimate monitor is a knowledgeable and dedicated anaesthesiologist at the head end of the patient!.
6. In many hospitals the involvement of anaesthesiologist is limited to the intraoperative period. This is very unfortunate. There are areas where his expertise can become handy, for example, management in severe haemorrhage. At this juncture it will be pertinent to mention about the utility of C.V.P. measurement. It is a well known fact that blood pressure and pulse rate are poor indicators of intravascular volume. In severe

haemorrhage, where large volumes will have to be administered, C.V.P. will be a good guide in replacement as it is a better indicator of I.V. volume than pulse rate or blood pressure. Setting up a C.V.P. line can be learned quite easily and the necessary catheter set must be available in all obstetric units.

7. It is quite unfortunate that even the threat of medicolegal problems has not made doctors (including our own colleagues) realize the importance of proper medical record keeping. Some of the case files we reviewed revealed absolutely shoddy record keeping. Only proper records will save us when something goes wrong and the case gets to the court. We must cultivate the habit of keeping proper records in each and every case we do and not write notes when something goes wrong. A proper anaesthetic record must contain details of preoperative evaluation, the time and dose of all the drugs used (anaesthetic gases, volatile agents included), record of pulse, B.P, SPO<sub>2</sub> (CVP, EtCO<sub>2</sub>, urine output when indicated) atleast every 5 mts, details of extracellular fluid loss, blood loss, I.V. fluids administered, post anaesthesia status, monitoring in recovery area etc. Use of a dedicated graphic anaesthesia record will make record keeping easier. Remember that proper, routine record keeping makes one a true professional and it can come handy in case, unfortunately, one of the patients approaches the court.
8. We came across a number of instances where undue hurry was shown in shifting a critically ill patient to another hospital. May be this is done to avoid the danger and violence from the patient's relatives if death occurs. The process of shifting a seriously compromised patient, traveling in an ambulance for a couple of hours in suboptimal conditions, will definitely affect the chances of patient survival adversely. The most common reason mentioned is the absence of a ventilator. We must understand that a patient requiring ventilatory support can very well be paralysed and ventilated manually for quite some time. If at all the patient has to be shifted, it should be done when her condition is reasonably stabilized.

## ANNEXURE – A

### ACUTE COLLAPSE IN OBSTETRICS

#### CAUSES

1. Peripartum haemorrhage (most common cause).
2. Rupture uterus/ ectopic.
3. Amniotic fluid embolism.

#### 4. Anaesthesia related – Aorto caval occlusion

Total spinal

#### ASSESSMENT:

- Keep a cool head
- Elicit brief clinical history
- Do a quick clinical examination
  - Level of consciousness
  - Pulse/ BP / Respiration
  - Look for obvious haemorrhage

#### MANAGEMENT:

##### A. Hypotension with signs of poor tissue perfusion

- Elevate legs.
- Oxygen with mask 4.0 L/ mt.
- Get I.V. access and start I.V. fluids.
- Take blood for group/ cross match/ coag. Studies.
- Steps to arrest ongoing loss.

**I.V. Access:** Large cannula (16 G), at least at 2 sites.

**I.V. Fluids:** In massive haemorrhage, the immediate cause of death is **loss of I.V. Volume.**

Hence **rapid repletion, of I.V. volume** is of prime importance.

R.L or N.S. 1.0 L in 10 mts and reassess.

Colloids 1.0 L

Blood as early as possible

#### Monitoring:

- Pulse/ B.P/ E.C.G/ SpO2 / C.V.P.
- Urine output/ level of consciousness
- Establish a central Line (Antecub fossa, subclavian or Int., jugular) at this time. For the inexperienced, the antecubital route using a CAVAFIX unit will be easy for obtaining the central vein access.
- Consider Inotropes when hypotension persists even after volume repletion & C.V.P. has come up.
- Resuscitation & definitive therapy (arrest of bleeding) to go simultaneously

## B. Acute circulatory arrest (Cardiac Arrest)

**Diagnosis:** Absence of pulsation in major vessels (carotid/ femoral)  
Apnoea/ Unconsciousness/ cyanosis/ dilated, fixed pupils.  
Not to take more than 10 secs. to elicit these signs.

**Management :** Call for help.

**Airway :** Maintain patent airway - Intubation.

**Breathing :** Ventilate with 100% oxygen ( 10 – 12 L /mt).

**Circulation :** External cardiac compression – ( 80 – 100/mt)

**Defibrillation :** 200 J – 200 J – 360 J

Learn to diagnose asystole/ ventricular fibrillation from the cardiac monitor  
familiarise yourself with defibrillator and its usage.

Inj. Adrenaline 1 mg I.V. every 3- 5 mt till a spontaneous heart rhythm is established. Hand over patient to Anaesthesiologist/ Intensivist.

## ANNEXURE B

### EMERGENCY TROLLEY

The trolley ideally should have separate drawers to keep drugs, 'I.V. fluids', I.V. sets, Laryngoscopes & E.T. tubes. Facility on the side to fix oxygen cylinders, space on top to keep monitors.

• **Drugs :**

Inj. Adrenaline	(5)	Inj Nitroglycerine	(2)
Inj. Atropine	(5)	Inj. Magnesium sulphate	(5)
Inj. Ephedrine	(5)	Inj. Sodium Bicarbonate	(5)
Inj. Dopamine	(2)	Inj. Calcium Chloride	(5)
Inj. Dobutamine	(2)	Inj. Midazolam	(2)
Inj. Lignocaine	(2)	Inj. Hydrocortisone	(2)
Inj. Normal saline	(5)	I.V. sets	(5)
Inj. Ringer lactate	(5)	Blood set	(5)
Inj. Hydroxy Ethyl Starch	(5)	I.V. cannula 16 G	(5)
		20 G	(5)

• **Endotracheal Tubes .**

24 g	(2)
7.5 mm (Adult)	- 2 Nos.
3.0 mm ( Normal)	- 2 Nos
2.5 mm (Premature)	2 Nos.

<b>Syringes :</b> 2 ml	(10)
5 ml	(10)
10 ml	(10)

- Adhesive Tape
- Laryngoscope - Adult - 1 Oxygen cylinder with flow meter  
Paediatric- 1
- Cardiac Monitor
- Pulse oximeter
- Defibrillator
- Suction/ catheters
- Typed list of items to be prominently displayed on trolley –
- Replace used item immediately
- Daily morning check of item by Nurse in charge.
- Weekly check by supervisory staff.

Monthly check of expiry dates of drugs and replace expired item .

<sup>1</sup> The values mentioned here are only approximations.





## CHAPTER 10

# Blood and Fluid Management

**Dr.A K Unnikrishnan**

**Dr.N Viswanath**

On behalf of Editorial Board

Obstetric practice is often associated with sudden massive bleeding which, when not managed properly, will compromise patient safety. Acute blood loss leads to two immediate problems:

- Loss of intravascular volume
- Loss of oxygen carrying capacity leading to tissue hypoxia

The immediate cause of death from acute blood loss, however, is loss of I/V volume leading to reduction in venous return eventually ending in a cardiac standstill. This fact should be remembered in the first line management of such cases.

I/V Access:

The speed at which I/V repletion is done in massive blood loss is critical. The single most important factor which determines the speed of I/V infusion is the size (diameter) of the I/V cannula. At least two I/V cannulae of minimum 16G should be sited in two large veins by the first doctor who sees the patient. This is important because, as time goes on, vasoconstriction will make it difficult to access veins and take blood samples for grouping, cross-matching and coagulation studies.

### **I/V Fluids:**

As blood will not be available immediately in an emergency, volume repletion should be started with crystalloids and plasma volume expanders. The cardiovascular integrity can be maintained for quite some time while waiting for blood to arrive.

### **Crystalloids (Normal saline or Ringer lactate)**

When infused, these fluids partly diffuse into the interstitial space. Only a third of infused volume is retained in the intravascular compartment. This means that to replace lost I/V volume, three times that volume of crystalloids will have to be administered.

### 5% Dextrose

5% Dextrose is distributed throughout the fluid compartments and only 1/8 of the infused volume will remain in the I/V space. As such, *5% Dextrose is inefficient for I/V volume repletion and is contraindicated for this purpose.*

### Plasma Volume Expanders (Dextran, Gelatin (Haemaccel), Hydroxy Ethyl Starch (HES))

Plasma Volume Expanders (PVEs) are colloids and hence they remain in the intravascular compartment. They are, therefore, extremely useful in acute I/V volume repletion. The main drawback of crystalloids and PVEs is that they cause haemodilution leading to reduction in oxygen carrying capacity and reduction of clotting factors which can aggravate bleeding. However, these fluids are extremely useful in sustaining life while waiting for blood to arrive.

Start with N.S. or R.L 1.0 L infuse in 10 – 20 minutes & reassess.

HES 1.0 L

Try to get blood as early as possible. Meanwhile, continue with NS or RL

### Monitoring:

Apart from routine monitoring of indicators like heart rate, BP, ECG, and Pulse Oximetry, two other indicators — Urine Output and CVP (Central Venous Pressure) — must also be monitored during acute volume repletion. Urine output is a good indicator of tissue perfusion. This being very simple to monitor, must be done routinely in all acutely bleeding patients. CVP is a better indicator of intravascular volume than heart rate or BP and CVP measurement must be an integral part of managing acute volume loss. After volume repletion has been started, a Foley's catheter should be inserted and a central line established. Placing a central line can be mastered without much difficulty and all obstetricians must master this very useful technique.

Some indicators of the adequacy of volume repletion are:

- Improving heart rate & BP
- Urine output > 1.0 ml/kg/hr
- CVP 6-8 cm H<sub>2</sub>O
- Improving level of consciousness

Finally, it has to be stressed that resuscitation and definitive therapy, in the form of arresting ongoing blood loss by arterial ligation, hysterectomy, etc., must go hand in hand.

## Use of Whole Blood and Components

### Whole Blood:

Though use of whole blood is not recommended in most advanced countries, it is often the only blood available in many settings in our state. If access to a good blood bank is available, use of components is recommended.

Banked whole blood (blood stored optimally for more than 24 hours) replaces only the oxygen carrying capacity and some colloids (albumen etc). If the patient is very anemic and is in volume overload, one could infuse only the packed cells after removing most of the plasma with or without intravenous furosemide. Fresh blood would replace coagulation factors and platelets but a lot of volume may need to be infused e.g. to correct an INR of 1.8 to 1.5, at least 5-6 units of fresh blood may be needed. One unit of fresh whole blood may raise platelets by less than  $5000/\text{mm}^3$  when infused. When one is compelled to use fresh whole blood, use it as soon as it is bled, as some coagulation factors have half-lives of only a few hours.

### Packed Red Blood Cells (pRBC):

Red blood cells will need replacements if oxygen carrying capacity is low after correcting hypovolemia by crystalloids (and, if needed, colloids). However in the acute setting if there has been loss of about 30-40% of blood volume, red cell transfusion would be required. If the person is anemic to start with, even a smaller loss would need correction. When acute loss has occurred over-reliance on haemoglobin or packed cell volume values should be avoided as changes in these values would be noticeable only after fluid shifts have taken place or after volume replacement. One unit of whole blood or pRBC would, in general, raise haemoglobin by 1g/dl or packed cell volume by 3%. In an actively bleeding patient, the aim would be to maintain a haemoglobin of over 9-10g/dl. Greater reliance is to be placed on the total assessment of circulatory state and tissue perfusion than on individual values.

### Fresh Frozen Plasma (FFP):

Fresh frozen plasma contains all the coagulation factors and other proteins present in the original unit of blood, slightly diluted by the citrate containing anticoagulant solution. Plasma frozen at  $-18^{\circ}\text{C}$  or colder within 6 hours of donation can be stored for up to one year before use when it needs to be thawed for 20-30 minutes. Once removed from  $-18^{\circ}\text{C}$ , it should be used as early as possible and no later than 24 hours.

### Dose and indications

In general haemostasis can be achieved when activity of coagulation factors is at

least 25-30% of normal, in the absence of inhibitors such as heparin, and when the level of fibrinogen is at least 75-100mg/dl. As the plasma volume is approximately 40ml/kg, this requires a dose of at least 15ml/kg, which may have to be repeated in critically ill patients or in those with massive bleeding. The adequacy can be judged by the clinical response as well as by shortening of the coagulation times.

### Side effects

- Transmission of disease
- Unexpected red cell antibodies may cause hemolysis
- Infusion of large amounts may cause pulmonary edema, especially in those with preexisting cardiac disease. This can be reduced by infusion at a rate at or below 1 ml/kg/hour, or by using cryoprecipitate in suitable cases.
- Fever, chills, and allergic reactions
- Anaphylaxis

### Platelets:

They are indicated prophylactically when platelets are less than 20,000/mm<sup>3</sup> and definitely below 10,000/mm<sup>3</sup>. They should be given to patients with platelets less than 50,000/mm<sup>3</sup>, if there is a bleeding tendency or if an invasive procedure is being planned. Prophylactic platelet transfusion is ineffective when the thrombocytopenia is caused by increased platelet destruction. There is no evidence to support prophylactic platelet transfusion along with massive blood transfusion (defined as the replacement of the blood volume in 24 hours).

