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CASE SNIPPETS

Dr. Aswathkumar
President KFOG

Dr. Venugopal
Secretary KFOG

PRESIDENT'S MESSAGE

Dear Colleagues and Friends,
Greetings from the Kerala Federation of Obstetrics and Gynaecology (KFOG)!

I am delighted to present to you the second edition of the KFOG case snippets for the year 2023. Case reports are much more interesting and gives you practical information in short sessions.
I would like to express my heartfelt gratitude to Dr. Shyama Devadasan, the editor, for her commendable work in bringing out this edition. I also appreciate the efforts of the authors for their valuable contributions.
I hope you will find this edition of the KFOG case snippets informative and useful for your clinical practice. Thank you.

Dr. Aswath Kumar
President KFOG (2023-24)

EDITOR'S MESSAGE

"**Learn continually. There's always "one more thing" to learn.**" - Steve Jobs

It gives me immense pleasure to introduce the second issue of KFOG Case Snippets showcasing relevant cases of practical importance. I thank each one of the authors, for their timely submissions to the journal. I warmly welcome the participation and support of every reader with their valuable contributions. Kindly send them along with any queries to drshyamadevadasan12@gmail.com

With regards,
Dr. Shyama Devadasan
Editor, KFOG Case Snippets

INDEX

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CHAPTER 01

AN UNUSUAL OVARIAN MASS IN A YOUNG GIRL

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Introduction

Ovarian tumours in children and adolescent girls form an uncommon but important part of gynaecological malignancies. They account for 8% of all abdominal tumours and 1% of malignancies in children. It is estimated that almost 10-30% of all the ovarian neoplasms occurring in girls up to 17 years of age are malignant. Tumours of the ovary are divided into neoplastic and non-neoplastic processes. While the non-neoplastic conditions include corpus luteal cysts follicular cysts, and endometriotic cysts, the neoplastic processes include both benign tumours like mature cystic teratomas and highly malignant tumours like yolk sac tumours. According to literature, paediatric ovarian tumours mostly belong to the group of germ cell tumours in contrast to the tumours occurring in the adult females, which are mostly surface epithelial in origin. Since ovarian cysts are thought to arise from mature follicles, these tumours were considered to be infrequent in the paediatric population but now the trend is changing.

Here is a case of a massive ovarian tumour in a very young girl whose clinical findings turned out to be quite unusual.

Case Report

A 12 year old girl presented to the OPD with one episode of abdominal pain which had started 2 days back. The pain had subsided within a day. There was no history of vomiting or fever. She had attained her menarche 8 months prior and her cycles were irregular (3/45-60 days duration). She had no other illnesses or past history of surgeries. She had been told to have an abdominal mass and had come for a second opinion.

On examination, the girl was of average height and weight. Abdominal examination revealed a mobile mass arising from the pelvis corresponding to 30 weeks size. It was non tender.

USG revealed a 29 X 20 cm mass with solid and cystic areas, possibly arising from the ovary. Uterus was normal. There was no ascites or renal hydronephrosis.

Blood samples were sent for tumour markers and a contrast CT was done. Serum levels of tumour markers were normal (Serum AFP, BhCG, CEA and LDH) . Serum CA 125 was 158 IU/ml and Serum CA 19-9 was 68.67 ng/ml. CECT revealed a large well

capsulated heterogeneous abdomino-pelvic mass with enhancing solid areas and cystic components with loculations, dense calcifications and fat components measuring 24X18X10 cm. Enhancing vessels were noted within the mass. There was no evidence of any ascites or loculated collection in the peritoneal cavity.

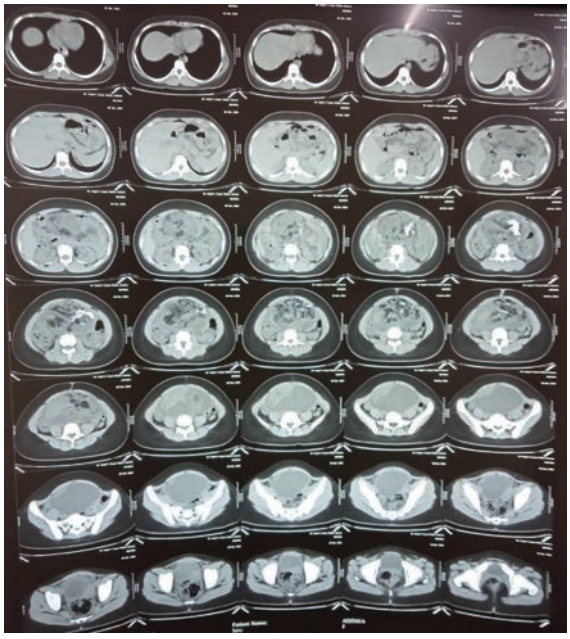

Figure 1. CECT Abdomen and Pelvis

A provisional diagnosis of Ovarian mass of possible malignant etiology was made with a differential diagnosis of Ovarian Teratoma. She was posted for an exploratory laparotomy followed by unilateral salpingoovariotomy with pelvic lymphadenectomy (if required). The surgery was performed with the help of an onco-surgeon in view of suspected malignancy and young age of the patient.

Under General + Epidural Anaesthesia, the abdomen was opened by a midline vertical incision extending above umbilicus. A large solid and cystic tumour of 30x20 cm was visualized arising from the left ovary. The mass had an irregular capsule with hyperemia. The capsule was found to be ruptured on the superior aspect. There was no ascites. Uterus and right ovary was normal. The peritoneum and omentum were studded with what appeared to be miliary deposits. There was no bowel matting or omental caking.

Peritoneal wash was taken and sent for cytology. Left salpingoovariotomy was done without tumour rupture. Infra-colic omentectomy was done. Peritoneal biopsy was taken from multiple sites. A suspicious area in the anterior abdominal wall was also resected and sent for HPE. One unit of PRC was transfused intra-op.

