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CASE REPORT

Acute Pancreas to a Cute Pancreas

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ABSTRACT

Acute pancreatitis in pregnancy is rare, with the incidence being 3 in 10,000 pregnancies. Its occurrence is of great concern to clinicians as they are dealing with two lives and increased incidence of morbidity.

Here, we report two unique cases of acute pancreatitis in the 3rd trimester. Both presented with symptoms of vomiting and pain abdomen. One patient was a primi at 35 weeks with gestational hypertension on tablet labetalol 50 mg tds, tablet metformin for polycystic ovarian syndrome (PCOS) and thyroxine replacement for hypothyroidism. Her baseline amylase and lipase values were 157 and 475 respectively. She had emergency lower segment cesarean section (LSCS) for severe oligohydramnios. Intraoperative period was uneventful. The second patient was a primi at 34 weeks and 4 days and preterm premature rupture of membranes (PPROM). Her baseline amylase and lipase values were 1449 and 550. Patient was induced with prostaglandin E2 (PGE2) gel and delivered normally. Both patients were managed conservatively with a multidisciplinary team approach.

Keywords: Acute pancreatitis, Oligohydramnios, Pre-eclampsia.

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INTRODUCTION

Acute pancreatitis is a rare event in pregnancy.[1] The complications, such as a high maternal-fetal morbidity and mortality, have decreased due to early diagnosis and intervention. Acute pancreatitis is usually seen in the 3rd trimester or the early postpartum period.[2] Of all the causes, cholelithiasis is the commonest etiology for acute pancreatitis in pregnancy.[3]

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The most common presentations of the condition include low-grade fever, nausea, vomiting, anorexia, mid-epigastric pain, left upper quadrant pain radiating to the left flank and decreased bowel sounds.

A multidisciplinary team consisting of a surgeon, radiologist, obstetrician, and a neonatologist should be available, though the strategy for treatment is similar to the general nonpregnant patient with acute pancreatitis.[4] Hence, we report these two cases owing to the rarity of the condition and good maternofetal outcome.

CASE REPORTS

Case 1

A 21-year-old primi at 35 weeks +2 days presented with complaints of vomiting and abdominal pain of one day duration. She perceived fetal movements well. The obstetric history revealed that she had gestational HTN in the 2nd trimester and was started on tablet labetalol 50 mg tds. She was on metformin 250 mg tds for polycystic ovarian syndrome (PCOS) and on thyroxine 25 mcg.

On examination, she was afebrile and her vitals were stable. On per abdominal examination, uterus corresponding to 34 weeks size, cephalic presentation, relaxed, clinically liquor reduced, fetal heart sound good. Her Bishop score was 3.

Her baseline investigations showed elevated levels of serum amylase and lipase values which were 157 and 475 mg/dl respectively. Serum calcium and parathormone levels and lipid profile were normal. After surgical opinion, a diagnosis of acute pancreatitis was made and the patient was managed conservatively with IV fluids, broad spectrum antibiotics and serial monitoring of sugars, ketones, electrolytes and fetal well-being. She was asymptomatic after a week and serum amylase and lipase values were found to be 95 and 492 mg/dl respectively. After a week, emergency lower segment cesarean section (LSCS) was done in view of severe oligohydramnios and preeclampsia and she delivered a baby girl weighing 2.8 kg. Postoperative period was uneventful. Patient was discharged on postoperative day 7.

Case 2

A 31-year-old primi at 34 weeks and 5 days presented with complaints of pain abdomen and vomiting of 5 days duration. She perceives fetal movements well. All trimesters were uneventful.

On examination, she was afebrile and her vitals were stable. On per abdominal examination, uterus corresponds to 36 weeks, cephalic presentation, relaxed, clinically liquor adequate, fetal heart sounds good. Her Bishop score was 4.

Her baseline investigations showed that her serum amylase and lipase values were elevated to 1449 and 550 mg/dl. Serum calcium, parathormone levels and lipid profile were normal. After a surgical opinion and a diagnosis of acute pancreatitis was made and patient was started on conservative management with IV fluids, broad spectrum antibiotics and serial monitoring of sugars, ketones, electrolytes and fetal well being. After 3 days, she was induced with prostaglandin E2 (PGE2) gel in view of preterm premature rupture of membranes (PPROM) and delivered a baby boy weighing 2.7 kg. Postnatal period was uneventful and the mother was monitored regularly. Magnetic resonance imaging (MRI) showed features of acute pancreatitis, dilated pancreatic duct, common bile duct and bilateral pleural effusion. She was asymptomatic after a week and her serum amylase values were 192 mg/dl and lipase values were 193 mg/dl. Medical gastro-opinion was sought and the patient was advised to undergo endoscopic retrograde cholangiopancreatogram (ERCP) after 4 weeks. Patient was then discharged and now patient is on follow-up.

DISCUSSION

The management of acute pancreatitis during pregnancy poses a dilemma. It is usually self-limiting but can occasionally progress to severe disease and even death. The knowledge of the signs and symptoms along with the management of this condition is of prime importance so that the risk of mortality of the mother and fetus can be reduced.

In the first case, the investigation of the cause of the episode of acute pancreatitis sheds little light on the causative agent. Since her imaging studies show no evidence of a gall bladder disease, the other causes were explored. However, the use of tablet metformin 500 mg on a daily basis could be linked to her current episode as there have been instances of metformin induced pancreatitis.[5] But, it is seen that available evidence suggests that acute pancreatitis is only caused by metformin accumulation, resulting from a combination of drug overdose and acute renal failure, in turn triggered by vomiting in a patient with concealed renal insufficiency. With normal renal function and correct dosage, metformin-induced acute pancreatitis was never reported.[6] Hence, the cause for the episode of acute pancreatitis can be said to be idiopathic.