Usually a single unit of platelets contains — check with your blood bank —  $5.5 \times 10^{10}$  platelets in 50-100 ml of plasma. On average, one unit raises the count by 5000/mm<sup>3</sup>. In adults, the usual dose may range from 6-10 units.

### Care Advised With Massive Transfusion

Massive transfusion — defined as the replacement by transfusion of more than 50% of a patient's blood volume in 12 to 24 hours — may be associated with a variety of haemostatic and metabolic complications. The issues include volume, coagulation, ionized calcium, potassium and acid-base status.

Some experts do not agree on all that is suggested below. However, the following has a general consensus that could form a guideline:

- Patients with severe bleed may go in for DIC. Even when there is no DIC replacing with packed cells and crystalloids lead to dilution of clotting factors. PT and PTT should be monitored. Two units of FFP may be given if the

values of PT and PTT are 1.5 times control. (Dose may vary according to the needs.)

- A similar dilutional effect is seen with platelets. 10 to 12 units of packed cells can reduce platelets by 50%. Monitor platelets, and if platelets fall below  $50,000/\text{mm}^3$ , in the setting of ongoing bleed, platelet transfusions may be used. Each unit of platelets may raise the platelet count by about  $5000/\text{mm}^3$ .
- Since blood is anticoagulated with sodium citrate and citric acid, metabolic alkalosis and decline in free calcium are expected complications. In the setting of renal and/or hepatic function impairment, citrate toxicity could be worse and acid base status, potassium and calcium levels may need close monitoring. Monitoring for clinical signs of low calcium and ECG are essential in addition to regular blood measurements.
- Rapid transfusion of multiple units of chilled blood can reduce core temperature rapidly and precipitate cardiac dysrhythmias. Take care to warm blood and even fluids to at least room temperatures when large volumes are infused.
- Stored blood could raise plasma potassium. This needs watching.

All the above need even greater care in the setting of hepatic and renal disease.

## Learning Points

To sum up managing acute blood loss should involve careful assessment, close monitoring of clinical and lab parameters, prompt and proper use of volume expanders, blood and its components, and surgical intervention if and when needed.



**Why Mothers Die**  
**Kerala - 2004-05**

Part-Four

**Indirect / infrequent  
causes and solutions**

**4**





## *Chapter 11*

# Heart Disease in Pregnancy

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On behalf of Editorial Board

### **Cardiac disease: Key summary points**

- Heart disease is the most common non obstetric cause of maternal death after hypertension.
- Peripartum cardiomyopathy is increasingly being recognized.
- Despite under reporting in the present series, Rheumatic heart disease especially Mitral stenosis remains the most common form of heart disease in pregnancy and is instrumental in significant maternal mortality.

### **Cardiac disease: Key recommendations**

- Women with severe cardiac disease require multidisciplinary care
- In general, heart disease tends to get worse over time. Women with any form of heart disease should be advised to have children early.
- Patients with heart disease should undergo full cardiac evaluation including ECHO prior to conception
- Pregnancy should be avoided in patients with primary pulmonary hypertension, Eisenmenger syndrome, cyanotic congenital heart disease and any patient with severe cardiac disability.

### **Summary**

When data for years 2004 and 2005 were analyzed together, it was noted that there were 29 deaths from heart disease in pregnancy. This forms 9 % of all maternal deaths that was reported in these two years in Kerala. Heart disease thus becomes the second most common non obstetric cause of maternal deaths. Of the 29 deaths reported, only 15 cases were reported to CRMD and other 14 cases were noted from DHS, details of the latter being not available. Hence it is not

possible to find out the distribution of different kinds of heart disease observed in maternal deaths. There were 3 cases of Rheumatic heart disease and 2 cases of primary pulmonary hypertension among the 15 cases of CRMD. However congenital heart disease was not seen in the maternal deaths. This is in sharp contrast to western experience where nearly 75 % of heart disease in pregnant women is congenital. Peripartum cardiomyopathy was not uncommon and of the 3 cases suspected, 2 had definite features of the disease.

It is possible that significant underreporting of heart disease has occurred in the data for year 2004 and 2005. It is generally agreed that Rheumatic heart disease especially Mitral stenosis is the most important heart disease encountered in pregnancy in our state. With better socio economic status and greater awareness, incidence of Rheumatic fever has come down dramatically in Kerala over past decade. Yet, impact of this decline in Rheumatic fever is only slowly being felt and still there are large number of pregnant patients with Rheumatic heart disease coming for antenatal care. Better treatment modalities and availability of percutaneous transmitral commissurotomy (PTMC) is changing the outlook of these patients.

### Key summary points

- Heart disease is a common non obstetric cause of maternal mortality in Kerala
- Rheumatic valvular disease, primary pulmonary hypertension and peripartum cardiomyopathy appear to be the dominant cardiac causes of deaths during pregnancy.
- With the availability of PTMC and other treatment modalities, mortality due to rheumatic heart disease especially Mitral stenosis may come down in the near future.

### Pulmonary Hypertension:

**Primary Pulmonary Hypertension:** Pregnancy is associated with a high maternal mortality. Recent review of literature reveals a maternal mortality of 30-40%. Clinical deterioration during pregnancy or death cannot be predicted on the basis of patient's pre conceptual clinical status. Symptomatic deterioration usually occurs during second trimester. Death has occurred in a few hours to several days postpartum usually due to sudden collapse or progressive right ventricular failure.

Pregnancy should be avoided in these patients and tubal ligation recommended. Early abortion is indicated in patients with Primary pulmonary hypertension who become pregnant. If the patient elects to continue pregnancy, physical exertion should be restricted to reduce the circulatory load. Because of the beneficial effect of anticoagulation in patients with Primary pulmonary hypertension and the in-

creased incidence of thromboembolism during pregnancy, such therapy is recommended throughout gestation or at least during 3<sup>rd</sup> trimester and early post partum period.

Because of the high rate of early postpartum maternal death, close monitoring is recommended for several days post partum.

**Eisenmenger's Syndrome:** Recent analysis has shown a maternal mortality of 40-45% in this condition. Cause of maternal death is, as in case of Primary pulmonary hypertension, unclear; usually occurs in the first few days after delivery and is preceded by desaturation and hemodynamic deterioration. Right ventricular ischemia and failure, cardiac arrhythmias and pulmonary embolism are likely mechanisms.

### **Congenital Heart Disease:**

Because of increased survival in children with congenital heart disease, pregnancy has become more common in this patient population. Many of these patients have undergone corrective cardiac surgeries but nevertheless pose special problems in pregnancy.

### **Key Points:**

- Pre-conception management should include complete cardiac evaluation with special emphasis on functional status and pulmonary hypertension.
- In general good maternal outcome is expected in non cyanotic congenital heart disease.
- Maternal outcome is determined by nature of the disease, surgical repair, pulmonary vascular disease, functional capacity, myocardial dysfunction, left ventricular obstruction and history of arrhythmias.
- Unfavorable outcome is seen in patients with congestive heart failure, cyanosis, hypertension, arrhythmias and those with impaired functional capacity
- Elective induction of labour / caesarean may be considered in high risk patients, if fetal maturity confirmed.
- Oxygen to be given to hypoxemic mothers
- Hemodynamic and blood gas monitoring for high risk patients.
- Blood volume loss must be anticipated and treated promptly.

### **Surgically corrected congenital heart disease**

Most congenital cardiac defects are amenable to surgical corrections and more

such women are likely to become pregnant. These patients require special care. Their evaluation should include accurate measurement of PA pressure serially, assessment of ventricular function, functional status, measurement of arterial O<sub>2</sub> saturation and ruling out arrhythmias. Even asymptomatic patients who had surgically corrected ASD, VSD or PDA may have elevation of PA pressure. Even mild to moderate PAH will increase risk of sudden death especially in the immediate post partum period.

### **Valvular heart disease:**

Valvular heart disease in young women is most commonly due to rheumatic heart disease, congenital abnormalities, or previous endocarditis and may increase the maternal and fetal risks associated with pregnancy. The likelihood of adverse outcome is related to the type and severity of maternal valvular disease and the resulting abnormalities of functional capacity, left ventricular function, and pulmonary pressure. Complications are more likely among women with reduced left ventricular function (Ejection fraction less than 40%), Left heart obstruction (Aortic stenosis and Mitral stenosis with valve areas less than 1.5 cm<sup>2</sup>, previous cardiovascular events (heart failure, transient ischemic attack, or stroke), NYHA functional class 2 or higher and pulmonary hypertension.

Rheumatic mitral stenosis is the most common clinically significant valvular abnormality in pregnant women and may be associated with pulmonary congestion, edema and atrial arrhythmias. Adverse maternal outcomes are more common if valve area is less than 1.5 cm square and if abnormal functional capacity is present before pregnancy. Patients with severe symptoms or tight mitral stenosis (valve area of less than 1.0 cm<sup>2</sup>) should be considered for percutaneous balloon mitral valvuloplasty.

Mitral regurgitation is usually well tolerated during pregnancy. Symptomatic women with severe MR should undergo MV repair or replacement before conception.

Aortic stenosis, when severe, is poorly tolerated during pregnancy. Symptomatic patients or those with peak outflow gradient of more than 50 mm Hg are advised to delay conception until after surgical correction. Termination of pregnancy is strongly considered if the patient is symptomatic before the end of the first trimester.

Aortic regurgitation: Reduced systemic vascular resistance of pregnancy reduces the volume of regurgitant blood and hence may be tolerated. ACE inhibitors should be discontinued. If marfan's syndrome is present great care is needed.

## Key points

- Careful clinical assessment and echocardiography warranted before conception
- Abnormal functional capacity, left ventricular dysfunction, significant valve obstruction, history of heart failure or embolic events or pulmonary hypertension, all should alert to the possibility of adverse maternal outcome.
- Should be counselled regarding the risk
- Should be seen by cardiologist once each trimester and more often if complications ensue.

## Cardiomyopathy and Myocarditis

### Peripartum Cardiomyopathy:

Peripartum Cardiomyopathy is a dilated cardiomyopathy of uncertain etiology that is defined as:

1. Development of cardiac failure in the last month of pregnancy or within 5 months after delivery.
2. Absence of demonstrable cause for the cardiac failure
3. Absence of demonstrable heart disease before the last month of pregnancy
4. Documented systolic dysfunction

Peripartum Cardiomyopathy is more common in multiparous women. It has been reported more often in twin gestations and in women with pre-eclampsia. The exact cause is unknown – myocarditis, auto antibodies and low selenium levels are implicated. Because there is no specific test for diagnosis of Peripartum Cardiomyopathy, it is established by exclusion of other causes of left ventricular dilatation and systolic dysfunction.

Common symptoms and signs are shortness of breath, fatigue, chest pain, palpitations, weight gain, peripheral edema, peripheral or pulmonary embolization and arrhythmia. Physical examination often reveals an enlarged heart, S3 and murmurs of mitral and tricuspid regurgitation. ECG may show tachycardia, ST, T wave changes, conduction anomalies and arrhythmia. Chest x-ray usually shows cardiomegaly and pulmonary venous congestion. Doppler echocardiography, the most important investigation shows enlargement of all 4 cardiac chambers and depression of left ventricular systolic function. Clinical presentation and hemodynamic changes are indistinguishable from those found in other forms of dilated cardiomyopathy.

## Key Points

- Mortality rate range from 7 - 56% and is directly related to recovery of ejection fraction.
- 30% of patients return to baseline ventricular function within 6 months and 50% of patients have significant improvement in symptoms and ventricular function.
- Future pregnancy is not recommended in women with persistent ventricular dysfunction.
- Prior to subsequent pregnancy, women should undergo ECHO and if findings are normal, Dobutamin stress ECHO should be performed to know the contractile reserve.
- Patients with normal findings upon ECHO but decreased contractile reserve should be warned that they might not tolerate hemodynamic stress of pregnancy.
- Patient with full recovery should be told that while a small chance of recurrence exists, the mortality rate is low and majority of such women have normal pregnancy.

## Hypertrophic cardiomyopathy:

The therapeutic approach to pregnant patients with hypertrophic cardiomyopathy depends on the presence of symptoms and LV outflow obstruction. In the symptomatic patients with obstructive hypertrophic cardiomyopathy, attempts should be made to avoid blood loss and use of drugs that can lead to vasodilatation or sympathetic stimulation during labour and delivery. Tocolytic agents with beta adrenergic receptor activity may aggravate Left ventricular outflow obstructions. Spinal and epidural anesthesia to be avoided

## Myocarditis:

Myocarditis may be an acute or chronic process (as in peripartum cardiomyopathy) though the incriminating evidence of myocarditis being the cause for peripartum cardiomyopathy is subtle. When myocarditis results in significant LV dysfunction the picture is similar to peripartum cardiomyopathy. As the maternal mortality rate is high, early diagnosis and prompt treatment will save many lives.

## Coronary Artery Disease:

- With increasing maternal age, importance of coronary artery disease in pregnancy is becoming more relevant.
- It is still rare among women of child bearing age and occurrence of peripartum acute myocardial infarction is anecdotal.

