

Acute Pancreatitis In Pregnancy

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Citation

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Abstract

Acute pancreatitis during pregnancy is rarely encountered and can have a high maternal mortality and fetal loss. We report here a case of a 23-year-old nulliparous woman at 33 weeks of gestation presenting with hypertension, epigastric pain, vomiting and pedal edema. Investigation revealed hyperamylasemia and gallbladder sludge. The patient underwent an uncomplicated caesarean section after stabilization in the intensive care unit.

INTRODUCTION

Pancreatitis during pregnancy is rare. Schmit in 1818 first reported this condition in a 30-year-old multigravida. Review of literature also reveals that Lawrence described the earliest series of 53 cases in 1838.¹

CASE

A 23-year-old nulliparous woman was admitted at 33 weeks of gestation with pain in the upper abdomen, which was radiating to the back, and with nausea and vomiting since the last 3 days. Vital monitoring at the time of admission showed a pulse rate of 103/min, a blood pressure of 170/110mmHg and a respiratory rate of 22/min. Physical examination revealed bilateral pedal edema, epigastric tenderness, decreased bowel sounds and a gravid uterus. Fetal heart tones were at 140/min.

Laboratory tests showed a white blood count of 16900/cumm, a haematocrit of 40.75 and a platelet count of 220000. Random blood sugar, arterial blood gas analysis, liver function tests and renal function tests were within normal limits. Serum amylase was 1020.3 IU/l (ref. <90 IU/l), lipase 348 IU/l (ref. 8-57 IU/l), albumin 16g/l (ref. 35-50g/l), calcium 7.8mg/dl (ref. 8.5-11mg/l), triglycerides 294mg/dl (ref. <150mg/dl) and uric acid 6.9g/dl (ref. 2.4-6.7mg/dl). Urine analysis was positive for albumin and red blood cells.

Abdominal ultrasonography showed a single live intrauterine fetus with normal cardiac activity and mild to moderate oligohydramnios. (Fig-1) The gallbladder revealed echogenic sludge. The pancreas could not be visualized due to obscuration by bowel gases. (Fig-2)

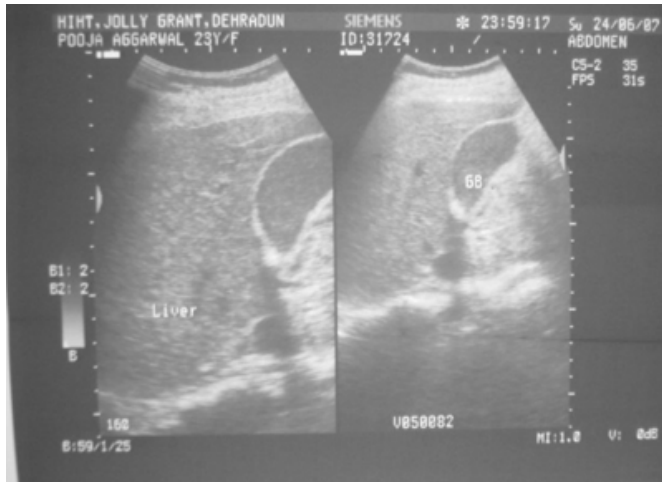
Figure 1

Figure 1: Sonography Showing Femur Length Of 61.4mm Corresponding To 33 Weeks Of Gestation With Oligohydramnios.



Figure 2

Figure 2: Sonography Of The Abdomen Showing Gallbladder Sludge.



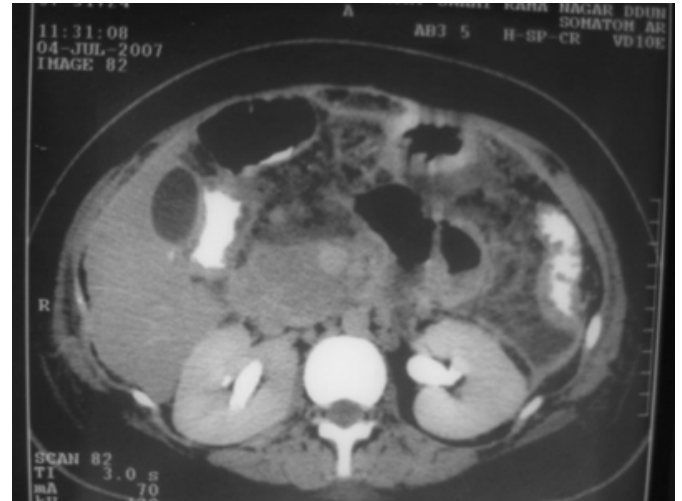
The patient was shifted to the intensive care unit and managed initially by nil orally, nasogastric aspiration, intravenous fluids, antibiotics, analgesics and total parenteral nutrition with strict fetal monitoring. An uncomplicated caesarean section was performed on hospital day 2, owing to the persistent uncontrolled pregnancy induced hypertension, increase in pedal edema and fetal tachycardia. Intra-operatively, the pancreas was found to be edematous and there was evidence of fat necrosis in the omentum. An omental biopsy was taken for confirmation. The abdomen was closed with placement of a peripancreatic drain. Postoperatively, the patient was transferred back to the intensive care unit.

The patient developed mild bilateral pleural effusion on the 1st postoperative day, which was managed conservatively. Culture of the drain fluid was found to be sterile.