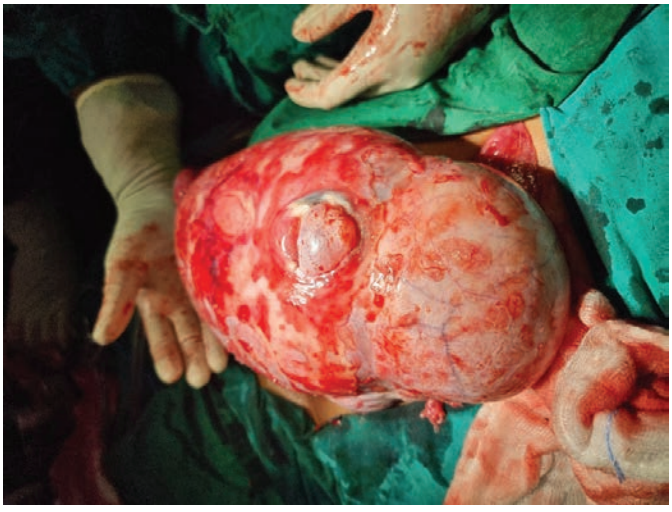

Figure 2. Ovarian mass with ruptured capsule. Note the loop of hair around the rupture site

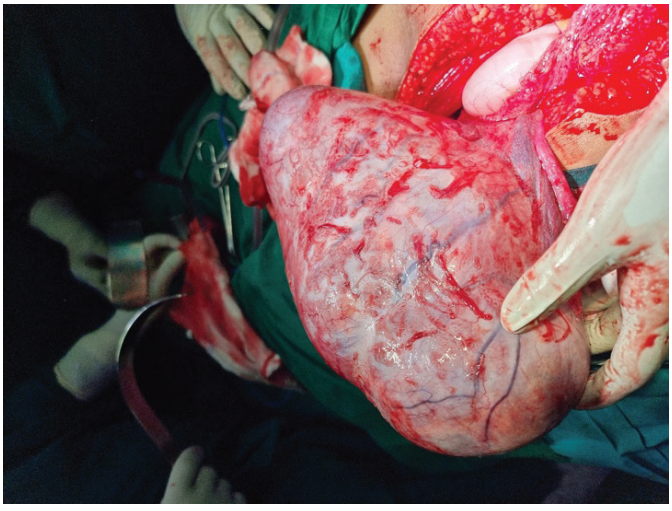

Figure 3. Ovarian Mass with miliary deposits on the infundibulo-pelvic Ligament peritoneum

The ovarian mass was cut open and inspected after removal. It contained well developed lungs, brain, liver and limb appendages. There were clumps of hair and thick sebaceous material also. The diagnosis appeared to be dermoid cyst with possible peritoneal miliary tuberculosis which could account for the peritoneal deposits and elevated CA 125 levels. Since the mass appeared to be an ovarian teratoma, possibly benign, lymphadenectomy was not performed. Abdominal wash was given and the incision closed in layers.

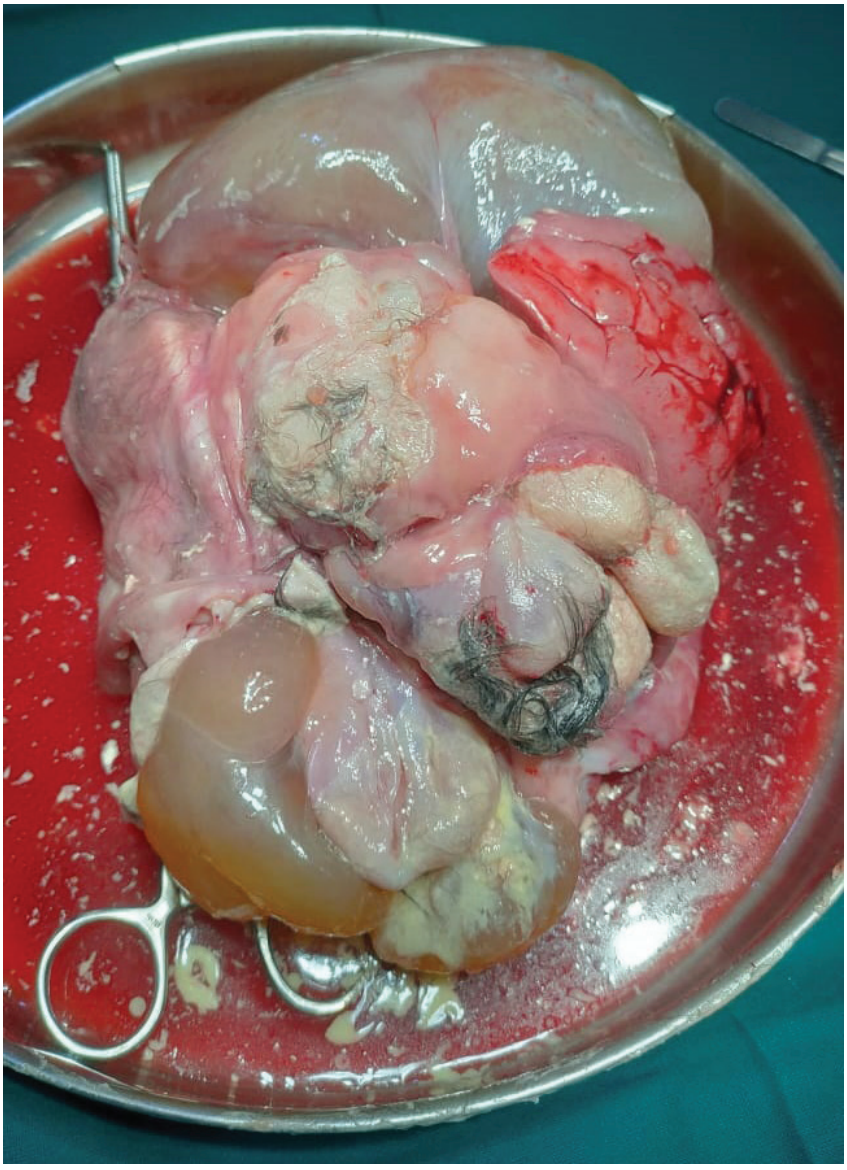

Figure 4. Cut open view of ovarian mass with features s/o dermoid cyst

The patient did well post operatively. Ryle's tube and CBD were removed on POD1. She was discharged on POD4. There was no evidence of malignant cells on cytological assessment of peritoneal fluid. She was reviewed after 2 weeks in the OPD when all stitches were removed.