- Risk factor assessment is of paramount importance.
- Highest incidence is in 3<sup>rd</sup> trimester, in women older than 33 yrs of age and in multigravida.
- Acute Myocardial Infarction (AMI) occurs commonly in anterolateral wall.
- Most maternal deaths occur at the time of infarction or within 2 weeks
- Although atherosclerotic diseases seems to be the primary cause of AMI, peripartum AMI is often associated with normal coronaries and has been suggested as being due to decrease in coronary perfusion caused by spasm or in situ thrombosis or spontaneous dissection.

### Arrhythmias in pregnancy

- Multiple frequent atrial and ventricular ectopics can occur in normal pregnancy without any effect on maternal mortality
- PSVT is more common during pregnancy
- AF and flutter usually seen in patients with underlying heart diseases.
- Hemodynamic impairment can occur with tachy arrhythmias during normal pregnancy.
- Electrical cardioversion is safe
- Always rule out electrolyte disturbances, thyroid disease and arrhythmogenic effects of drugs, caffeine and smoking

### Anaemia in Pregnancy

In the two cases of deaths occurring during pregnancy reviewed in this chapter, Haemoglobin was 8.2 and 8.5 gm% respectively which may have influenced the eventual outcome of these patients. Hence anaemia should be looked for in pregnant women with cardiac symptoms and every attempt should be made to correct it.

### Learning from examples

#### Case 1:

A 35 year old fourth gravida, 28 weeks of pregnancy presented to a tertiary care center with retro sternal chest pain and heaviness of three days and breathlessness of one day duration. She had no prior history of heart disease and was apparently asymptomatic till then. Details of antenatal check up were not available. Physical examination showed pallor and evidence of congestive heart failure. Pulse was 100/mt and BP 130/90. There was no evidence of valvular heart disease. Lab investigations showed Hb 10.9 %, raised total WBC count, mildly elevated renal parameters, , and normal LFT.



ECG showed T changes in anterior leads. Cardiologist confirmed initial clinical impression of congestive heart failure and ordered ECHO which showed global hypokinesia and severe left ventricular dysfunction. A diagnosis of peripartum cardiomyopathy was made. She had bleeding PV and was thought to have Abruptio placentae. Patient was aggressively managed in the ICU with nitroglycerine, Dobutamine and frusemide infusions, but failed to respond and died after 17 hours of admission.

There are many atypical features for the diagnosis of peripartum cardiomyopathy in this case. Presentation at 28 weeks of gestation is unusual. In fact the standard teaching is that PPCM is rarely seen before 36 weeks. This time of onset is in contrast to patients with underlying cardiac disease (e.g., ischemic, valvular, or myopathic) who tend to develop symptoms of heart failure during the second trimester of gestation, coincident with the greatest hemodynamic burden imposed by the gravid state. Very short duration of the illness and patient succumbing to the disease within 4 days of onset of symptoms, point to an unusual malignant course which is more common with myocarditis or ischaemic cardiomyopathy.

### Learning points:

- Peripartum cardiomyopathy or myocarditis should be considered in a pregnant patient presenting with dyspnoea and congestive heart failure
- ECHO examination is a must in patients presenting with increasing breathlessness
- A team work involving multiple specialists is needed to tackle such problems.

It is difficult to fault management strategy in this unfortunate patient. Reasonable investigations were done in the shortest available time which is commendable. In retrospect nothing more could have been done except that in such desperate situations one can consider cardiac catheterization and coronary angiography so that a remediable lesion if found can be corrected.

### Case 2:

A 22-year-old second gravida in 30 weeks of pregnancy was admitted in a secondary level hospital with complaints of palpitation and pedal edema. Details of CVS examination and ECG are not available. She was seen by a cardiologist. USG done 2 days after admission reported IUD. Next day an ECHO was performed which showed normal LV dimensions, Ejection fraction was 52%, and there was no MVP though mild MR was noted. PSVT was observed during ECHO. She was diagnosed as PSVT and treated with adenosine injection 6 and 12 mg and oral verapamil. Same day evening patient was referred to a tertiary care hospital with a diagnosis of refractory supraventricular tachycardia. On arrival, she was tachypnoeic with BP 60 mmHg systolic, pulse irregular at 156 per minute and had facial and pedal

edema. Chest was clear. CVS findings noted were irregular with loud heart sounds and no murmur. She was treated with repeated DC shocks. Rhythm transiently changed to AF but did not convert to sinus rhythm despite attempts after IV amiodarone. She died within 3 hours of admission.

### Learning points:

- Tachyarrhythmias in pregnancy could be life threatening and should be managed in centers with facilities for all cardiac interventions.
- Thyroid disorders and electrolyte abnormalities should be ruled out.
- ECHO is valuable in ruling out structural heart disease.

Supraventricular tachycardia can occur more frequently during pregnancy and can cause heart failure or hypotension even if the heart is structurally normal. However refractory PSVT is extremely unusual. Thyroid disorders and electrolyte abnormalities should have been ruled out. If elective cardio version had been carried out at the secondary level hospital, this unfortunate young woman could have been saved. Myocarditis however was still a possibility.

### Case 3:

A 23 year old primigravida in 37 weeks of pregnancy was admitted with class III effort dyspnoea. She was diagnosed to have RHD, severe mitral stenosis, and severe pulmonary artery hypertension before conception and was advised balloon mitral valvotomy. She had two antenatal visits and was on oral penicillin prophylaxis. At 34 weeks of pregnancy she was admitted with respiratory tract infection and treated with frusemide, verapamil and antibiotics. She did not continue verapamil or diuretics. After admission she was treated with diuretics, verapamil and oral penicillins. Cardiology consultation and ECHO again confirmed severe mitral stenosis, suitable for balloon mitral valvotomy. She remained in heart failure despite maximum decongestant therapy and went into labour after 10 days. She was delivered of a healthy baby after CS and was ventilated over night. She was stable for 24 hours, but developed refractory pulmonary edema and succumbed 2 days after CS.

Death of this young lady was certainly avoidable. She should have undergone BMV before conception as soon as severe MS was diagnosed or at least during pregnancy by 5 – 6 months. This could have been considered again at 34 weeks when she presented with respiratory infection. Respiratory infection in a patient with severe mitral stenosis in pregnancy often indicates heart failure and should merit aggressive measures and continuing hospitalization under close cardiology supervision.

Antenatal medical treatment was certainly inadequate. Vigorous attempt to control heart rate which is so important in treating heart failure in mitral stenosis was

not made till she worsened. If decision was to take her up for CS, this could have been done 2 to 3 days after admission itself when it was already clear that she is unlikely to improve with medical treatment alone. If BMV was possible, that would have been ideal. However in the context of nonavailability of BMV, earlier CS might have given here a better chance of survival.

### Learning points

- Women with severe mitral stenosis in pregnancy especially when associated with severe pulmonary hypertension often do badly
- Aggressive antenatal drug therapy is mandatory. Drugs to control heart rate along with optimal dose of diuretics coupled with diet and adequate rest can often do wonders
- PTMC (Percutaneous Trans Mitral Commissurotomy) should be considered in all women with severe mitral stenosis before conception. This procedure can also be performed safely during pregnancy
- Women with severe valvular heart disease in pregnancy require specialized care and should be under supervision of a team consisting of cardiologist, obstetrician and anaesthesiologist.
- There is a need for government or non government agencies to evolve strategies to offer advanced cardiac interventions to financially stressed pregnant women with valvular heart disease.

### Case 4

A 24 year old, gravida 2, married for 6 years, presented with features of right heart failure at 25th week of pregnancy. She had been diagnosed to have primary pulmonary hypertension 5 years earlier and was advised against pregnancy. Her first pregnancy was terminated at 3 months of gestation on the advice of cardiologist. She was again advised MTP at 5 weeks of the present pregnancy. Patient and her husband opted to continue pregnancy at their own risk. Antenatal visits were regular. She was promptly admitted under cardiology and treated. Cardiologists preferred an early induction of labour, but obstetrician and radiologist preferred to wait as the patient was relatively asymptomatic and fetal salvage was poor in the institution before 34th week. 3 days before scheduled date of initiating induction, she went into spontaneous labour and delivered vaginally. Apart from mild postpartum hemorrhage, there were no major problems and baby was feeding well. She developed fever on third day (antibiotics started), and breathlessness from fourth day (oral warfarin started). She complained of chest pain on eighth day. Pulmonary embolism was suspected and was put on parenteral heparin. However she failed to improve and died on ninth postpartum day.

## Learning points

- Primary pulmonary hypertension during pregnancy is associated with high maternal mortality and all patients should be strongly advised against pregnancy. MTP should be done in case the woman becomes pregnant.
- There could be a role for sildenafil in managing primary pulmonary hypertension in pregnancy
- A multidisciplinary team approach is valuable in treating such complicated pregnancies
- Post partum period is a vulnerable period for patients with primary pulmonary hypertension
- Prophylactic anticoagulation should be considered in patients with primary pulmonary hypertension

This patient had very good antenatal care under a multi disciplinary team. Early induction of labour may have resulted in a different outcome. However she was free of symptoms and delivery was accomplished without much problems. There could have been a bit of complacency in the postpartum period. It is well known that most of the complications of primary pulmonary hypertension occur in this period and ICU care and prophylactic heparin may have avoided pulmonary embolism which certainly appears to be the cause of this unfortunate death.

**Table 11.1 Types of Heart diseases observed by the CRMD**

Type of Heart disease	2004	2005
Valvular	nil	6
Cardiomyopathy	2	3
Pulmonary Hypertension	1	3

## Conclusions

- Heart disease is still potentially dangerous in pregnancy, even though disease patterns have changed.
- Patients with heart disease planning pregnancy should have full cardiac evaluation structural heart disease, left ventricular function and pulmonary hypertension.
- Pregnant patients with heart disease should be managed in centers with facilities for cardiology supervision and good emergency medical care. A multidisciplinary approach is often desirable.
- Anaemia in pregnant women with heart disease often adversely affects outcome and needs aggressive management

- Women with pulmonary vascular disease have a very high risk of dying in pregnancy. Patients with primary pulmonary hypertension and Eisenmenger's syndrome have an estimated mortality of 30 to 50 %. These patients require intensive monitoring during pregnancy and in the post partum period. Patients should be counselled against pregnancy, if their condition is diagnosed before conception.
- With widespread availability of cardiac surgery, more number of pregnant patients with surgically corrected congenital cardiac defects are being seen. These women even if asymptomatic may have residual pulmonary hypertension and can die during pregnancy or immediate post partum. PA pressure should be carefully measured in these patients and if found elevated should be monitored closely.
- Rheumatic heart disease and mitral stenosis are still common among pregnant patients in our state. They should have a complete cardiac evaluation including ECHO and should be closely followed up by cardiologist. PTMC after 20 weeks should be considered in patients with symptoms of heart failure and functional class more than 2 or severe mitral stenosis.
- Peripartum cardiomyopathy is increasingly being recognised . This possibly should not be overlooked especially since these women may not have any symptoms of heart disease before pregnancy or till the last month.
- Arrhythmias can rarely complicate pregnancy when an aggressive approach in management is needed.

## Annexure I

### Predictors of Maternal Risk for Cardiac Complications

- Prior cardiac events (heart failure, transient ischemic attack, stroke prior to pregnancy)
- Prior arrhythmia (symptomatic sustained tachyarrhythmia or Brady arrhythmia requiring treatment)
- NYHA functional class > 2 or cyanosis
- Valvular and outflow tract obstruction (aortic valve area < 1.5 cm<sup>2</sup> mitral valve area < 2 cm<sup>2</sup> or left ventricular outflow tract peak gradient > 30 mmHg)
- Myocardial dysfunction (LVEF < 40% or restrictive cardiomyopathy or hypertrophic cardiomyopathy).

NYHA: New York Heart Association

LVEF: Left ventricular ejection fraction

## Annexure II

### **Recommendation for Anticoagulation during Pregnancy in Patients with Mechanical Prosthetic Valves** (Adapted from ACC/AHA guidelines and ACCP consensus conference)

1. Heparin during the first trimester or to continue oral anticoagulation throughout pregnancy: Discuss with patient and her partner. Warfarin embryopathy 4% to 10%. If she chooses heparin for the first trimester, she should be made aware that heparin is less safe for her, with a higher risk of both thrombosis and bleeding.
2. High-risk women (a history of thromboembolism or an older-generation mechanical prosthesis in the mitral position) who choose not to take warfarin during the first trimester should receive continuous unfractionated heparin intravenously in a dose to prolong the mid-interval (6 h after dosing) activated partial thromboplastin time to 2 to 3 x control value. Transition to warfarin after first trimester
3. Women at low risk (no history of thromboembolism, newer low-profile prosthesis) might be managed with adjusted-dose subcutaneous heparin (17,500 to 20,000 U twice daily to prolong the mid-interval (6 h after dosing) activated partial thromboplastin time to 2 to 3 x control value.
4. In patients receiving warfarin, the international normalized ratio should be maintained between 2.0 and 3.0 with the lowest possible dose of warfarin, and low-dose aspirin should be added.
5. Warfarin should be stopped no later than week 36 and heparin substituted in anticipation of labor
6. If labor begins during treatment with warfarin, a cesarean section should be performed.
7. In the absence of significant bleeding, heparin can be resumed 4–6 h after delivery, and warfarin begun orally.
8. Heparin can be substituted with LMWH in dose adjusted according to weight or as necessary to maintain 4-h postinjection anti-Xa heparin level of about 1.0 IU/ml.
9. Catheter placement for epidural anesthesia is not advisable within 10 to 12 h of the last dose, because of longer half-life of LMWH. For this reason, and to prevent spinal or epidural hematoma, LMWH should be withdrawn 18 to 24 h before an elective delivery and substituted with intravenous UFH.