In the second case, the imaging clearly reports the presence of a gall bladder disease. Pancreatitis in pregnancy was more likely to be associated with cholelithiasis as seen in the nonpregnant population.[7]

The clinical characteristics of acute pancreatitis in pregnancy are similar to the nonpregnancy state. The usual symptoms seen are abdominal pain, anorexia, nausea, vomiting, dyspepsia, low-grade fever, tachycardia, fatty food intolerance. Leukocytosis, increase in the inflammatory and pancreatic markers and in case of biliary disease rise of cholestatic markers can be seen on hematological and biochemical examination.[8] But, a diagnosis of acute pancreatitis is made only after a thorough investigation since the hematological, biochemical and clinical findings are also seen in the nonpregnant state.

Though acute pancreatitis is more commonly seen in the 2nd and 3rd trimesters, it can also occur in the 1st trimester.[9]

In our case report, both patients had abdominal pain associated with multiple episodes of vomiting. However they were both in the 3rd trimester of pregnancy during the onset of symtoms. In addition, the imaging done show features suggestive of acute pancreatitis in the 1st and biliary disease leading to pancreatitis in the 2nd case.

Acute pancreatitis can be diagnosed based on the presence of two or more of the three criteria seen: (1) abdominal pain consistent with pancreatitis, (2) serum amylase and lipase increased to more than three times the normal values and (3) characteristic findings suggestive of acute pancreatitis seen on the USG or CT or MRI.[10] A crucial role is played by the serum biochemical markers in identifying the condition early and leading to prompt treatment—amylase and lipase. There is a rise within the first 12 hours after the onset of symptoms and returns to normal within 3 to 5 days in the case of serum amylase.[11] Serum lipase activity remains increased for longer (up to 8-14 days), and is of increased sensitivity in patients with a delayed presentation. Hence, lipase is considered to be diagnostically accurate than amylase.[12]

The imaging techniques in use today form a more accurate picture of the condition of acute pancreatitis and hence they are to be used to make a complete clinical diagnosis. The commonly used imaging techniques are ultrasonogram, CT, MRI, magnetic resonance cholangiopancreatography (MRCP) and endoscopic retrograde cholangiopancreatography (ERCP). The safest mode of imaging used for diagnosis is the transabdominal ultrasound as there is no danger of emitting harmful radiation to the fetus. However, parenchymal or peripancreatic pathology cannot be seen by an ultrasound. Computed tomography is the most efficient imaging technique as it is used for confirming the diagnosis, classifying pancreatitis in interstitial edematous or necrotizing, depicting the local complications and also for defining

the severity of the disease. Computed tomography is now used only where severe acute pancreatitis is suspected as mild pancreatitis does not require any intervention.

The peri/pancreatic tissues and the biliopancreatic duct systems can be visualized by the MRI and the MRCP. Magnetic resonance imaging should be used only when other nonionizing forms of diagnostic imaging are inadequate or the patient care depends on further imaging.[13] In severe biliary acute pancreatitis, ERCP is being used as a therapeutic intervention, by combining the ERCP with endoscopic sphincterectomy. Complications can be prevented by the decompression of the common bile dust and removal of the gall stones and hence morbidity as well as mortality of severe biliary acute pancreatitis will be reduced.[14] In both our cases initially, an ultrasound of the abdomen was taken which confirmed the features of acute pancreatitis. After the regression of their symptoms and delivery both women are currently being followed up for an ERCP.

Preterm delivery and mortality is the complication seen in the fetus if acute pancreatitis occurs in the first trimester.[15] In the 3rd trimester, there may be maternal morbidity and fetal mortality.[16] Both of the cases reported above are in the 3rd trimester. In the first case report, the episode of pancreatitis settles down and causes no harm while in the second case, the onset of the pancreatitis could well be the cause of the PPROM reported. Hence, the resolution of symptoms at the earliest could prove to be a boon for the fetus as it would avoid a preterm delivery and would prevent an NICU admission.

In the management of acute pancreatitis in pregnancy, the initial treatment is similar to the nonpregnant patient but the subsequent management might differ due to the risk of fetal disturbances or teratogenesis. Therefore, it is important the medical team to consist of several different specialties, such as obstetrician, surgeon, radiologist and a neonatologist. The treatment given focuses primarily on reducing the pancreatic secretions, restoring the third space fluid collection, and supporting the patient by providing the necessary nutrition, oxygen, analgesics and monitoring the vital signs of mother and fetus. Mild acute pancreatitis subsides within the first 7 days and the patient does not require nutritional support as long as oral feeds are tolerated.[17]

Pain, which is the main symptom in most cases, is managed by the use of analgesics, mainly meperidine and opioid group of drugs like morphine.[18] It is preferable to use the broad spectrum antibiotics like ciprofloxacin plus metronidazole, imipenem/cilastatin and piperacillin/tazobactam or meropenem, administered for at least 10 to 14 days.[19] There should be continuous monitoring of the lab tests as well as imaging methods, since the administration of antibiotics is adequately adjusted based on the values. Any surgical intervention is delayed as long as possible.[20]

As seen in both our cases, the patients were put on Nil per oral, initially started on injection piperacillin tazobactam. After the regression of the symptoms, they were started on oral feeds and the antibiotics were stopped. No surgical interventions were required immediately as both were cases of mild acute pancreatitis.

CONCLUSION

The management of acute pancreatitis should be a multidisciplinary team approach. Early diagnosis and timely intervention reduces both maternal and fetal morbidity and mortality.

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