The histopathology report came as Mature Cystic Teratoma with Gliomatosis Peritonei (GP). The latter was a surprise as it is unusual to be associated with mature teratoma. The patient went back to attending school and is on follow-up. She got her periods 2 weeks post surgery. She is doing well at present.

Discussion

It is unusual to have a mature cystic teratoma of more than 30 cm in size. Larger sized masses go more in favour of immature teratoma. Yet, this has to be one of the largest mature teratomas ever operated upon, in literature. Although there was the possibility of a mature teratoma, the size and the high CA 125 levels misled us. Hence it is always important to keep an open mind.

Gliomatosis Peritonei refers to the implantation of glial(neuronal) tissue on the peritoneal surface. Its peak incidence is in the second decade of life. It is usually associated with immature teratomas. It is rare for GP to be associated with mature teratomas. GP is usually composed of miliary-like greyish nodules of mature glial and neuronal tissue without any other teratomatous component on the peritoneal surface and omentum. Hence, it is considered a benign process. It is a rare finding and less than 100 cases have been reported in the literature.

Though the origin of GP remains uncertain, two theories to explain its etiology have been proposed. According to the first theory, implantation of glial tissue in the peritoneum occurs either through rupture of the capsule with subsequent implantation of teratomatous tissue or via lymphatic spread as in metastasis of carcinomas. The second theory is that, pluripotent stem cells in the peritoneum or subjacent mesenchyme undergo glial metaplasia. In our case, there was a capsular rupture and the presence of brain tissue inside the teratoma confirmed our hypothesis. The GP must also have been the reason for the elevated CA 125 levels.

At imaging, GP is seen as the nodular enhancement of the peritoneum in CT, indistinguishable from conventional metastases by imaging alone. Grossly, glial tissue implants are 1–10 mm in size, usually less than 3mm, without any fatty component making them grossly indistinguishable from tuberculous peritonitis and peritoneal carcinomatosis, and therefore, the diagnosis is made by histopathological

examination of the implants. Omental caking and ascites can be observed in some cases.

Pathologically, GP is considered as a grade 0 teratoma, according to the WHO grading system used for immature teratoma. It usually has a favourable prognosis and is managed conservatively. Further, because the lesion is often extensive, complete resection is usually demanding and impractical. Fortunately, residual GP deposits are asymptomatic and may remain inert over a long period or may disappear over time. The presence of GP, regardless of its extent, is usually not associated with adverse outcomes. However, GP has been reported to transform into malignant glial neoplasms. Thus, the treatment decision is based on the grade of the primary tumour and not the glial tissue implants, provided they are extensively sampled and all are mature. However, if immature glial tissue or other teratomatous components or both are present in the peritoneum or omentum, the treatment should be the same as for metastatic immature teratoma.

Even though the prognosis of patients with GP is excellent, long-term follow-up, even in the face of mature peritoneal glial implants, is highly recommended. This is because of the established cases of malignant transformation of the glial components long after the initial surgery. The patient should be ideally followed up with FDG PET CT. However, as this condition is rare and sample sizes are inadequate, there is no widely accepted guidance as to how long and by which means these patients should be followed up.

Conclusion

Gliomatosis Peritonei in mature cystic teratoma of the ovary is a rare occurrence. Vigilant conservative management with continued clinical evaluation and long-term follow-up is paramount. Moreover, it is pivotal to recognize the benign nature of the peritoneal seedlings that can avoid unnecessary extensive surgery, especially in young girls.

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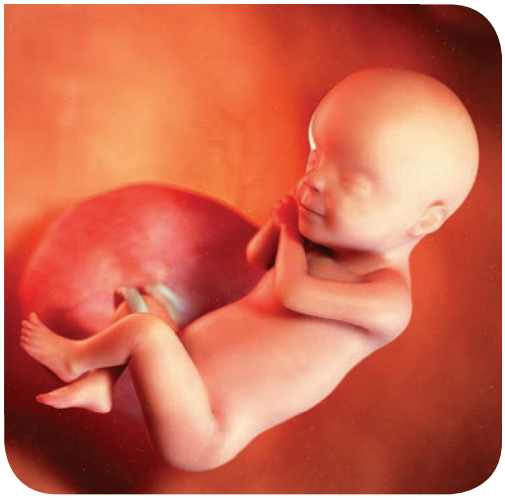
CHAPTER 02

INTUSSUSCEPTION IN A 30 WEEKS PREGNANT WOMAN



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INTRODUCTION

Intussusception is a rare but potentially life threatening complication in pregnancy challenging to both gynaecologists and surgeons. Its diagnosis is difficult and emergency surgery indicated.

CASE REPORT

30 year old G2P1L1, previous FTND, was referred from local hospital at 30 weeks 5 days in view of persistent abdominal pain. She gives a history of acute onset of severe cramp like abdominal pain.

On examination, vitals were stable. Abdominal examination revealed a relaxed non-tender uterus corresponding to 30 weeks gestational age with good fetal heart sounds. Speculum examination showed a long cervix with os closed. Initial blood investigations were normal.

Emergency USG was done- Single live intrauterine fetus with adequate growth and liquor volume. Cervix long ,os closed, an anterior wall fibroid 6.1 x3.9 cm was seen, no other pathology identified.

She was given symptomatic treatment. But next day her symptoms aggravated and a mass of 8x6 cm was palpable in right hypochondrium. Emergency surgery consultation was sought for.

Emergency USG showed evidence of intussusception like appearance with multiple loops extending from RIF to right hypochondrium causing bowel obstruction with dilated proximal bowel loops filled with fluid, adjacent edema of bowel wall was seen. She was shifted to SICU and Ryle's tube aspiration was done.

She was taken for EMERGENCY EXPLORATORY LAPROTOMY in view of intestinal obstruction.