### Annexure III

#### Endocarditis prophylaxis

The committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the American Heart Association does not recommend routine antibiotic prophylaxis in patients with valvular heart disease undergoing uncomplicated vaginal delivery or caesarean section unless infection is suspected. Antibiotics are optional for high-risk patients with prosthetic heart valves, a previous history of endocarditis, complex congenital heart disease, or a surgically constructed systemic-pulmonary conduit. Many practitioners routinely provide antibiotics.

### Annexure IV

#### Valvular heart disease that is associated with high maternal risk or fetal risk during pregnancy

- Severe aortic stenosis with or without symptoms
- Aortic regurgitation (AR) with NYHA functional class III-IV symptoms
- Mitral stenosis (MS) with NYHA functional class III-IV symptoms
- Mitral regurgitation (MR) with NYHA functional class III-IV symptoms
- Aortic or mitral valve disease that results in severe pulmonary hypertension (pulmonary pressure > 75 % of systemic pressure)
- Aortic or mitral valve disease with severe left ventricular (LV) dysfunction (ejection fraction (EF) < 40 %)
- Mechanical prosthetic valve that requires anticoagulation
- AR in Marfan's syndrome

### Annexure V

#### Valvular heart disease that is associated with low maternal and fetal risk during pregnancy

- Asymptomatic AS with low mean gradient (< 50 mm Hg) in presence of normal LV systolic function (EF > 50 %)
- NYHA functional class I or II AR with normal LV systolic function



- NYHA functional class 1 or II MR with normal LV systolic function
- Mitral valve prolapse (MVP) with no MR or with mild to moderate MR and normal LV systolic function
- Mild to moderate MS (mean valve area  $> 1.5 \text{ cm}^2$ , gradient  $< 5 \text{ mm}$ ) without severe pulmonary hypertension
- Mild to moderate pulmonic stenosis

## Annexure VI

### Severity of Mitral stenosis

The severity of mitral stenosis is classified based on the valve area. Normal mitral valve area is 4 to 5 cm<sup>2</sup>

Mild MS:	Valve area $> 1.5 \text{ cm}^2$ .
Moderate MS:	Valve area 1.1 to 1.5 cm <sup>2</sup> .
Severe MS:	Valve area $\leq 1 \text{ cm}^2$ .

## Annexure VII

### *NYHA functional status classification*

Class I	Asymptomatic
Class II	Symptoms with greater than normal activity
Class III	Symptoms with normal activity
Class IV	Symptoms at rest





## Chapter 12

# Liver Diseases in Pregnancy

**Dr. N. Viswanath**

On behalf of the Editorial Board

### Liver diseases in pregnancy; Key summary points

- Liver disease is a major cause of death in our mothers. There were seventeen deaths in the two years of which 15 were reported to CRMD.
- Liver cell failure, massive bleeding, and multiorgan failure are the major causes of death among patients with liver disease.
- Though our data is incomplete, the survival chances of our mothers with major hepatic diseases and their fetuses when compared to West are very poor. Their mortality had come down in the last decade or two, with early diagnosis, and proper and expeditious management.
- Early diagnosis is the key. In five cases there was evidence that diagnosis could have been made earlier.
- Correct diagnosis would lead to starting management at an earlier stage, or referral to a higher center, when salvage would be more likely.
- Use of blood components, have been sub optimal in most cases. Availability and financial constraints appeared to have played a role. Lack of awareness of proper use of the components was also apparent in most cases. This is an area that could be addressed.
- In one case, the management of severe sepsis appeared to have shortfalls. Greater diligence could have been exercised in identifying the probable source of sepsis.

### Liver disease in Pregnancy: 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. Any 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 would need checking. Repeat these before any intervention such as induction or caesarean 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.

### **Liver disease in pregnancy; learning points**

- Liver diseases in pregnancy, is a heterogeneous group of conditions. The more dangerous ones need prompt diagnosis and optimal management to avoid maternal and fetal losses.
- A correctly performed prothrombin time is an invaluable test for assessing the synthetic function of liver and has prognostic significance.

- Objectivity in clinical assessment , ordering and analysis of results of investigations and planning treatment coupled with frequent assessment of clinical status ( progress or deterioration ) is suggested.
- In liver cell failure, hypoglycemia, hypokalemia and sepsis are to be carefully looked out for and corrected. Clearly and carefully laid down protocols for infection control should be enforced.
- Nursing care is a vital component of the total care of such patients. Continuing education, regular assessment and ensuring active wholehearted involvement of nursing and paramedical staff are vital links in imparting quality care.
- In house audit will be very useful in the units that manage critically ill patients.

### Summary and general comments

Fifteen patients had liver disease related deaths reported to CRMD; twelve due to acute liver disease; one was a patient of portal hypertension (probably extrahepatic portal vein obstruction) with haemoperitoneum, in all probability due to splenic rupture. Most of the deaths were understandably due to liver failure. Severe bleed and multiple organ dysfunction were also contributory. Two were probably due to severe sepsis. One patient; a known case of von Willebrand's disease was jaundiced and died due to bleeding for which hepatic dysfunction also would have contributed.

### Suboptimal management

Suboptimal management may have contributed to a variable extent in three cases. Delay in arriving at the diagnosis was the main factor in two cases.

Table 12.1 Case Summary

No	Age	Gravida	Clinical features	delivery	days	Probable cause of death
1	24	2	Epigastric pain, weakness, disorientation	LSCS	2d	Liver cell failure, ongoing bleed
2	24	2	Itching , vomiting, fever, jaundice	vaginal	5d	Liver cell failure, hypotension
3	24	3	Triplet pregnancy abdominal distension	LSCS	23d	Liver cell failure? acute abdomen
4	30	5	PIH, icterus, absent fetal movements	LSCS	2d	Liver cell failure MODS
5	22	P	Pain abdomen, distension	vaginal	30mts	Intraperitoneal bleed, ? ruptured spleen
6	25	P	PPH , jaundice	vaginal	3d	Liver cell failure
7	26	G1	Fever, chills, itching, jaundice		26d	Sepsis, ?biliary tract disease
8	22	G1	Pain abdomen, jaundice , fever	vaginal	14d	Liver cell failure, cerebral edema
9	24	G2	Prev CS,now repeat CS, jaundice noticed next day	LSCS	9	Liver cell failure and MODs
10	26	2	Vomiting, fever, general weakness, jaundice	LSCS	17	Hepatorenal failure, Ventilator associated pneumonia
11	29	1	IUD at term	craniotomy	1	Hepatocellular failure
12	26	1	Jaundice, hepatorenal failure,	LSCS	3	Hepatorenal failure
13	15	1	Unplanned pregnancy, jaundice, Known von Willebrand's disease	Vaginal	1	Coagulation failure
14	17	1	Jaundice noticed on . the day of EDC	Vaginal	1	Hepatic failure
15	26	2	Jaundiced on 2 <sup>nd</sup> post op day. Coagulation failure & internal bleeding	LSCS	10	Hepatorenal failure

## Learning lessons from the deaths

### Case 1

A 24year old second gravida, wife of a farmer, presented to the local hospital at 37

weeks of gestation with epigastric discomfort and heartburn since the previous two weeks. She was admitted, given I V fluids and discharged in two days. A few days later in addition to continuing epigastric pain, the patient developed generalized weakness, fever, giddiness and disorientation and was admitted to a level 2 hospital. Investigations done there showed deranged liver functions. The patient was referred to a level 3 hospital with a diagnosis of jaundice complicating pregnancy progressing to encephalopathy. Her BP at admission was 100 systolic. Some of the investigations were as follows; Hb 9.6, TC 28500/mm<sup>3</sup>, N61, L36, platelets 180,000/mm<sup>3</sup> random blood sugar 70 mg%, B1 urea 40 mg%, serum creatinine 2.0 mg%, Bilirubin 12.2 mg/dl, direct 9.8 mg/dl, SGOT 652 IU, SGPT 322 IU Alkaline phosphatase 956 u Protein total 5.7 G/dl, albumen 3.2 G/dl, globulin 2.5 G/dl and prothrombin time patient 30 sec to a control of 12 seconds. Patient underwent caesarean section after being given 3 units of FFP. The newborn survived. But the mother continued to bleed post op despite multiple transfusions of plasma, and blood and was in hypotension and died 36 hours after delivery.

The case highlights many areas where significant changes have to come in our approach to the care of the pregnant patient.

- Epigastric discomfort which comes on in the third trimester may often have sinister implications as this case went on to prove. Though the patient was “admitted for two days and administered IV fluids”, it probably represents a missed opportunity. A careful examination, monitoring of blood pressure and a few basic blood tests such as transaminases, prothrombin time, bilirubin, renal functions may have enabled a diagnosis of at least liver disease complicating pregnancy and led to timely referral to a tertiary care center, or instituting a proper line of management.
- When the patient returned a few days later, severe hepatic and probably renal dysfunction had set in.
- A timely diagnosis of acute fatty liver of pregnancy, or at least suspecting a complicated pregnancy needing careful management could have saved the mother.
- Managing surgery in a patient with coagulopathy and deteriorating liver function is never easy. However this case (and many other cases) highlight the need to use blood components as one would use medicines in the correct dose at the right time and frequency. (This is taken up in the annexure).
- When one is forced to operate, there is need to modify technique. A vertical midline incision may help to reduce bleeding from the wound, compared with a transverse incision.

## Case 2

A 24 year old housewife, wife of a manual labourer, a second gravida at 34 weeks, presented to hospital with generalized itching since the previous two weeks, vom-

iting and fever since four days. No abdominal pain noted. No hypertension noted in antenatal evaluation. Earlier pregnancy was uneventful. Examination revealed a conscious, icteric and mildly dehydrated patient with a BP of 120/70. No hepatomegaly. Relevant investigations were as follows; Random blood sugar 98 and 28 mg/dl, bilirubin total 5.8 mg/dl, direct 2.8 mg/dl, SGPT 620 IU/L, alkaline phosphatase 748 IU/L, Prothrombin time 28 sec to a control of 12 seconds. Platelet count 200,000/cmm. Antibodies to hepatitis A, E and C viruses hepatitis B surface antigen, VDRL and HIV antibody were all negative. She was given supportive care and fresh frozen plasma. She delivered vaginally, a macerated still-born. No postpartum haemorrhage. Eight hours after delivery, she went into confusion, hypotension and needed vasopressor and ventilatory support. She died on the third day postpartum.

This patient appeared to have received reasonable care after admission. The diagnostic possibilities include Acute fatty liver of pregnancy as well as viral hepatitis. The fever, prodrome and lack of abdominal pain suggest the possibility of viral hepatitis in spite of the negative viral markers. This and many other cases like this have an inevitability of a Greek tragedy. Mortality of AFLP and HELLP syndrome have come down in the west by early diagnosis and prompt referral to centers geared to deal with them. The committee's suggestions include,

1. Increasing awareness of practitioners at primary level to the importance of careful antenatal care and highlighting the "red flags" such as epigastric distress or pain coming on in the third trimester, vomiting coming on in an uneventful pregnancy till then, lassitude, tiredness, or just "not feeling well".
2. Most of the patients had attended AN Clinics, which is a good place to start educating expectant mothers and families to report to hospital in the event of new "warning signs".
3. A standard protocol for antenatal care and a system of documenting these findings in a card that should be with the patient wherever she goes or is referred, may prove to be useful. The committee is not unaware of the work load in many outpatients and hospitals at all levels. Some of the data collection and documentation could be done by trained paramedics. This would be a source of immensely useful information to the caregiver even as the patient is wheeled in. A caregiver seeing the patient for the first time could detect subtle changes in what was till then an uneventful pregnancy.

### Case 3

A 24 year old housewife, wife of a manual labourer, a third gravida had regular antenatal check up and was noted to have triplet pregnancy at 5<sup>th</sup> month on ultrasound examination. Pregnancy induced hypertension was noted at 34 weeks and admitted. No abdominal pain or nausea noted. Examination revealed a moder-

ately built and nourished lady with markedly distended abdomen, BP 150/100, no pedal edema. Investigations were as follows

**Table 12.2 Investigation results**

investigations	3 days after admission	15 days after admission	20 days after admission
Bl.urea	17mg/dl		
s. creatinine	0.8mg/dl		0.9mg/dl
Platelet count	270,000	306,000	215,000
SGPT	49 IU/L	231IU/L	664IU/L
Bilirubin		0.6mg/dl	
Alk.phosphatase		400IU/L	906IU/L
Prothrombin time	pt/ c		27 /14
S. albumin			2.3 G/dl

Caesarean section was done on the 17<sup>th</sup> day after transfusing fresh frozen plasma. By second postoperative day, patient developed abdominal distension and respiratory distress. An acute abdomen was suspected. Even as intervention was being debated patient succumbed.

