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Acute Pancreatitis in Third Trimester of Pregnancy: A Case Report

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Abstract
Acute pancreatitis is a rare and serious complication during pregnancy, which can have a high maternal mortality and perinatal mortality. Acute pancreatitis in pregnancy poses diagnostic and therapeutic challenges. The most common cause of acute pancreatitis in pregnancy is gallstones and hypertriglyceridemia. We report a case of acute pancreatitis in a pregnant woman in her third trimester and discuss the analytical and restorative challenges of acute pancreatitis in pregnancy. Even though it is a rare, acute pancreatitis should be suspected in all pregnant patients admitted for non-obstetric abdominal pain. Early diagnosis and good supportive care by a multidisciplinary team are essential to ensure good maternal and fetal outcome.
Keywords: *Acute pancreatitis, pregnancy, hypertriglyceridemia, prognosis, treatment.*

Introduction

Acute pancreatitis is an uncommon but serious problem occurring during pregnancy. The reported yearly incidence of acute pancreatitis has ranged from 4.9 to 35 per 100,000 populations[1]. Acute pancreatitis is unusual during the first and second trimester (12%), typically occurring in the third trimester (50%) or early postpartum period (38%)[2]. Gallstones (66%), alcohol (12%), hypertriglyceridemia (4%), idiopathic (17%) are the commonest identified causes of acute pancreatitis in pregnancy[2,3,4]. We are reporting an unusual case of acute pancreatitis in the third trimester in a pregnant woman and importance on the diagnostic and management challenges.

Case Report

A 30-year-old G3P1L1A1 a case of Previous LSCS, had history of hospitalisation in two private hospitals for fever on and off with epigastric pain for past 15 days. She had elevated serum enzyme lipase- 2015 IU/l and amylase- 1011 IU/l and was referred to tertiary care centre for further care with the diagnosis of acute pancreatitis. She was received on 25/09/2017 at 38 weeks of gestation, with severe pain confined to the upper abdomen, which was radiating to the back for past 2 days. Patient also complained of fever and nausea. On examination, pulse rate was 101/min, BP was 100/70 mmHg. Temperature was 99.9^0F and respiratory rate was 20/min. per

abdominal examination revealed epigastric tenderness with guarding. Uterus was term size, relaxed, fetal heart tones at 146/min, previous Lscs scar healthy, No scar tenderness. Laboratory tests done on the day of admission showed a white blood count of 13,900/mm3, polymorph 58.7% and a platelet count of 1,35,000 lakhs/cumm. Serum lipase was 1,331 IU/l (ref: 6-51 IU/l), amylase 515 IU/l (ref: 22-80 IU/l), calcium 8.4 mg/dl (ref: 8.6-11 mg/l), triglycerides 476 mg/dl (ref: <150 mg/dl) and uric acid 4.5 mg/dl (ref: 2.4-6.7 mg/dl). Liver function tests showed raised alkaline phosphatase - 258 mg/dl (ref: 35-104 mg/dl) Lactate dehydrogenase was 322 mg/dl (ref: 135-214 mg/dl). Random blood sugar and renal function tests were within normal limits. Urine analysis was normal. Abdominal ultrasonography showed a bulky edematous pancreas and mild left hydroureteronephrosis, suggestive of acute pancreatitis with single live intrauterine fetus of 38 weeks of gestational age with normal cardiac activity and adequate liquor. Medical gastroenterologist opinion was obtained and suggested for conservative management. Daily Non-stress test was done and found to be reactive. Daily kick chart was monitored. Patient was kept in High dependency unit (HDU) and was managed by nil orally, nasogastric aspiration, intravenous fluids, blood glucose monitoring, antibiotics, analgesics, antipyretics with strict fetal heart monitoring. Conservative treatment was continued for 48 hours after which pain started decreasing gradually. As she complained of pain abdomen and had tenderness over the scar, was taken for emergency lower segment cesarean section with sterilisation on 27/09/2017 and delivered a healthy Female baby. Post-operative period & follow up was uneventful. Her general wellbeing and blood parameters enhanced remarkably. She was asymptomatic and her last blood test results prior to discharge were normal. Repeat serum amylase was 39 IU/L, lipase was 95 IU/L. Repeat Usg abdomen was performed on 6th day and found to be normal. Patient was discharged on the 6th postoperative day in good condition.