Intra op findings:

There was an ileo ileal intussusception 2 m from DJ flexure with ~180 cm bowel inside ileum. Impending perforation with gangrenous segment ~180 cm of ileum was seen with minimal ascites. A gravid uterus ~30 weeks gestation, tubes and ovaries were normal. The large bowel and liver appeared normal. An ileal segmental resection (180cm) with end to end anastomoses was done.

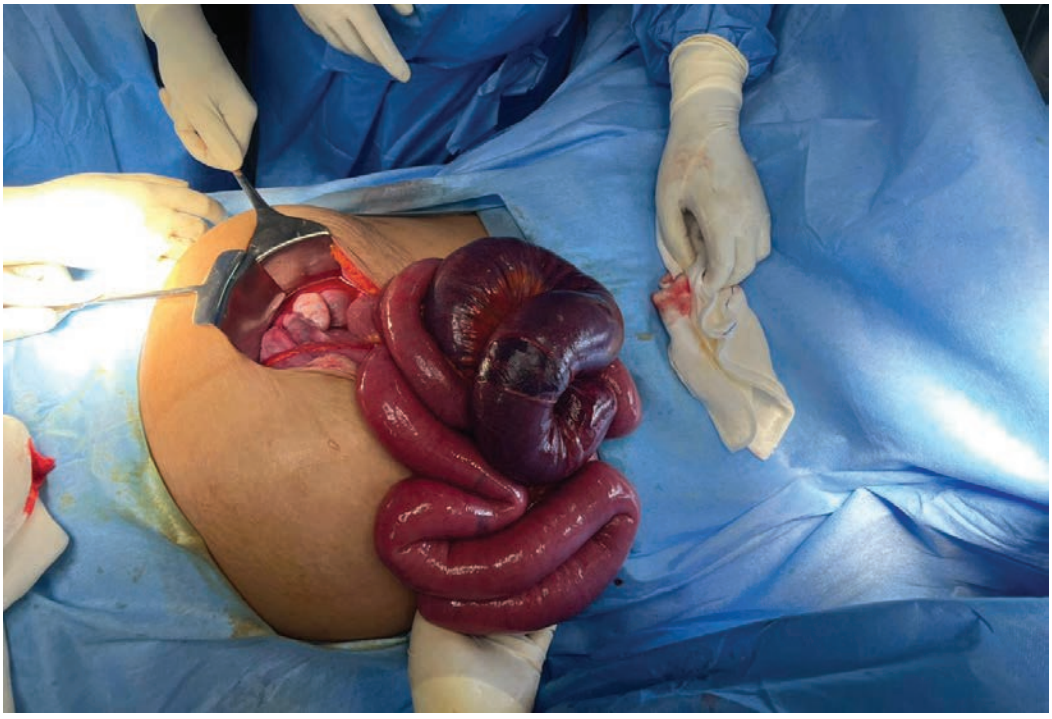

Fig 1. Impending perforation and gangrenous ileal segment

Post operative period was uneventful. Fetal well-being was confirmed by USG post operatively. She was shifted to gynecology ward from the surgery ward on post op day 7 and sent home on day 14. She was admitted with spontaneous labour pain at 38 weeks 2 days and delivered a live healthy male baby weighing 2.6 kg. Mother and child was discharged on day 3.

HISTOPATHOLOGY

A segment of small bowel 100cm long with ischemic necrosis seen with the resected bowel ends showing edema, congestion, serositis. The mucosa was viable. Polypoidal intramural mass 3.4x 3.2cm toward one resected end was a submucosal lipoma. Separately received segment of small bowel 15cm showed congestion and serositis.

DISCUSSION

Intussusception is a rare but serious complication in pregnancy necessitating emergency surgery. Even though the patient is usually admitted under the care of the gynaecological team it is the surgeon who shall make the final diagnosis and ultimately to operate. Intussusception is most common in childhood accounting for 15% of bowel obstructions. Intussusception in adults accounts for only 5% of the total number of intussusceptions and only 1% of cases of bowel obstruction. Its incidence is 1/30000 in hospital admissions or 1/3000 of all abdominal operations. The mean age at presentation for adults is between 51 - 54.4 years. Symptoms on presentation are most often secondary to obstruction and include nausea, vomiting, constipation, abdominal pain and in contrary to childhood intussusception where a leading lesion is found in up to 95% of cases. These can be of many different aetiologies in adults including carcinoma of the colon, meckel's diverticulum, submucous lipomas, Peutz Jegher polyps, leiomyomas, neurofibromas, adenomas, heterotopic pancreatic tissue and many others.

The imaging modalities of choice in adults are the CT/ MRI scan and the double contrast enema. Their diagnostic yields are 52%-58% and 41%-73%, respectively. The abdominal X-ray usually shows features of obstruction. The classical finding on CT is the "target lesion" formed by one part of the bowel telescoped into another.

The treatment of intussusception in the adult is almost always surgery. Intussusception is difficult to diagnose at the best of times but in pregnancy even more so. Some of the presenting symptoms like abdominal pain and vomiting are common symptoms in pregnancy. Furthermore, it is not only the life of the mother that is at risk but also the life of the foetus.

Intussusception in pregnancy is rare. Intestinal obstruction in pregnancy has been reported to occur with an incidence between 1:2500-3500, most commonly secondary to adhesions