**This case summary presents certain lessons which need to be learnt.**

- Triplet pregnancy is believed by some to be at greater risk of AFLP as fatty acids derived from the three fetuses have to be dealt by the maternal liver.
- Patient had no “warning abdominal symptoms”. She had marked distress due to the grossly distended abdomen. This could have masked the early warnings.
- The investigations serially performed suggest that early warnings were there. In a triplet pregnancy with hypertension, even a marginally raised SGPT three days after admission should at least have prompted more tests the same day or sooner than after 15 days when it was eventually done. The diagnosis of acute fatty liver could have been made earlier and treatment may have modified the outcome.
- The postoperative phase with threat of an acute abdomen suggests a few possibilities. Acute pancreatitis is occasionally seen to complicate AFLP. Another life-threatening condition described recently is the Abdominal compartment syndrome.

## Case 5

Twenty two year old housewife, a known patient of portal hypertension, esophageal varices and splenomegaly, delivered vaginally, a full-term baby over a week



prior to present admission. She presented to hospital on the ninth postpartum day with acute pain abdomen, distension, marked pallor and peripheral circulatory failure. Examination revealed a pale, very ill patient, BP was 760 mm of Hg, with a pulse rate of over 140/mt. Uterus was well contracted. Ultrasound showed free fluid in peritoneum. Paracentesis revealed frank blood. Patient continued to deteriorate and succumbed in thirty odd minutes after admission.

This patient had massive hemoperitoneum, secondary to probably a ruptured spleen. The large spleen of portal hypertension is prone to trauma. Even apparently trivial trauma could lead to rupture. Though there are case reports of ruptured spleen in the peripartum phase, in this patient the timing may have been accidental. Occult domestic violence may also rear its ugly head as a cause in such instances.

#### Case 7

A primigravida aged 26 years was admitted with high grade fever often with chills, jaundice, vomiting and itching all over the body for the previous two days. She was three weeks from term and had no history of hypertension or pedal edema. Her temperature was 103 degrees F, BP was 130/80 and heart rate was 116/mt. She had mild icterus. She delivered a term, male baby weighing 2kg, fifteen days after admission. Her hospital stay was marked by recurrent high-grade fever, often associated with chills and pain abdomen. She developed pedal edema and ascites post-delivery. During the last few days of her life she became stuporous and succumbed twenty-six days after admission. Investigations include the following; Hb 10.0 G/dl, Total count repeatedly over 15000/mm<sup>3</sup> with neutrophilic leukocytosis, toxic granulation and shift to the left. LDH done repeatedly was ranging between 900 and 1500. Malarial parasite was not detected. Bilirubin ranged between 3 and 12 mg/dl, with conjugated fraction predominating. Transaminases were three to five times normal, but alkaline phosphatase ranged from 450 soon after admission to 1133 and 1593 eight and ten days later. Prothrombin time and platelets were repeatedly normal. Serum proteins were 5.7 G (total) with albumin being 2.7 G/dl. Urea & creatinine were normal. HBsAg, HAV Ab IgM, HEV Ab IgM, HCV Ab were negative. Reticulocyte count was 0.9 %. Ultrasound apparently done only once, showed mild hepatomegaly with normal echotexture, no dilation of intrahepatic radicles and pregnancy of 33 weeks size. Ampicillin was added on the seventh hospital day. Collagen workup and HIV antibody were negative. Blood culture done on the sixteenth day of hospital stay grew *Acinetobacter* sensitive to ampicillin, cotrimoxazole, norfloxacin, amikacin, gentamicin, ceftriaxone and resistant to ceftazidime. Patient received ampicillin 1.5 to 2.0 grams a day from the sixth hospital day till demise, Cefotaxime 1 G eighth hourly was added from the sixteenth day of hospitalization, but changed to ceftazidime and metronidazole on the eighteenth day till death. A trial with

antimalarials was started on the sixteenth day of hospitalisation.

The patient had a rather long hospital stay and extensive investigations. However certain features need to be stressed. Though jaundice was one of the presenting symptoms, she also had high-grade fever and chills and abdominal pain before and during hospitalization. The high counts and the toxic granules suggest sepsis. Viral hepatitis looked unlikely clinically and the serological tests were negative. Other diagnoses such as HELLP syndrome and AFLP were excluded. Certain points need to be stressed.

- When there are features of sepsis one or more blood and other cultures should be taken at the earliest. Antibiotics (broad spectrum), should be started after that on a presumptive basis. Based on culture reports these may need modification.
- The choice of Ampicillin cannot be faulted, but the dose for a suspected septicemia is inadequate especially when the patient continued to have high-grade fever and chills and had a steady downhill course. It was added only after a week of hospitalization. Though cefotaxime was added later, the reviewers feel more powerful antibiotics in adequate doses should have been started earlier
- The illness started with pruritus, fever with chills, jaundice and rising alkaline phosphatase. One ultrasound did not show biliary dilation and the gall bladder was contracted. As the fever continued, with all the above features and a neutrophilic leukocytosis, reassessment of biliary tree, we feel, was warranted. Choledocholithiasis is notorious for not causing dilation of biliary tree in many as the obstruction is intermittent. Repeated ultrasound examinations are occasionally needed to pick up such disease and even endoscopic retrograde cholangiography may be done as one could also treat the obstruction if any was present. During pregnancy, ERCP may be done if the indication is strong. Radiation may be kept to the minimum.

### Case 8

A 22 year old primigravida in her 17<sup>th</sup> week of gestation, complained of pain right half of the abdomen since one month prior to admission. She had high grade fever with chills on and off, high colored urine, nausea, a variable diminution of appetite and worsening of the abdominal pain since the previous week. She had been to Bombay recently. Examination revealed a person of average build and nutrition, moderately icteric, with no stigmata of chronic liver disease. Systemic examination was unremarkable with no abdominal organomegaly. Haemogram was normal.

Table 12.3 Investigation results

	Day One	6 <sup>th</sup> Day	8 <sup>th</sup> Day	11 <sup>th</sup> Day
Bilirubin t/d/I	7.7/4.4/3.3	15/8.8/6.2	13.7/8.2/5.5	24.5/6.0/18.5
SGOT/SGPT/AP	1386/1308/143	841/712/		
Bl urea/s.creatinine	10 mg/	06mg/0.5	- /0.6mg	— /0.8mg
Platelets	200,000	230,000		170,000
Prothrombin time	16 sec/12	58sec/12	53/11	>1mt/11
Protein/tot/alb/glo	7.2/3.6/3.6			5.1G/2.3/2.8
Na/K		138/4.6	144/4.7	145/4.8

Viral markers for hepatitis A to E negative, antibody to herpes simplex negative, autoantibodies, copper studies, normal. Blood cultures and smear for malaria repeatedly negative. Ultrasound on at least three occasions showed no dilatation of bile ducts.

She aborted on 6th day, no increased bleeding. Sensorium worsened and features of raised intracranial tension developed. Supportive care and ventilation in vain.

The total picture is of viral hepatitis though viral markers were negative. The travel suggested HEV as a possibility but antibody was negative. Care was adequate. A question about whether ventilation was delayed remains. But again a different result looked unlikely even if early ventilation was given.

## ANNEXURE-A

### HEPATOBIILIARY DISEASE IN PREGNANCY

Hepatobiliary disease occurring in a pregnant patient poses a challenge for the treating physician. Western data indicate a prevalence of 3% of deliveries. Traditionally this has been grouped as

- **Those present at the time of pregnancy.** e.g. Hepatitis B, C, chronic infection, cirrhosis, Extrahepatic portal vein obstruction, Wilson's disease, Autoimmune hepatitis etc.
- **Those that occur coincidentally with pregnancy** e.g. Viral hepatitis, drug induced hepatitis, choledocholithiasis etc.
- **Those that occur exclusively during pregnancy.** e.g. Acute fatty liver of pregnancy, HELLP syndrome, Hyperemesis gravidarum, Cholestasis of pregnancy etc.

### Annexure A-1

Table 12.4 Variations in liver test results in normal pregnancy

Test	Effect	Trimester of maximum change
Albumen	Less by 10-50%	Second
Fibrinogen	Increase by 50%	Second
Bilirubin	Normal	
Alkaline phosphatase	Increase 2-5 fold	Third
SGOT/ SGPT	Normal	
Cholesterol	Increase by 2 fold	Third

### ANNEXURE A-2

#### Conjugated versus unconjugated bilirubin; Gilbert's syndrome, haemolysis

The solubility in water of bilirubin is increased by conjugation with glucuronic acid. Congenital deficiency of the enzyme bilirubin glucuronyl transferase leads to increase in unconjugated bilirubin in 4-6 % of healthy young adults. This is increased in the event of calorie deprivation (fasting, persistent vomiting, postoperative state) or any febrile illness. The person is otherwise asymptomatic. Her transaminases are normal, urine bile is negative (this is important because fractionation of bilirubin is prone to technical errors). So raised bilirubin with a negative test for urine bile would indicate unconjugated hyperbilirubinemia. The above person may be having **Gilbert's syndrome** which is not a disease. Haemolytic disorders could also cause raised unconjugated bilirubin, but patient would have other features of hemolysis such as raised reticulocyte counts, low haemoglobin, low serum haptoglobin and clues in the peripheral smear. HELLP syndrome is associated with hemolysis due to microangiopathy and may have raised unconjugated bilirubin, but transaminases are elevated and platelets are decreased. The peripheral smear would show evidence of microangiopathy induced hemolysis.

### ANNEXURE A-3

**Prothrombin time.** This is an invaluable test for liver function in our setting. It tests the coagulation factors synthesized by the liver. These factors have short half lives and hence reflect acute changes in hepatic synthetic function especially when the patient has already been on vitamin K (vitamin K fast prolongation of pro-

thrombin time or raised INR). Prolongation of prothrombin time in a patient with liver disease is an ominous sign. Laboratories vary in their reliability while performing this test. An experienced, competent technician in your area who could give you reliable results would be a great asset.

### Annexure A-4

Table : 12.5 Suggested Serology test for Viral Hepatitis

Viral etiology suspected	Suggested serology test(s)	Anticipated results
Acute viral hepatitis A	Antibody to HAV, IgM	Elevated
Acute viral hepatitis E	Antibody to HEV, IgM	Elevated
Acute viral hepatitis B	Antibody to HBcore IgMHepatitis Bsurface antigen (HBsAg)	Elevated Positive
Hepatitis due to hepatitis C (acute hepatitis uncommon)	HCV antibody ( takes few wks to appear) HCV RNA by Polymerase chain reaction	Positive Detected
Acute hepatitis D (v. rare) (occurs only with HBV inf.)	HDV Antibody IgM	Positive
Hepatitis due to herpes simplex virus and varicella zoster virus	Respective antibody IgM Liver biopsy	Positive Inclusion bodies Necrosis

### Annexure-B

## A FEW SELECTED TOPICS

### VIRAL HEPATITIS IN PREGNANCY

- Viral hepatitis could occur in any stage of pregnancy
- Only in Hepatitis E and herpes simplex hepatitis, pregnancy is noted to adversely influence outcome. In other viral hepatitis, neither pregnancy affects the outcome of hepatitis nor does hepatitis affect the outcome or course of pregnancy.

#### 1. Hepatitis A.

- No prevalence data. However physicians in the field feel that in Kerala, acute hepatitis in the young adult is on the increase probably because of drop in childhood infections. Western figures are in the range of 1 in 1000 pregnancies. Ours is likely to be several times this figure.

- Course and management is unaffected by pregnancy
- Perinatal transmission is rare
- In the exposed mother immune globulin is safe for mother and fetus.
- Infants of mothers with acute hepatitis A at or soon after delivery may be protected by immune globulin at a dose of 0.02 ml/kg, im. Hepatitis A vaccine may be given after the age of two years.

## 2. Hepatitis B.

### Epidemiology

- 3-5 % of general population are chronically infected with hepatitis B virus. Two studies from North India found frequency of HBV related acute hepatitis as 30-40 % of all acute hepatitis.
- Hepatitis B surface antigen positivity among the pregnant in Kerala is not known.

### Transmission to infant without immunoprophylaxis

- When mother is HBsAg +ve and HBeAg-ve, risk of chronic infection in the child has been 40%
- When mother is HBsAg and HBeAg+ve, the risk of the child developing chronic infection is 90%
- Following 1<sup>st</sup> trimester acute infection of mother, 10% of neonates became positive.
- Following maternal infection in third trimester, 80-90 % of neonates became HBsAg+ve.
- Transmission of infection to the neonate usually occurs at birth.

### Protecting the newborn

- Hepatitis B immune globulin 0.5 ml in one thigh with hepatitis B vaccine in the other thigh within 12 hours of birth protects the child in 90-95% instances. Follow up vaccination at 1 and 6 mo after birth
- Three doses of vaccine alone, the first one at birth, though inferior may be adequate where most HBsAg +ve mothers are HBeAg-ve.

## 3. Hepatitis C

- No adverse effect on pregnancy
- Pregnancy does not adversely affect the infection

- In India approximately 1-1.5% of the general population is chronically infected by hepatitis C virus.
- Rate of transmission from mother to infant is generally low, in the range of 1-5%; higher rates are seen when viral titres are high and with HIV co-infection.
- No effective prophylaxis available for infants.