Discussion

Acute pancreatitis in pregnancy is rarely encountered and can have a high maternal mortality and perinatal mortality. Acute pancreatitis is an inflammatory condition of the pancreas characterized clinically by abdominal pain and with elevated levels of pancreatic enzymes in the blood[5,6]. Pancreatitis can occur during any trimester, most commonly found in the third trimester of around 52% of cases; it is rarely seen in the postpartum period.

In the hepatic bile, Cholesterol secretion increases in the second and third trimester compared to bile acids and phospholipids which leading to supersaturated bile. In addition, fasting and postprandial gallbladder volumes are greater with reduced rate of volume of emptying. This large amount of residual volume of supersaturated bile in the gallbladder which leads to cholesterol crystals and finally gallstones[7]. Cases of More than 80% of acute pancreatitis are associated with Gall stones along with alcohol abuse.

In pregnancy, Hypertriglyceridemia also seems to be the peak in third trimester and tends to be a more severe form of pancreatitis than that due to gallstones[9]. Throughout pregnancy, there is a physiologic estrogen– induced increase in triglyceride–which leads to rich in lipoprotein production and suppression of lipoprotein lipase activity in the liver and adipose tissue which causes decrease in clearance of triglyceride. There are two possible mechanisms have been implicated for the pathogenesis of hypertriglyceridemia. Hydrolysis of the extreme quantity of triglyceride in the pancreas results in local release of extremely concentrated free fatty acids, which might exert their cytotoxic effect on acinar cells and vascular endothelium. An additional theory postulated that high concentration of chylomicrons might increase blood viscosity and still precipitate capillary obstruction in the pancreas, which causes local pancreatic ischemia, acidosis and activation of trypsinogen[10].

In pregnancy, Pancreatitis may be associated with

HELLP syndrome or preeclampsia which leading to increase fetal mortality or preterm delivery[11]. Type 2 Diabetes mellitus is associated with 2.8-fold increase risk for acute pancreatitis[12]. Due to the physiological changes in pregnancy such as weight gain, increased triglycerides, and elevated levels of oestrogen which causes acute pancreatitis. Hyperparathyroidism, connective tissue diseases, infections, drug induced and trauma -both iatrogenic and accidental—are other rare causes of acute pancreatitis.

The symptoms like upper abdominal pain, nausea, vomiting, anorexia, fever and elevated serum amylase or lipase levels usually appears during the third trimester or early postpartum period.

Threatened preterm labour, preterm deliveries and intra uterine fetal death are complications of acute pancreatitis during pregnancy[3].

Laboratory tests for diagnosing acute pancreatitis include serum amylase, lipase, complete blood count, serum triglycerides, calcium and liver function tests. An elevated serum amylase level has a diagnostic sensitivity of 81% and adding serum lipase increases this sensitivity to 94%[13].

Ultrasound abdomen imaging plays an important role, safe and quite inexpensive but it has low down diagnostic value for acute pancreatitis and in establishing the underlying etiology. An additional alternative imaging in cases of uncertain ultrasound findings is magnetic resonance cholangiopancreatography (MRCP) without contrast medium which has more than 90% sensitivity with no ionizing radiation to the mother and fetus. MRCP also restricted the use of endoscopic retrograde cholangiopancreatography (ERCP) and used only to women who require therapeutic measures. Endoscopic ultrasound has better sensitivity than MRCP in imaging of choledocholithiasis and micro stones but it needs sedation[14, 15].