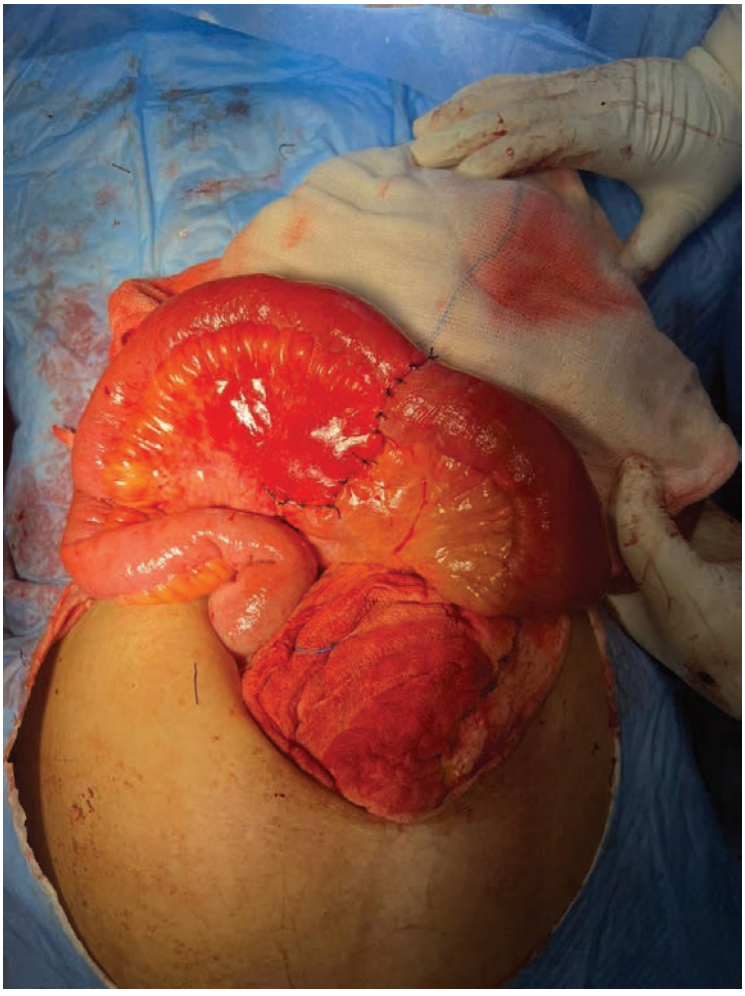

Fig 2. Ileal segmental resection with end to endanastomoses

or gastrointestinal volvulus. Intestinal obstruction in pregnancy is associated with a high maternal and perinatal mortality of 6% and 26%, respectively. Additionally, diagnostic imaging in pregnancy is restricted to USS and MRI, with MRI not available readily in many hospitals. In our case the diagnosis was made by abdominal ultrasound scan. Recent publications have shown ultrasound scan to be an useful imaging technique for intussusception in pregnancy. It can exclude many of the differential diagnoses such as a twisted ovarian cyst and does not carry the risk of radiation to the foetus.

CONCLUSION

Even though it is a rare occurrence, surgeons should be aware of the possibility of intussusception in pregnancy which needs urgent and decisive intervention. Ultrasound scanning is a safe investigation in pregnancy and frequently confirms the diagnosis.

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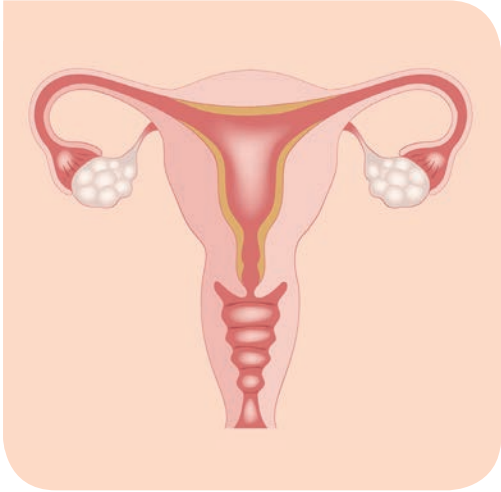
CHAPTER 03

OVARIAN COLLISION TUMOR – A RARE ENTITY



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INTRODUCTION

A collision tumor is the coexistence of two distinct tumors without any histologic intermixing in the same organ or tissue. Though these types of tumors are often seen in various organs like gastrointestinal tract, skin, lung, adrenal, central nervous system, lymph nodes, uterus etc but their occurrence is rare in ovaries. They are commonly composed of ovarian teratoma (typically mature cystic teratoma) and an ovarian cystadenoma (typically mucinous) while other histologic combinations are much less common (e.g teratoma and granulosa cell tumour, cystadenocarcinoma and sarcoma).

Collision tumors should be differentiated from composite tumors where there is intermixing of tumor lines, such as in malignant mixed Mullerian tumors. They are mostly diagnosed postoperatively with the help of histopathological examination. When an ovarian teratoma has imaging findings that cannot be explained solely by the teratoma, the possibility of collision tumor should be considered.

Adequate excision and meticulous histopathological examination need to be done for understanding the various components of collision tumors to avoid them being misdiagnosed as malignancy.

CASE PRESENTATION

A 55 year old P2L2 post-menopausal lady presented with abdominal distension and sensation of fullness in her lower abdomen since 2 months. Her vitals were stable and abdominal examination showed a large globular cystic mass of size 20 x 18 cm occupying the right iliac fossa, right lumbar region and extending up to the umbilicus. Speculum examination revealed a healthy cervix and vaginal examination showed normal sized uterus and a large cystic mass felt higher up in the right adnexa. Tumour markers were normal with CA -125 - 12.8, CEA - 1.51ng/ml, CA 19.9 – 122.1 U/ml, alpha feto protein 1.37 ng/ml.

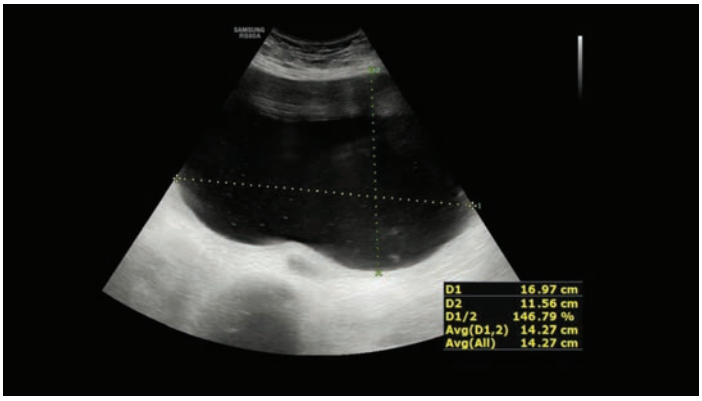

Fig 1. Ultrasound image showing a large abdominopelvic thin walled cystic lesion with multiple internal echoes, thin incomplete septations and no solid areas.