#### 4. Hepatitis E

- Jaundice is nine times more common in pregnant than nonpregnant women
- Mortality in nonpregnant persons is 0.5- 4%, whereas in the third trimester, mortality is 20%.
- Incidence of abortion and intrauterine death is 12%.
- Vertical transmission is known to occur.
- No therapy yet.
- No data to indicate if termination of pregnancy affects survival. This has probably not been looked into scientifically.

#### 5. Hepatitis due to herpes simplex virus

- Rare, often in the second or third trimester .with only half presenting with typical mucosal lesions.
- Often anicteric, may even cause fulminant hepatitis
- Fever, nausea, vomiting abdominal pain, leukopaenia, coagulopathy, thrombocytopaenia and markedly elevated transaminases are often seen.
- Liver biopsy shows typical intranuclear viral inclusion particles, haemorrhage, and necrosis
- Hepatic necrosis, DIC, hypotension and even death can occur if antiviral therapy is not started rapidly. Acyclovir is the drug of choice.

### 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 48hours, 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 haemoglobin 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 may be needed sometimes.



## Obstetric management

- Assessment of fetal and maternal status , intervention to stabilize when needed
- Delivery is indicated for pregnancies  $\geq 34$  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 haemorrhage, renal failure, or abruptio placentae.
- Normal vaginal delivery is possible in most patients following induction. Caesarean section can be considered in very preterm gestations (under 30 weeks) when cervix is unfavourable.
- In pregnancies  $<34$  weeks , where mother and fetus are stable, corticosteroids to enhance fetal lung maturation should be given while both mother and fetus are closely monitored. Delivery within 24 hours of last dose recommended even when biochemical “improvement” is seen.
- Magnesium sulphate to prevent convulsions (*4g IV followed by 1g per hour as continuous infusion.*) It may be stopped 24 hours postpartum or when remission is noted, whichever is longer.
- Control of hypertension
- Expectant management of stable, preterm pregnancies should be considered investigational.
- Corticosteroids (*dexamethasone 10 mg IV every 12 hours*) can be given till remission is noted.

Table 12.6 Clinical features of AFLP and HELLP ; a comparison

	AFLP	HELLP
Glucose	decreased	normal
Creatinine	increased	Normal
Uric acid	^^	^
Bilirubin	^(conjugated)	^(occ unconjugated)
LDH	Normal	^^
SGOT/SGPT	Increased	Increased
Platelets	Normal	Decreased
PTT	Prolonged	Normal
PT	Prolonged	Normal
Fibrinogen	Decreased	Normal
Histology	Microvesicular fatty infiltration	Periportal haemorrhage
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. US Scan may show hyperechoic liver. CT which shows decreased attenuation is more reliable than US 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
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## Chapter 13

# Thromboembolism

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on behalf of editorial board

### Key Summary Points

- Eighteen maternal deaths reported during the period 2004 & 2005 are assigned to venous thromboembolism. Only ten of them were reported to CRMD. The others were reported through DHS. This chapter analyses only those ten reported to CRMD.
- Seven of the ten were assigned to pulmonary embolism and three to cerebral venous thrombosis.
- Six of the seven assigned to pulmonary embolism, followed caesarean delivery.
- Only one of the three with cerebral venous thrombosis followed caesarean delivery

### Key Recommendations

There is a significant number of maternal deaths due to venous thromboembolism. Hence an assessment of risk factors for this should be done in all antenatal women at the time of booking for antenatal care and admission. In cases of severe pre-eclampsia of early onset, thrombophilia should be ruled out.

Thrombophilias should be looked for in women with previous history of thromboembolism and recurrent pregnancy loss. The guidelines on thromboprophylaxis during pregnancy and labour should be considered. Any symptom suggestive of thromboembolism should be treated on an emergency basis and anticoagulation may be started even before a firm diagnosis is established. Use of newer diagnostic methods like D-Dimer assay, echocardiography and spiral CT can help in establishing early diagnosis of pulmonary embolism. In established cases of massive pulmonary embolism, interventional procedures may help in saving lives.

## Summary

There were 10 cases of maternal deaths assigned to venous thromboembolism – seven to pulmonary embolism and three to cerebral venous thrombosis. But the diagnosis is not confirmed by definitive tests like perfusion studies or even CT scan. So, there will be a question mark on the diagnosis. Similarly there are cases assigned to other causes eg.severe PIH where the final cause of death might have been pulmonary embolism. A summary of the cases assigned to pulmonary embolism is given in the table13.1.

**Table 13.1**  
**Pulmonary embolism**

S.No	age	G/P	Period of gestation	Mode of delivery	When died	Remarks
1	25	G4P4	40	Emergency CS	20 <sup>th</sup> day	Sepsis,DIC, ventilator
2	27	G2P2	40	Elective CS	7hours	dyspnoea,
3	33	G3P2	37	Elective CS	4 <sup>th</sup> day	dyspnoea,Cardiac Arrest
4	37	G2P2	40	Elective CS	4 <sup>th</sup> day	dyspnoea
5	35	G2P1	32	Vag del	7 <sup>th</sup> day	jaundice, raised LDH
6	27	G2P2	38	Elective CS	2 <sup>nd</sup> day	Chest discomfort
7	21	G1	40	Elective CS	4 hrs	breathlessness
						Hypotension

The BMI of these patients is not known. Looking at each case no definite risk factor other than caesarean as the mode of delivery is identified in these cases. Hence it becomes difficult to make recommendations regarding prophylaxis.

## Learning from example

### Case 1

A 19 yr old primigravida , 32 wks pregnant, was referred to tertiary care centre with history of oliguria. She was on antihypertensive since 26 weeks. At the time of admission she had a BP of 140/106 mmHg, pedal edema, puffiness of face and abdominal wall edema, uterus was only 28 weeks size. Blood investigations showed hypoalbuminemia and mild elevation of renal parameters. Ultrasonogram done 14 hours after admission showed maternal ascites. Renal parameters were increasing and decision for termination was taken 36 hours after admission. BP remained under control. Urine output was adequate. Induction was started but she did not get delivered even after 44 hours of induction. She suddenly developed breath-

lessness and hypotension while in active labour. She was managed with steroids, oxygen and bronchodilatation. Anaesthesiologist and physician were involved in the management. In about 4 hours she became restless, cyanosed and collapsed. ECG showed changes suggestive of right ventricular hypertrophy. The Cardiologist diagnosed massive pulmonary embolism. At end of 90 hours after admission patient was declared dead.

Analysis of this case reveals many issues. The patient was admitted to a tertiary centre with early onset severe PIH with ascites and elevated renal parameter; early decision for termination could have been taken. Even after initiation of induction, delivery did not occur for 44 hours. In such situations, abdominal delivery may have to be resorted to. When the patient developed breathlessness, though physician and anaesthesiologist have seen the case nobody suspected the possibility of pulmonary embolism. Hence investigation or management for this condition was not sought early. In this patient with early onset pre-eclampsia the possibility of antiphospholipid antibody syndrome also had to be thought of and if it was diagnosed earlier, probably thromboprophylaxis might have been started earlier.

### **Case 2**

37 year old second gravida with no living children was admitted at 38 weeks with pain abdomen. She had regular antenatal check up. She had a caesarean section for foetal distress under spinal anaesthesia. No antenatal or intranatal complication. On 4th postoperative day patient became dyspnoeic. Then she was given steroids and bronchodilators. She was shifted to ICU. By that time she was in shock. She was intubated and put on ventilator. But within 20 minutes of onset of symptoms the patient was declared dead.

This case is a good example of a sudden death in the postoperative period. Though resuscitation measures were instituted urgently the patient could not be saved. The single risk factor which could be identified for an embolism is the age of the patient; whether thromboprophylaxis could have saved her life or not is debatable.

### **Case 3:**

#### **Atypical Presentation**

A 26 yr old primigravida presented with hemoptysis at 16 weeks of pregnancy. She was investigated and causes like tuberculosis, malignancy, connective tissue disorders, A-V malformations were ruled out. She was readmitted with hemoptysis. At 31 weeks LSCS was done. Patient developed right heart failure. She was managed in ICU. Meanwhile work up for thrombophilia was done and she was found to be positive for lupus antico-

agulant. She was then started on steroids and anticoagulants. Gradually the condition worsened and she expired on the 25th postoperative day.

### Thromboembolism – Learning points

- Failure to appreciate the significance of symptom is one of the main causes of delay in diagnosis.
- Any symptom as chest pain, cough, hemoptysis, headache, convulsion and abnormal behaviour must be given due importance.
- Suboptimal management in these cases has led to the deaths
- Risk stratification must be done in antenatal and labouring women
- Implement adequate thromboprophylaxis
- Any symptom suggestive of embolism is an emergency and anticoagulation is indicated even before diagnosis is clearly established.
- Appropriate investigations must be done to confirm the diagnosis as D-Dimer assay echocardiography and spiral C.T.

### Cerebral Venous Thrombosis

There were three cases assigned to this category. A summary of those cases are as follows

**Table 13:2**

No	Age	G/P	Mode of delivery	
1	24	G5P2	Normal delivery	Brought back 20days later dead.
2	25	G1P1	Discharged normally	h/o convulsions at home & on the way
			Elective CS,discharged on day 5	Brought back same night with headache. CT confirmed thrombosis & hge. Craniotomy done.
3	20	G1P1	Normal labour, normal discharge	There was septicaemia also. Brought back after two weeks with history of convulsions

No definite risk factors can be identified in any of these patients except the second case who had caesarean section . Often the warning sign that one can get is only headache. A new onset headache in a postpartum patient should not be ignored.

The same etiological factors operate for cerebral venous thrombosis and for pulmonary embolism.

## Learning from example

This 25yr old G2 had an elective caesarean for transverse lie. Her immediate post-operative period was uneventful and hence she was discharged on 5<sup>th</sup> day. By evening she was readmitted with headache which was treated symptomatically. By early morning the symptoms intensified further and she went into decerebrate posture. She was transferred to a higher centre where CT scan confirmed cerebrovenous thrombosis. Relatives took her to a higher centre where 3 days later craniotomy was done. She succumbed 4 days later.

## Learning Points

There are no identifiable risk factor in this patient and hence no recommendations for thromboprophylaxis can be made. However, it is doubtful whether the gravity of the situation was realized when she came with headache. Proper investigation and definitive treatment even at that point would have made a difference.

## 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 (table 7.1). 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 caesarean. Reducing episiotomy rate and using transverse incision for caesarean 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 postpartum.



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. Caesarean or midcavity instrumental delivery or caesarean hysterectomy
- h. Enforced bed rest for 4 days or more
- i. Sick cell anaemia

It is suggested that if 3 or more of the factors exist, thromboprophylaxis with 5000units per day of low molecular weight heparin (or equivalent) 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 caesarean, a risk assessment should be done regarding thromboprophylaxis and if they belong to moderate or high risk group appropriate medication be given.

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### RCOG Recommendations

( Adapted from RCOG guidelines. for details refer to RCOG guidelines)

The Royal College of Obstetricians & Gynaecologists have given guidelines regarding thromboprophylaxis which is available in the internet. Some parts are reproduced here for convenience of the reader.

All women should undergo an assessment of risk factors for VTE in early pregnancy or before pregnancy. This assessment should be repeated if the woman is admitted to hospital or develops other intercurrent problems (Grade C)

Women with previous VTE should be screened for inherited and acquired thrombophilia, ideally before pregnancy (Grade B)

Regardless of their risk of VTE, immobilization of women during pregnancy, labour and the puerperium should be minimized and dehydration should be avoided. (\*Good practice point)

Women with previous VTE should be offered postpartum thromboprophylaxis with LMWH. It may be reasonable not to use antenatal thromboprophylaxis with heparin in women with a single previous VTE associated with a temporary risk factor that has now resolved. (Grade C)

Women with previous recurrent VTE, or a previous VTE and a family history of VTE in a first degree relative, should be offered thromboprophylaxis with LMWH antenatally and for at least 6 weeks postpartum. (Grade C)

Women with previous VTE and thrombophilia should be offered thromboprophylaxis with LMWH antenatally and for at least 6 weeks postpartum. (Grade B)

Women with asymptomatic inherited or acquired thrombophilia may qualify for antenatal or postnatal thromboprophylaxis, depending on the specific thrombophilia and the presence of other risk factors. (Grade C)

Women with three or more persisting risk factors should be considered for thromboprophylaxis with LMWH antenatally and for 3-5 days postpartum. (\*Good practice point)

Women should be reassessed before or during labour for risk factors for VTE. Age over 35 years and BMI greater than 30 or a body weight greater than 90kg are

important independent risk factors for postpartum VTE, even after vaginal delivery. The combination of either of these risk factors with any other risk factor for VTE (such as pre-eclampsia or immobility) or the presence of two other persisting risk factors should lead the clinician to consider the use of LMWH for 3-5 days postpartum. (\*Good practice point)

Antenatal thromboprophylaxis should begin as early in pregnancy as practical. Postpartum prophylaxis should begin as soon as possible after delivery (but see precautions after use of regional anaesthesia). (Grade B)

LMWHs are the agents of choice for antenatal thromboprophylaxis. They are as effective as and safer than unfractionated heparin in pregnancy. (Grade B)

Warfarin should usually be avoided during pregnancy. It is safe after delivery and during breast feeding. (Grade B)(See the chapter on Heart Disease)

Once the woman is in labour or thinks she is in labour, she should be advised not to inject any further heparin. She should be reassessed on admission to hospital and further doses should be prescribed by medical staff. (\*Good practice point)

**The grades of recommendations (B or C) are as follows:**

- B** Requires the availability of well-controlled clinical studies but no randomized clinical trials on the topic of recommendations (evidence levels IIa, IIb, III)
- C** Requires evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities. Indicates an absence of directly applicable clinical studies of good quality.