The initial management of acute pancreatitis in pregnancy does not differ from non pregnant state. The initial management are oxygen, fluid restoration, antiemetics, antipyretics, analgesics, and cessation of oral feeding to suppress exocrine function of pancreas, thus preventing auto digestion of pancreas[18]. Conservative treatment is preferred, particularly for mild acute pancreatitis. Hyperlipidemic pancreatitis patients must go through low fat diet, antihyperlipidemic therapy, insulin (to increase lipoprotein lipase activity), heparin (to increase lipoprotein lipase activity), and even plasmapheresis must be done as soon as it becomes essential.

It has been accepted that cholecystectomy during second trimester is safe for mother and fetus. It is well respected surgical concept that the second trimester is the best period for surgery since during this period organogenesis is complete and the uterus is not big enough to obliterate the surgical view for laparoscopic approach. Rigorous symptoms, obstructive jaundice, acute cholecystitis intractable to medical treatment, and peritonitis are indications for surgery in pregnancy.[7,2].

In uncomplicated mild acute pancreatitis (MAP), nutritional support is not needed and low fat diet can be started within 3–5 days[17]. In severe acute pancreatitis (SAP), treatments must consist of enteral feeding (EN) through either postpyloric feeding or nasojejunal and, if necessary, they will need parenteral feeding. There is a high chance of infections and metabolic derangement noted in total Parenteral Nutrition (TPN) feeding, but in enteral feeding (EN) is physiological and helps gut flora maintain gut immunity[18].

Antibiotics have no role in acute pancreatitis and it still remains controversial. In a systematic review and meta-analysis show antibiotic prophylaxis does not protect against infected necrosis or diminish the mortality and rate of recurrence of surgical intervention[19].

There is an excellent prognosis for mild acute pancreatitis (MAP) with no adverse outcome on the fetus or mother. In 1973, 31% maternal mortality noted in acute pancreatitis of pregnancy [20] but in 2009 it came downwards up to 1%. Out of 38 patients with acute pancreatitis in pregnancy, two maternal deaths were noted in the recent review[21]. In another review, out of 16

patients with acute pancreatitis in pregnancy, two maternal deaths were noted[22]. In 1973, perinatal mortality was 50% but in 2009 review not still one perinatal death noted out of 73 patients in second and third trimester of acute pancreatitis in pregnancy and all 73 patients delivered term babies[17].

Despite that, the fetal risks from acute pancreatitis during pregnancy which include threatened preterm labour, preterm delivery and intra uterine fetal death remain a concern[2].

Nevertheless, there are still obstetric struggle to be addressed in the first trimester. Only 60% out of 30 patients with acute pancreatitis in first trimester achieved term pregnancy with fetal loss of 20% [17].

Management of severe acute pancreatitis (SAP) occurring in first trimester carries a superior prognosis for mother but it is related with increased fetal loss of about 20%[16]. In a study Banks et al.[16], of 103 patients with acute pancreatitis in pregnancy, no maternal mortality noted in 30 patients in first trimester and out of 96 patient only one maternal death noted in the studied. Still this is not a universal situation. Shoaib Gangat et al.[23] study, out of 166 patients with acute pancreatitis in pregnancy, 46% had perinatal mortality and 30.76% had maternal mortality.

The contradictory tendency in acute pancreatitis in pregnancy is the increase in the quantity of patient diagnosed but generally decline in perinatal and maternal morbidity and mortality associated with it. Increase in occurrence can be credited to various factors such as superior diagnostic facilities, better consciousness of the disease, and increase in numbers of obesity all over the world. The begin of quick assay methods for serum amylase, improved supportive care of pancreatitis, newer curative method for gallstone pancreatitis, and generally enhancement in antenatal care have absolutely contributed to improved maternal and fetal outcomes.

Conclusion

Acute pancreatitis in pregnant woman management should involve multidisciplinary approach and should be individualised as a case to case basis. By enlarge conservative and supportive management plays an important role in third trimester of pregnancy and with vigorous fetal monitoring to avoid maternal and perinatal morbidity and mortality.

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