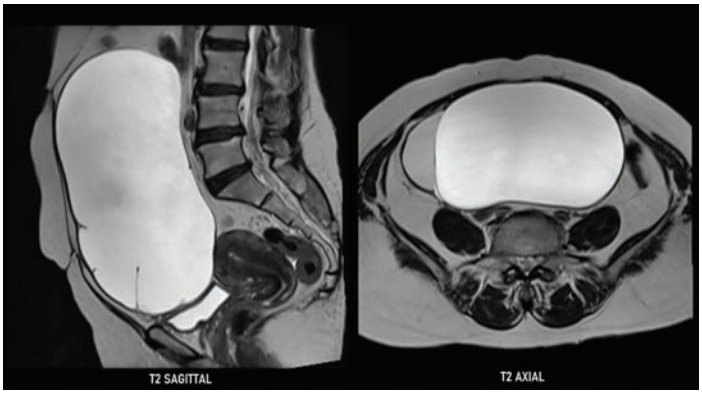

FIG 2.A T2 SAGITTAL AND T2 AXIAL MRI IMAGES

MRI showed a large abdominopelvic T2 hyperintense cystic lesion with thin incomplete internal septations. No solid areas ,thick septations or papillary projections were detected within the lesion.

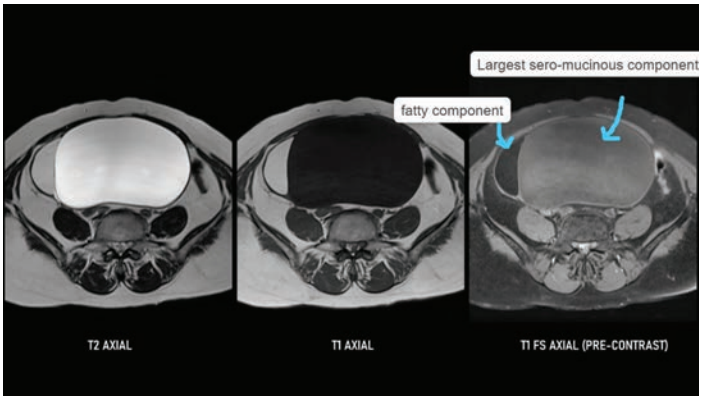

Fig 2 B T2 AXIAL, T1 AXIAL , T1 FS PRECONTRAST MRI IMAGES

The lesion showed a large T2 hyperintense locule/ component in the left side, with an intermediate T2 signal with a smaller locule in right side. The smaller locule showed T1 and T1 FS hyperintense signals suggestive of fat. Signal intensity favoured a dermoid cyst / collision tumor.

She underwent total laparoscopic hysterectomy and bilateral salpingo oophorectomy. Operative findings included a large right ovarian cyst measuring 20 x 20 cm mostly cystic with sero -mucinous fluid and a section of ovary with features of mature teratoma.

Histopathological examination revealed fibro collagenous cyst wall lined partially by mucinous epithelium and partially by cuboidal to flattened epithelium. Cyst wall also showed skin appendages, bone, cartilage, muscle, glial tissue, salivary gland tissue, colonic mucosa and melanin pigment. Foci showing hair shafts surrounded by foreign body giant cell reactions was also seen. Hence a collision tumour having a sero-mucinous cystadenoma with mature cystic teratoma was diagnosed.

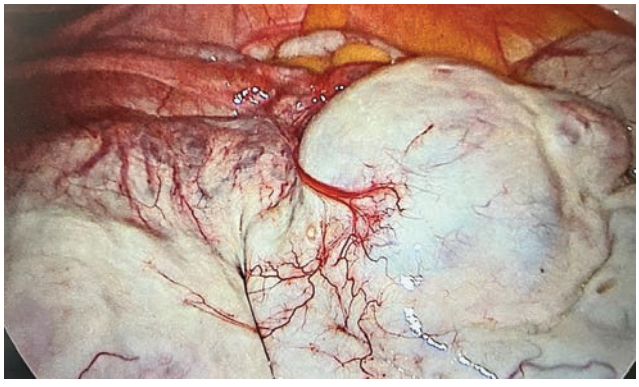

FIG 3 A. A Large seromucinous cystadenoma with a section of mature teratoma

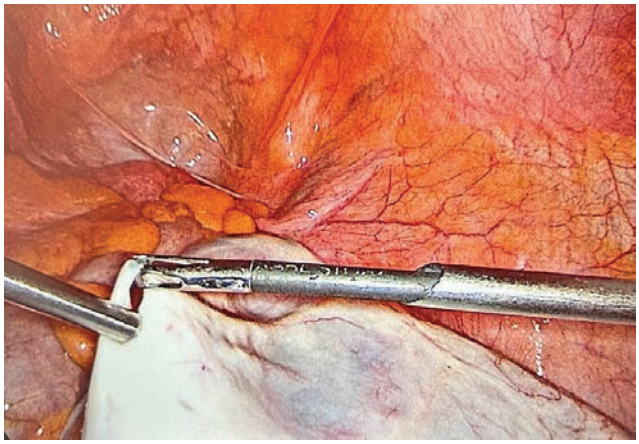

FIG 3 B - Cystic fluid being aspirated without spillage

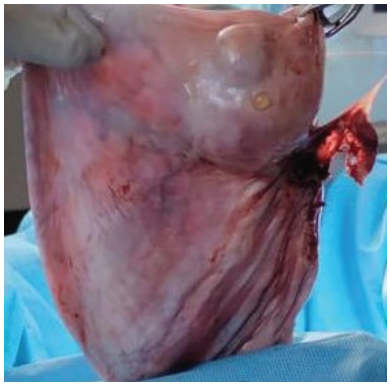

FIG 3 C : Specimen showing a large seromucinous cyst along with mature teratoma.