(Evidence level IV)

**\*Good practice points** The other recommendations represent good practice points; i.e. recommended best practice based on the clinical experience of the guideline development group.

For further details, see the full report

### Risk factors for venous thromboembolism in pregnancy and the puerperium<sup>a</sup>

#### Pre-existing

Previous VTE

Thrombophilia:

Congenital

Antithrombin deficiency

Protein C deficiency

Factor V Leiden

Prothrombin gene variant

Acquired

Antiphospholipid syndrome

Lupus anticoagulant

Anticardiolipin antibodies

Age over 35 years

Obesity (BMI >30 kg/m<sup>2</sup> either pre-pregnancy or in early pregnancy)

Parity > 4

Gross varicose veins

Paraplegia

Sickle cell disease

Inflammatory disorders (e.g. inflammatory bowel disease)

Some medical disorders (e.g. nephrotic syndrome, certain cardiac diseases)

Myeloproliferative disorders (e.g. essential thrombocythaemia, polycythaemia vera).

### **New onset or transient<sup>b</sup>**

Surgical procedure in pregnancy or puerperium (e.g. evacuation of retained products of conception, postpartum sterilization)

Hyperemesis

Dehydration

Severe infection (e.g. pyelonephritis)

Immobility (>4 days of bed rest)

Pre-eclampsia

Excessive blood loss

Long haul travel

Prolonged labour

Midcavity instrumental delivery

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Immobility after delivery<sup>c</sup>

- <sup>a</sup>. Although these are all accepted as thromboembolic risk factors, there are few data to support the degree of increased risk associated with many of them.
- <sup>b</sup>. These risk factors are potentially reversible and may develop at later stages in gestation than the initial risk assessment or may resolve; an ongoing individual risk assessment is important.
- <sup>c</sup>. Risk factors specific to postpartum VTE only.

## **RCOG Risk assessment profile for thromboembolism in caesarean section**

### **Low risk: Early mobilization and hydration**

Elective caesarean section : uncomplicated pregnancy and no other risk factors.

### **Moderate risk : consider one of a variety of prophylactic measures**

- Age > 35 years
- Obesity (>80 kg)
- Parity 4 or more
- Labour 12 hours or more
- Gross varicose veins
- Current infection
- Pre-eclampsia
- Immobility prior to surgery (> 4 days)
- Major current illness (e.g. heart or lung disease, cancer, inflammatory bowel disease, nephrotic syndrome)
- Emergency caesarean section in labour

### **High risk : Heparin prophylaxis with or without leg stockings**

- A woman with three or more moderate risk factors from above
- Extended major pelvic or abdominal surgery (e.g. caesarean hysterectomy)
- Women with personal or family history of deep venous thrombosis, pulmonary embolism or thrombophilia, paralysis of lower limbs.

- Women with antiphospholipid antibody (cardiolipin antibody or lupus anticoagulant).

## Management of different risk groups

### Low risk women

Women undergoing elective caesarean section with uncomplicated pregnancy and no other risk factors require only early mobilization and attention to hydration.

### Moderate risk patients

Women assessed as of moderate risk should receive subcutaneous heparin (doses are higher during pregnancy) or mechanical methods. Dextran 70 is not recommended until after the delivery of the fetus and is probably best avoided in pregnant women.

### High-risk women

Women assessed as high risk should receive heparin prophylaxis and, in addition, leg stockings would be beneficial.

Prophylaxis until the fifth postoperative day is advised (or until fully mobilized if longer).

The use of subcutaneous heparin as prophylaxis in women with an epidural or spinal block remains contentious. Evidence from general and orthopaedic surgery does not point to an increased risk of spinal haematoma.

## References

1. Royal College of Obstetricians and Gynaecologists. Thromboprophylaxis during pregnancy, labour and after normal vaginal delivery. Guideline No.37, London: RCOG; 2004.
2. Royal College of Obstetricians and Gynaecologists, Report of RCOG Working Party as Prophylaxis against thromboembolism in Gynaecology and Obstetrics. London : RCOG: 1995.



## Chapter 14

# Less common causes

**Dr.Vasanthi Jayaraj**

**Dr. V.P. Paily**

On behalf of the Editorial Board

Among the less common causes of maternal death reported to CRMD there were

- Ten cases of sepsis.
- Four cases of ectopic pregnancy.
- Four cases of anemia complicating pregnancy.
- Two patients committed suicide.
- Two cases of pregnancy with SLE
- Three Cerebral hemorrhage
- One case of Road traffic accident at 26 weeks of pregnancy.

## Sepsis

There were ten deaths where the committee felt that sepsis would have been the main factor. These presented varying scenarios like genital tract sepsis spreading to become septicemia with its consequences, wound sepsis or peritonitis. Very few centres seem to have a sensible antibiotic policy. Prophylactic antibiotics in the form of latest generation cephalosporins were given following normal delivery. The blind trust in antibiotics seems to make people ignore the importance of aseptic practices. The “ward sterilizer” is still the most commonly used method to sterilize instruments used for labour. Only very few centres use all inclusive autoclaved sets for conducting labour. “Cidex” use is irrational. Updating and reorientation regarding infection prevention and antibiotic use are badly needed.



## Learning from examples

### Case report

This patient had induction of labour which failed. Realising that the dates were wrong she was discharged and readmitted a month later for elective caesarean. On the 6<sup>th</sup> postoperative day she was very ill and was referred to a higher centre. Septicaemia and ARDS was diagnosed. A relaparotomy produced 2 litres of pus. She subsequently died.

### Learning points

We have very little details of the entire case. But certain points come up. Did the attempted induction one month earlier contribute to the sepsis? It is difficult to answer.

Were the signs of infection missed at the primary centre? Possibly yes. Regular inspection and palpation of abdominal wound, looking for rise of temperature and paying attention to vital signs are the only ways to pick up sepsis in postoperative patients.

### Ectopic Pregnancy

There were four cases of ectopic pregnancy ending in maternal death. Ruptured ectopic and intraperitoneal bleeding will be diagnosed only if there is an index of suspicion. Whenever there are vague symptoms in early pregnancy this possibility should be suspected.

### SLE

**Pregnancy with SLE – intra cranial hemorrhage one week after mid trimester termination of pregnancy.**

The data given in the case file is insufficient. We are not sure whether proper preconception counseling was given or whether the patient became pregnant when the SLE was active. Available data shows that she was having active SLE. A post termination higher dose of prednisolone could have been more appropriate. It is well known for relapses to occur after delivery or even after termination of pregnancy. It could have been prudent to keep the patient in the hospital under close watch on her hematological and immunological parameters before discharge.

### Key points

- SLE with pregnancy is a dreaded combination.
- Pre pregnancy counseling is very important.
- Pregnancy should be attempted only in remission.

- Problems associated with mid trimester termination are similar to that of term delivery and close follow up during post partum/post abortal period is very important.

## Cerebral Haemorrhage

There were three cases of cerebral haemorrhage. We did not include hypertensive encephalopathy under this group. These three cases were suspected to have other causes like aneurysmal or av malformation rupture. A sample case was like this

### Case report

This primigravida had an uncomplicated antenatal period. She was admitted at term with spontaneous onset of labour and developed headache when she was almost fully dilated. She also had weakness of left hand so that she was finding it difficult to hold on to her thighs while straining. She delivered spontaneously and had tonic-clonic seizures following delivery. Even though she was promptly shifted to neuro-ICU, a CT scan revealed fronto-parietal hemorrhage from an AV malformation. She expired within 2 hours of admission in ICU.

### Road traffic accident

There were 3 cases of which only one was reported to the CRMD. This case of polytrauma occurred to a 26 wks pregnant woman who was a passenger in the vehicle. When pregnant women begin to drive more commonly, contribution from accidents is bound to increase. At present this does not seem to be a major concern.

## Anaemia

There were 11 cases assigned to this cause but only 4 were reported to CRMD. We included only those cases where anaemia was the primary cause. There is no doubt that a low haemoglobin level would have contributed to the bad outcome in many cases who finally succumbed to PPH. A case of aplastic anemia presented like this.

## Aplastic anemia complicating pregnancy

### Case report

A para 2 with 2 living children attended primary care center with symptoms of severe anemia. A diagnosis of aplastic anemia was made at that time and 4 units of blood were transfused and was discharged home. She presented 4 months later with symptoms of cardiac failure to another hospital where after transfusion of 1 unit of blood was referred to a tertiary centre. She reached the tertiary care centre in gross cardiac failure. Even though the patient was managed

by an expert team of doctors in a well equipped centre she expired 24 hours after expelling a dead fetus.

Appropriate counseling , early referral to a well equipped centre for termination of pregnancy, good follow up and management of aplastic anemia could have saved the patient. As the details of the case are not available we are not sure whether poor socio economic status or improper counseling was the cause of patient not reaching the tertiary care centre at the proper time.

## Chapter 15

# Miscellaneous

## Suggestions to improve Antenatal Care

**Dr.P.K.Syamala devi**

on behalf of the editorial board

### Antenatal care : key recommendations

- Routine antenatal care definitely reduces maternal as well as perinatal mortality and morbidity by bringing down the complications of pregnancy, labour and puerperium.
- Pre pregnancy counseling should be encouraged in the following situations:
  1. Medical disorders complicating pregnancy  
(diabetes, cardiovascular disease, epilepsy etc.)
  2. Women above the age of 35.
  3. H/o previous congenital anomalies, recurrent pregnancy loss etc.
- Aim- 100% coverage
- Strategy - To identify high risk pregnancy

Look for any risk factors already present or sets in as pregnancy advances. Risk factors should be highlighted in the case record and further antenatal check up should be done by senior obstetrician only.

### When to start Antenatal Care ?

Conventionally by 16 weeks onwards, but one in first trimester has to be considered.

Importance of first trimester examination.

To diagnose pregnancy

Helps dating of pregnancy

To rule out ectopic pregnancy.

## Number of visits

First visit in the first trimester, minimum two in the second trimester and at least two in the third trimester.

## Routine investigations.

At first visit-

hemoglobin

Blood grouping and Rh typing

RBS(high risk for GDM should have GCT or

GTT as required)

HIV screening

HBs Ag screening

VDRL

Urine routine

GCT between 24 and 28 weeks

After 34 weeks repeat Hb,GCT, and urine analysis

Investigations should be modified according to pregnancy complications.

## What to do at each visit?

Enquire for any symptoms

Record weight and blood pressure

Measure symphysio-fundal height in centimeters

Calculate the EDD at the earliest, if necessary using USS. Once reliable EDD is worked out, do not change it in later weeks based on sonologist's report of a small baby.

At each visit look for corresponding growth

## What about routine USS during pregnancy?

At least one USS at 16- 20 weeks helps to avoid mistaken dates and rules out major congenital anomalies.

Further USS as and when required

**What medications**

Inj.tetanus toxoid two doses at 4 to 6 weeks apart.

First dose at first visit

For 2<sup>nd</sup> pregnancy with in 1 year one booster dose

Folic acid (0.5 mg) may be given through out

Iron ( 100 mg elemental iron ) daily starting at 16 weeks of pregnancy

Calcium 500-1000 mg daily from 20 weeks.

(Iron and calcium should not be taken together)

**Antenatal records**

Should be concise and readily available when she comes in labour. Better left with the patient so that it is available to the obstetrician in whichever hospital the patient seeks admission.

**Antenatal classes**

About diet and exercise during pregnancy

Various aspects of pregnancy, labour, puerperium,

Care of breast, breast feeding and family welfare measures should be included

## Intrapartum Care

**Dr. S Ajithakumari**

On behalf of Editorial Board

- 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. Her prenatal records should be reviewed. Special points highlighted should be noted.

- Assess the fetal condition

Intermittant auscultation of FH every 15mts for 1mt in the first stage of labour and every 5mts for 1 mt in the second stage of labour is adequate for low risk labours.

- EFM remains the main stay for intrapartum fetal monitoring in high risk labours.
- A CTG admission test picks up the subtle changes in FHR such as shallow decelerations and poor variability.
- 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 ROM 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.
- 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 >6 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 1/5<sup>th</sup> 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 CS 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.



- Our aim should be to avoid complete perineal tears, but at the same time number of episiotomies also should come down. In the bargain, 1<sup>st</sup> and 2<sup>nd</sup> degree tears may increase and should be accepted.
- Active management of the 3<sup>rd</sup> stage of labour helps to prevent PPH.
- Methergin 0.2 mg IM is 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 IV 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 or oxytocin 5 units +methergin 0.2mg can be given IM. Oxytocin could be preferred because it is effective in 2-3 mts after injection, has minimal side effects and can be used in all women. Prostodin 125microgm IM, Misopristol 600mg rectally etc. are other methods to reduce PPH.
- Before delivery of placenta, make sure that it is separated from the uterine wall. While pulling on the cord, the left hand should hold the body of the uterus to prevent inversion.
- Immediately after delivery of the baby, while waiting for separation of the placenta, check for any tears of upper vagina or cervix.
- While suturing episiotomy, bring back proper anatomical alignment. Take care of apex and any active bleeders and use thick bites of the edges.
- A careful inspection of the lower vagina and a rectal examination are advisable before leaving the patient.