The patient was subjected to contrast enhanced computed tomography of the abdomen on the 6th postoperative day that showed a bulky head of the pancreas with heterogeneity suggestive of acute pancreatitis, a septate fluid collection in the greater sac, peripancreatic region and left anterior pararenal space, a left pleural effusion and an enlarged post-partum uterus. (FIG-3)

Figure 3

Figure 3: Contrast-Enhanced Computed Tomography Of The Abdomen Showing A Non-Echogenic Bulky Pancreas And A Septate Fluid Collection In The Greater Sac, Peripancreatic And Left Anterior Pararenal Space.



Omental biopsy confirmed fat necrosis.

By postoperative day 9, the patient tolerated oral intake of nutrition and total parenteral nutrition was stopped. Drain was removed on day 10.

The patient was discharged on day 14 with the advice to undergo laparoscopic cholecystectomy around 8 weeks post partum.

DISCUSSION

The incidence of pancreatitis ranges from 1 in 1066 live births to 1 in 3333 pregnancies. An attack of pancreatitis was previously thought to be common in nulliparous women. Ramin et al. reported pancreatitis during pregnancy in 72% of multiparous women.²

Pancreatitis can occur during any trimester but around 52% of cases are found in the third trimester; it is rarely seen in the post partum period.² Acute pancreatitis following medical abortion is also reported.¹⁰

Gallstones are the most common etiological factor accounting for about 67-100% of cases. Small stones are more prone to cause pancreatitis. Recently, sludge in the gallbladder has also been reported to cause the disease in pregnancy. Acute pancreatitis develops due to mechanical obstruction at the ampulla of Vater due to passage of stones or sludge.^{2, 3}

Hyperlipidaemia is the second most common causative

agent. Pregnancy increases the level of serum cholesterol and triglycerides and causes biliary stasis thus inducing the formation of gallstones. Hypertriglyceridaemia may also directly cause acute pancreatitis. The level of serum triglycerides required to induce an attack ranges from 750 to 1000mg/dl. Genetically, a mutation in the lipoprotein lipase gene causing hypertriglyceridaemia-induced pancreatitis in pregnancy has also been reported.^{4, 5}

Drugs such as tetracycline and thiazide (used in pregnancy induced hypertension) and alcohol consumption during pregnancy may induce pancreatitis.⁶

Acute necrotizing pancreatitis is also reported in preeclampsia due pancreatic microvascular alterations.⁷

Pancreatitis in pregnancy is also proposed due to reflux of gastrointestinal contents into the ampulla of Vater, mutation in the cystic fibrosis transmembrane conductance regular gene and immunological interactions between mother and child.^{8, 9}

Clinical presentations include pain in the epigastrium or left hypochondrium with or without radiation to the back, anorexia, nausea, vomiting and jaundice. Signs include abdominal tenderness with decreased bowel sounds. In 10% of cases pulmonary findings may be associated which may lead to full blown adult respiratory distress syndrome.⁶ Generalized anasarca may be associated with preeclampsia-associated pancreatitis.⁷

Diagnostic work up includes complete blood count, serum triglycerides, calcium and liver function tests in the form of serum bilirubin, transaminases and alkaline phosphatase. An elevated serum amylase level has a diagnostic sensitivity of 81% and adding serum lipase increases the sensitivity to 94%. The mean amylase in such type of patient is found to be 1400 IU/l. However, amylase levels do not correlate with disease severity.^{2, 11}

Imaging of the pancreas can be performed by using ultrasonography and computed tomography. Due to hazards of radiation to the fetus, sonography is preferred which can also detect gallstones with 90% sensitivity. However, the sensitivity for biliary sludge that appears as low level echoes within the gallbladder which shifts with positioning is lower.³

Severity of pancreatitis can be graded using scales such as Ranson's criteria, Imrie's criteria or APACHE II score similar to non-pregnant patients.³ The most common

differential diagnosis of acute pancreatitis in the first trimester of pregnancy is hyperemesis gravidarum. Biliary colic, acute cholecystitis, acute appendicitis and acute fatty liver of pregnancy are other differential diagnoses of this entity.^{3, 6}

The treatment of pancreatitis in pregnancy should be conservative as far as possible with delaying the definitive treatment until after delivery. Management includes nil orally, nasogastric aspiration, intravenous fluids, antispasmodics, antibiotics and total parental nutrition. Lipoprotein apheresis and plasmapheresis may be tried to lower serum triglycerides levels. Endoscopic sphincterotomy with fetal shielding with the help of a lead apron may be helpful in treating a gallstone-induced pancreatitis. The second trimester is thought to be the ideal time for endoscopic sphincterotomy to avoid any possible teratogenic effects of radiation. Fetus monitoring should be strictly done during the course of this treatment.^{6, 12}

The second trimester is also the optimum time for the patient to undergo any surgical intervention. Cholecystectomy after endoscopic sphincterotomy should be considered in gallstones induced pancreatitis in pregnancy with recurrent attacks. Exploration of the common bile duct may be done where endoscopic sphincterotomy facility is not available. Surgical drainage for acute pancreatitis may help in reducing the load of toxic materials by draining the peritoneal fluid but carries a high morbidity and mortality. Authors have advised to wait with any surgical intervention until delivery, if the patient develops uncomplicated pancreatitis in the third trimester of pregnancy. Complications such as pseudocysts should be surgically managed in the post partum period.^{3, 8}

In the past, pancreatitis during pregnancy had been associated with a high maternal death rate and fetal loss. However, recent studies have shown that these rates are declining due to earlier diagnosis and better treatment options.^{6, 11, 12}

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