HISTOPATHOLOGY

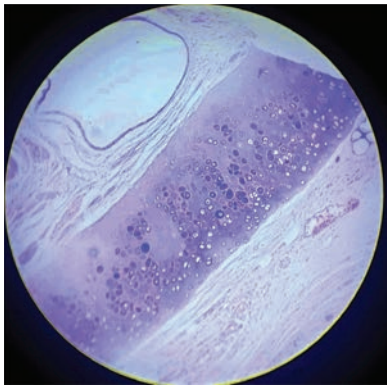

FIG 4 A - Cyst wall showing hyaline cartilage as a part of mature teratoma

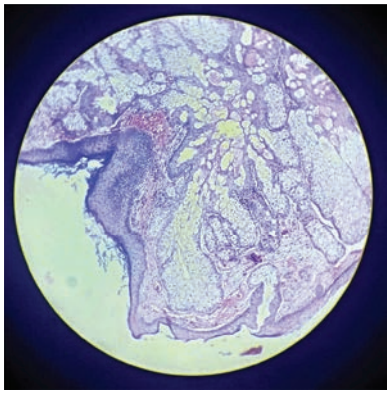

Fig 4 B Cyst wall showing skin appendages ,adipose tissue,melanin pigment,hair shafts surrounded by foreign body giant cell reaction.

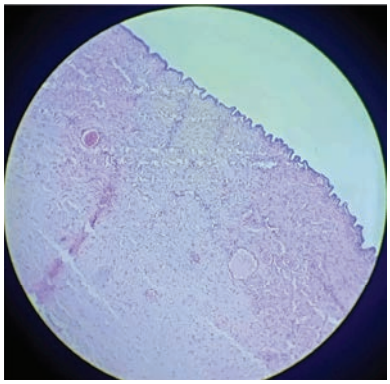

FIG 5 Cyst wall lined by cuboidal to flattened epithelium suggestive of serous cystadenoma

DISCUSSION

This is a case of collision tumor of ovary in a postmenopausal women. Although collision tumors are common in gastrointestinal tract, it is rare in ovaries. The exact etiology of this condition is not well understood. The most commonly reported combination in ovarian collision tumors is that of mature teratoma, a germ cell tumor of ovary and mucinous tumors (cystadenoma and cystadenocarcinoma) which orginate basically from the surface epithelium of the ovary. Though collision tumour with mucinous tumour has been reported, combination with sero-mucinous component is rare as in our case.

Whenever a large multiloculated cystic lesion of ovary is encountered, gynaecologists, radiologist and pathologist should be aware of collision tumor and this must be ruled out for accurate treatment. Thorough radiological evaluation is required in such scenarios with further imaging modalities like MRI.

CONCLUSION

Generally collision tumour is a histopathological diagnosis. But radiological detection is very important as it can affect the surgical management of collision tumors and hence the prognosis of the patient. The role of meticulous histological sampling and examination cannot be overemphasised as it will help to confirm the radiological diagnosis and be decisive in the further prognosis.

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CHAPTER 04

A RARE CASE OF PANCREATITIS IN PREGNANCY– IDENTIFICATION PREVENTS MORTALITY

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INTRODUCTION

Acute pancreatitis in pregnancy is a rare but serious condition occasionally causing maternal mortality. It affects approximately 1 in 1000 to 1 in 12,000 pregnancies. The spectrum of acute pancreatitis in pregnancy varies from mild to severe pancreatitis. Severe pancreatitis might be associated with multiorgan dysfunction, pancreatic abscess, pseudocyst formation and necrosis.

Diagnostic criteria in pregnancy is not defined clearly. Most common cause is secondary to gall bladder stones(70%), alcohol abuse, drugs and recently covid related pancreatitis. A very rare cause barely reported worldwide is hypertriglyceridemia (HTG) causing acute pancreatitis in pregnancy. It appears that HTG aggravates the severity score and prognosis of disease. Maternal fatality is about 40% and fetal 60-70%.

This case refers to a primigravida, with a history of familial HTG, at 33 weeks gestation who presented with acute pancreatitis.

CASE REPORT

A 22 year old primigravida at 33 weeks gestation, presented with complaints of severe epigastric pain and vomiting since 2 days. She is a known case of hypothyroidism on thyroid supplementation and pregnancy induced hypertension on single antihypertensive drug (combined alpha -beta blocker) since 2 weeks prior to admission. She has a history of familial HTG with death due to CAD in elder brother and acute pancreatitis secondary to HTG in sister.

Clinical examination revealed mild hypertension and moderate tachycardia . BMI was 20. She was conscious, oriented and afebrile, abdomen was tense and tender at epigastric area, uterus enlarged to about 36 weeks and fetal heart had severe tachycardia of about 220 beats per minute. Other systems were within normal limits.

Lab investigations were difficult as serum was lipemic. Blood count revealed neutrophilic

leucocytosis, thrombocytosis and hyponatremia. CRP ,serum amylase and lipase were elevated and triglycerides were >5000. Ultrasound was suggestive of acute pancreatitis and fetal heart dipping from 220 to 120 beats per minute.

Ryles tube aspirate was copious and bilious and urine output grossly reduced . Dehydration was corrected with IV fluids and in view of fetal distress she was taken up for emergency preterm caesarean section under spinal anaesthesia after antenatal steroids and antibiotic cover as she had an unfavourable cervix. Intraoperatively there was about 1 litre of milky, chylous ascites and blood was thick and murky. There was polyhydramnios and delivered an active male baby of birthweight 2.6 kg (probably due to undetected diabetes).

Post operatively she was kept NPO with RTA and CT on day 3 showed gross ascites, bilateral moderate pleural effusion and moderate splenomegaly with dilated large bowel loops possibly due to paralytic ileus. In view of severe HTG, plasmapheresis was done and Ca gluconate infused. Echo revealed normal biventricular function and EF of 60%. Therapeutic paracentesis was done on post op day 4 and ascitic fluid showed high protein and low SAAG ascites.