## Postnatal Care

**Dr. Sareena Gilvaz**

on behalf of the Editorial Board

### Key Summary Points

- Majority of deaths occur after delivery, thus emphasizing the importance of vigilance in this period.
- Many of the post natal deaths may be the result of problems which initiated antenatally or intrapartum.
- There is often a tendency for caregivers to relax vigilance once delivery has taken place. This has to be guarded against.
- Even though infection doesn't appear to be a direct contributor to maternal deaths in the present series, the importance of maintaining aseptic practices in the labour room and theatre cannot be over emphasised
- There are many established local practices which are harmful, example restriction of fluid intake and physical activities. Only education and persuasion starting from antenatal period can overcome these.
- The importance of early and exclusive breast feeding in the interest of the mother and baby has to be emphasized. However where needed, alternative feeding support for the newborn should be provided.
- Postpartum period is known for emotional swings popularly known as postpartum blues. Adequate emotional and medical support should be provided at this time. This is all the more important for the first time mothers.
- The change in the society like transition to nuclear families has impact on postnatal support. These issues have to be brought to the attention of the society.



# Appendix



## Appendix A

### List of Executive Committee Members 2004 -2005

<i>Chairman</i>	: Dr. V P Paily
<i>Secretary</i>	: Dr. K.Ambujam
<i>Treasurer</i>	: Dr. Lola Ramachandran
<i>Co-ordinator</i>	: Dr. Sheela Paily
Dr.Prema Deputy DHS	Dr. S Samantha Bhadran
Dr.Ajitha Kumari	Dr. P K Sekharan
Dr.Mrs Elizabeth Iype	Dr. P K Syamala Devi
Dr.T Narayanan	Dr. NS Sreedevi
Dr. V Rajasekharan Nair	Dr. K A Sreenivasan
Dr.CK Rajagopalan	Dr. Sulekha Devi

## Appendix B

### List of Obstetrician Assessors

Dr.Rani Santhakumari	Dr.CP Vijayan	Dr.N Syamala
Dr.Pramod Roy John	Dr.Kunjamma Roy	Dr.Linnie Eapen
Dr.Alice Jose	Dr.Balachandran	Dr.Valsamma Chacko
Dr.Gracy Thomas	Dr.G Rajalakshmi	Dr.Saravanakumar
Dr.Rajammal	Dr.T N Rajalakshmy	Dr.C Nirmala
Dr.Rajamma John	Dr.P V Jose	Dr.Krishnakumari
Dr.Suseela Bai	Late Dr.E K Varghese	Dr.Pushpa Bhat
Dr.Jayasree Thankachi	Dr.Lalithambika	Dr.Rasheeda Beegum
Dr.Leena G Pai	Dr.A Malathy	Dr.Jayanthi Raghavan
Dr.Hema Warriar	Dr.Ajith	Dr.V K Chellamma
Dr.Sareena Gilvaz	Dr.Hariprasad	Dr. Jyothi Ramachandran
Dr.Vasanthi Jayaraj	Dr.K Lalitha	Dr. K Sulochana
Dr.A V Ramachandran	Dr.Sheela Shenoy	Dr.Sangeetha Menon
Dr.D Radhamoni	Dr.Presannakumari	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.Rajagopalan

## Appendix D

### List of District Coordinators

Dr.Pushpa Bhat, Dr.Hariprasad, Dr.NS Sreedevi, Dr.Hema Warriar  
Dr.Kunjumoiden, Dr.Ambujam, Dr.Gracy Thomas, Dr.Kunjamma Roy  
Dr.M J Koshy, Dr Rani Santhakumari, Dr.Usha, Dr.Sathya Babu  
Dr.Omana Madhusudhanan, Dr.Radhamoni, Dr .Beenakumari



## Form A

Name of Deceased  
Age :  
Hosp.no. :  
Date & time of death :

Name of husband :  
(If unmarried, widow or divorced,  
note that and enter name  
& address of father)

Address  
(Husband's)

District in which husband's  
residence is situated :

Name of the doctor in charge :  
(where she died).  
Address of the doctor in charge :

Contact Telephone No. :  
E-mail address

Address of the hospital :  
(where she died)

**If referred from another centre, details of the referring centre:**

Name and address of the  
referring doctor. :

Name and address of the  
referring hospital :

Signature of the reporting doctor

Please send to  
Dr. VP Paily  
Coordinator CRMD  
Vakkanal House, East Fort,  
Thrissur- 5  
Tel No 0487 2336222







Status on admission	Ectopic		Was her condition?	Dead on arrival	
	Miscarrying/aborting			CPR needed	
	Post miscarriage or abortion			Critical	
	Antenatal			Worrying but stable	
	In labour			Stable	
	Delivering/third stage			Other: please specify	
	Postpartum				

Period of Gestation on admission

### 8. Details of antenatal care (ANC) (Details to be obtained from ANC record with the patient if necessary or available)

Did she receive antenatal care  How many ANC visits?   
 Y/ N/ NK (if known)

(not known)

Where did she receive ANC

Primary level		Government	
Secondary level			
Tertiary level			
At doctors private consultation			
Other :			

Private	

Who provided ANC ?

Consultant		Medical Officer/GP
Registrar/ Junior Dr		Midwife
		Nurse
		TBA

Other: please specify

Please provide a short summary of relevant past medical history (eg hypertension, immune disorders, thrombosis, heart disease, diabetes etc.)

Please provide a short summary of past obstetric history and any previous problems

Please identify any antenatal risk factors present in present/recent pregnancy with Y/N in relevant box

	Y	N	
Hypertension			HIV status
Proteinuria			
Glycosuria			T.T. immunisation
Abnormal lie			
Severe anaemia			
Previous C/Section			
Other: please specify			

Please provide short summary of ANC including any medications (prescribed or otherwise)

--

### 9 . Intrapartum care

Had the woman delivered before arrival? 

Y	N
---	---

If yes where?

Tick one

Home	CHC clinic	Level 1 Hospital	Level 2 Hospital	Level 3 Hospital	Other Specify	Tick one	Government	Private

### Details of labour:

	Hrs	Minutes	Was a partogram used?
Duration of labour	First stage		
Second stage			
Third stage			

Who supervised her labour?

No-one	Registrar or junior
TBA	GP/other Dr
Nurse	Other: specify
Midwife	
Consultant	

Type of delivery

Tick one

Undelivered	
Died during delivery	
Vaginal vertex unassisted	
Vaginal breech	
Vaginal assisted	
Caesarean section	

Person delivering

Specialist	
Specialist in training	
Other doctor	
Nurse/midwife	
Other: State	

Forceps

Vacuum

Elective

Emergency

If Caesarean Section state time (hrs and minutes) from decision to perform to actual delivery of baby:

Hrs

Mins

If there was a delay of more than 30 minutes what was this due to?

If assisted vaginal delivery, describe any problem or complications associated with it.

If caesarean, describe any problem or complications associated with it.

### 10. Neonatal outcomes

Baby(ies)	Gestation	Birth weight (gms)	Sex	Live birth M/F	Early neonatal death (within first 7 days)	Stillbirth
1						
2						
3						

Please provide any other relevant observations on labour or delivery

### 11. Anaesthesia

GA

Epidural

Spinal

Local

Status of anaesthetist (please circle) Consultant/ Registrar/Junior/Other

**12. Postnatal problems**

Please describe any postnatal problems including pyrexia, PPH, retained placenta

Was Post Mortem performed?

Y	N
Y	N

If performed please attach an anonymised copy of the report

Is there a police case registered?

**13. Results of any pathological investigations (please attach anonymous copies)**

**14. Please describe the involvement of any other specialists (eg nurse/midwife, anaesthetist). If their involvement was significant please ask them to provide, and attach, a brief anonymous statement of their actions.**

**15. Your case summary. Please supply a short case summary of the events leading to her death.**

16. Can you think of any steps/actions, which if taken earlier, might have prevented this death?

17. Any avoidable factors you could identify?

18. If you were treating this case again what changes would you make that will help to avoid maternal death?

19. What else would you recommend for avoiding maternal deaths in similar circumstances?

*Please return to* **Dr. V.P. Paily,**  
Vakkanal House, East Fort, Thrissur-680 005, Kerala, India  
E-mail : [vppaily@sancharnet.in](mailto:vppaily@sancharnet.in)

**TO BE KEPT CONFIDENTIAL AT ALL TIMES**  
**Assessor's Form**

Code No.

**PRIMARY(underlying) CAUSE OF DEATH**

**Your Summary of the case**



Please provide short assessment of any remediable actions that might have been taken to improve the outcome:

What, if any, were the good aspects of management of this case:

What general recommendations might emerge from this case?

Signature

Name of assessor  
Address

Date of Despatch to State coordinator

## Letter to Reporting Doctor

Date

Dear Colleague,

I regret to note that a maternal death has occurred in your centre. I am aware how distressing it is to have such an experience. The Kerala Health Services and Maternal Fetal Medicine Committee of Kerala Federation of Obstetrics and Gynecology have jointly instituted a system of Confidential Review of Maternal Deaths (CRMD).

Let me thank you in advance for cooperating with this review of maternal deaths. As you may be aware this is aimed at identifying the actual state of affairs regarding maternal deaths in our State and not to find fault with anybody. We will do everything possible to keep this confidential and not to reveal the identity of the patient or concerned doctor.

The form is to be filled up by the person who is likely to give maximum useful information about the patient. In larger departments more than one doctor would have been involved in the care of the patient. It is expected that the doctor in charge of the patient will fill up the form. He/She may discuss with other colleagues. If doctors from other departments had major part in the care of the patient, they may be asked to provide written report along with this form without revealing the name but only indicating the designation.

Please attach a photocopy of the case records including nurse's records along with the reports. While taking photocopy, the name of the hospital, doctors etc may be covered with paper so that identity of the case will not be revealed. To reduce bulk, lab results may be compiled with dates and submitted rather than photocopies of the reports.

If the patient was referred to you and any significant part of the care was from the peripheral team, that may be indicated in the report so that we can contact him or her for further details.

While filling up the form, please remember that as much useful information as possible is given.

The completed form may be returned to me in the envelope provided by registered post or courier latest within 2 weeks of this letter.

With regards,

Yours sincerely,

Dr. V.P.PAILY  
State Coordinator CRMD, Kerala.

## Instruction to Assessors

To

The Assessors of Confidential Review of Maternal Deaths (Kerala)

Dear.....,

I am forwarding herewith the data collection form and the other relevant materials received regarding a case of maternal death. Please study the case in detail and arrive at the possible cause, especially with a view to identify any remediable factor. The aim is to learn lessons from this tragedy to prevent recurrence of such mishaps in the future.

If you think the care was suboptimal, please indicate that with details so that we can point out that in our final report. Also some of the treating doctors would like to have feedback for future improvement.

Please give special attention to cause of death. The explanation I have given to the treating doctor is as follows-

**[Cause of death:**

**Cause of death is divided into two –primary (underlying) and final. An example: A patient developed eclampsia and subsequently died due to cerebral haemorrhage. The primary cause can be Eclampsia and the final cause cerebral haemorrhage. Another example can be Abruptio placentae leading to Disseminated Intravascular Coagulation shock and multiorgan failure leading to death. The primary cause is Abruptio Placentae with DIC and final cause Shock and Multiorgan failure.**

**Since the final mode of death in any patient is by the cessation of cardiac activity “cardio respiratory arrest “does not convey any useful information as a cause of death.**

**Sometimes it may be difficult to split cause of death into primary and final in which case assign a cause which you think is most appropriate.]**

Please treat all documents as confidential and do not reveal the contents with identity to anybody. Return all the papers to me after assessment as it has to be burned after assessment is over.

Kindly return the completed report at the earliest, in any case within 2 weeks.

Finally, I know how precious your time is. I can only **thank you** on behalf of pregnant women and their families in our State for cooperating in this venture to eradicate avoidable maternal deaths.

Yours Sincerely,

Dr. V.P. Paily  
State Coordinator

## Appendix F

### Feedback form

Dear colleague,

We thank you for taking time to go through this report and would be grateful for your feed back. We would appreciate your critical comments and suggestions for improvement.

Feedback form

Your personal details (optional)

Name

Designation

Contact address

email

Telephone

Did you find the information in the book useful?

Do you have any suggestions to improve the following

a. Data collection

b. Assessment

c>Your suggestions regarding future CRMD reports.

d. Any other suggestions

Signature

*Please return to* **Dr. V.P. Paily,**  
Vakkanal House, East Fort, Thrissur-680 005, Kerala, India  
E-mail : vppaily@sancharnet.in



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One of the many meetings of assessors to review the cases



Participants of the workshop at Thiruvananthapuram that started off the CRMD



**Maternal Fetal Medicine Committee**  
**Kerala Federation of Obstetrics & Gynaecology**