She improved symptomatically and WBC counts, CRP

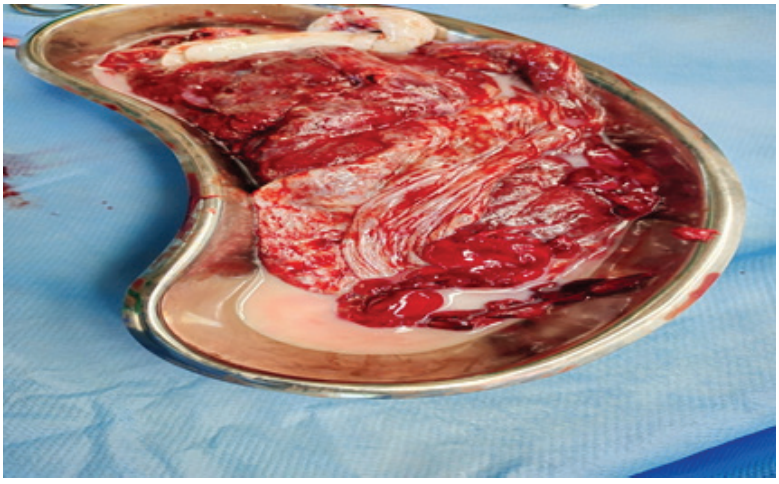

Fig 1:Placenta with highly lipemic plasma immediately after delivery by lower segment caesarean section.

,transaminase and triglyceride values were near normal.10 days later USG showed mildly bulky pancreas with loculated thick peripancreatic collection of 11x8x2 cm ,fatty hepato-splenomegaly, moderate ascites and pleural effusion. After 12 days, soft diet started and she was discharged when symptom free and haemodynamically stable and advised close follow up. 2 weeks later, peripancreatic collection reduced slightly but loculated (possibility of pseudocyst formation explained). She was given Fenofibrate and Saroglitazar.

FOLLOW UP AND SUBSEQUENT EVENTS

After 3 months into the post-partum period her organ functions were normal and lipid profile well under control. Subsequently, she presented with a second pregnancy within 1 year. She had an asymptomatic antenatal period with regular gastro medicine and cardiac evaluation. She continued on antilipidemic medication throughout. At 28 weeks ,severe lipid derangements occurred and she was started on plasmapheresis when serum Triglyceride value went above 4500mg/dl. She had lipemic skin changes like eruptions and xanthomas and mild anemia. Plasmapheresis was required once in 3 weeks and after the third pheresis and after antenatal steroids, pregnancy was terminated by elective caesarean at 34 weeks gestation and permanent sterilisation done. Both mother and neonate were stable.

The genetic analysis of the family was completed and suggestive of Type IV familial HTG with an autosomal dominant pattern with polygenic inheritance. The heterozygous mutation inactivating the LPL gene creates an inability to hydrolyse the triglycerides within the VLDL core. The firstborn infant also tested positive for the gene.

Her younger sister later presented antenatally after a prolonged period of infertility and also had a similar clinical course with derangement of lipid profile by the early third trimester and required plasmapheresis. That pregnancy was complicated by severe pre-eclamplsia and was terminated at 36 weeks.

DISCUSSION

Acute abdomen in pregnancy especially in the third trimester can be due to obstetric and non-obstetric causes ranging from minor conditions like urinary tract infections and gastritis to life threatening conditions like HELLP, placental abruption, rupture uterus etc. Acute pancreatitis requires a high index of suspicion and if undiagnosed early enough can lead to maternal septicemia, multiorgan failure, fetal distress and demise and severe maternal morbidity and mortality. The

absence of specific clinical symptoms makes it difficult to diagnose in an acute setting.

In an **acute setting** HTG-induced pancreatitis in pregnancy requires prompt management. Supportive measures and close monitoring for complications (pancreatic necrosis, adult respiratory distress syndrome, acute kidney injury, and systemic inflammatory response syndrome) should be conducted. Specifically, rapid lowering of triglyceride levels is crucial . Evidence for implementing this is currently limited to retrospective case reports with no clear guidelines available.

Therapeutic plasma exchange (TPE) - TPE lowers triglyceride levels by rapidly removing triglycerides and chylomicrons and may improve outcomes by removing inflammatory markers and cytokines . Replacement with FFP has been postulated to replace deficient LPL or apolipoproteins, facilitating the degradation of triglycerides. However, due to a lack of randomized controlled trials, TPE is considered as category III therapeutic measure for HTG-induced pancreatitis .

Intravenous insulin - Intravenous insulin therapy lowers triglyceride levels by activating LPL and increasing chylomicron degradation with an expected fall of 50–75% over 2–3 day. This is associated with shorter hospitalization stays and lower APACHE scores after 72 h of treatment. Its utility extends to non-diabetics, with frequent monitoring of electrolytes and a dextrose infusion to maintain euglycemia .

Heparin - Continuous heparin infusions could lower triglyceride levels by releasing stored LPL from endothelial cells. This potential benefit must be weighed against the risk of bleeding and haemorrhagic pancreatitis. Continuous heparin infusion may also deplete LPL, with rebound hypertriglyceridemia upon cessation. We refrained from using i.v. heparin infusion due to the potential bleeding risk during insertion of vascular catheters, and potential impending preterm labor.

In **non-acute settings**, management is usually conservative. Dietary modification remains the cornerstone of successful management. Total fat should be restricted to <20% of daily caloric intake. High glycemic index foods should be avoided since they enhance the hepatic synthesis of fatty acids. Low-fat diets pose a risk for essential fatty acid deficiency, and supplementation with oral omega-3 fatty acid should be prescribed as they are safe in pregnancy. At higher doses, these could reduce hepatic triglyceride synthesis and increase lipoprotein lipase activity, lowering serum triglyceride levels by 25–30%. There are limited data on the use of fibrates and niacin after the first trimester (category C). Gemfibrozil may be considered in very severe hypertriglyceridemia from the second trimester. However, the onset of action is gradual, limiting its efficacy in the acute setting.

CONCLUSION

HTG-induced pancreatitis in pregnancy is a rare but potentially devastating condition associated with high maternal and fetal morbidity and mortality. A multidisciplinary approach is needed in supportive management and measures to reduce triglyceride levels rapidly. TPE is the quickest method and should be considered as first-line therapy. Maintaining a very low-fat diet to prevent recurrence of pancreatitis must be weighed against the need to achieve adequate maternal and fetal nutrition. Timing of delivery is individualized according to the risk of further episodes of pancreatitis vs fetal maturity.

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