SUCCESSFUL OUTCOME OF ACUTE BILIARY PANCREATITIS IN PREGNANCY - A CASE REPORT

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Abstract

Acute pancreatitis though a rare event in pregnancy can lead to high maternal mortality and foetal loss. It is usually caused by gall stones or gall sludge. Here we report a case of acute pancreatitis at 36 weeks of gestation. The patient recovered successfully because of timely diagnosis and management. Later she underwent elective caesarean and delivered a healthy baby without any complication. This case highlights the importance of suspicion of acute pancreatitis in pregnant woman presenting with epigastric pain, nausea and vomiting. Timely diagnosis and management can lead to a successful outcome.

Keywords: Acute biliary pancreatitis, Pregnancy, Abdominal pain

Introduction

Acute pancreatitis (AP) is commonly seen in general population with an incidence of 5 to 80 per 10,000 people whereas in pregnancy it is rarely seen and the reported incidence varies from 1 in 1000 to 10,000 pregnancies. Approximately 70% of cases are biliary or gall stone induced followed by alcohol and hypertriglyceridemia. The other causes are hyperparathyroidism, connective tissue diseases, abdominal surgeries and infections. It is usually seen in the third trimester and is seen to be associated with pre-eclampsia and HELLP syndrome. Hence at times, signs and symptoms could be overlapping leading to misdiagnosis and symptoms may be attributed to pre-eclampsia. The recent advances in imaging modalities, surgical techniques, and neonatal intensive care have resulted early diagnosis and management leading to improved survival of mother and infant.

Case report

A previously healthy, booked, nonalcoholic 40 year old primigravida was admitted from casualty at 36 weeks of gestation with acute and severe pain in epigastrium, radiating to the back, associated with nausea and vomiting. During her regular antenatal visits, she was diagnosed to have gestational diabetes mellitus and was controlled on diet with satisfactory foetal growth. During routine antenatal ultrasonography, she was incidentally diagnosed to have multiple tiny calculi in the gall bladder which remained asymptomatic. At admission she was afebrile, pulse was 90/min, blood pressure 142/80 mm Hg, and respiratory rate was 20/min. There was epigastric tenderness. Foetal heart rate was 140/min. Initial laboratory reports showed a white blood cell count of 15,500/µL, hemoglobin level of 15.4 g/dL, hematocrit of 45.8% and platelet count of 1.21 L/µL. There was mild derangement of liver function test (T.bil-0.7, AST-135IU, ALT-212IU). Serum amylase was 1496 IU/L. Lipid profile was normal (Table 1).

Abdominal ultrasound was done after admission which showed contracted gall bladder, filled with multiple calculi of size 4-5 mm. Intra and extra hepatic biliary ducts, common bile duct, portal vein, visible part of head and body of pancreas appeared normal. As per laboratory and ultrasound findings and excluding other common causes, she was diagnosed with gall stone induced AP which was found to be mild in severity based on the Ranson criteria and given a score of 1 based on Bedside Index of Severity in Acute Pancreatitis (BISAP) scoring system.

In consultation with surgeon, she was put on conservative management which included keeping her nil per orally, intravenous fluids, parenteral proton pump inhibitors, antiemetic, analgesics and antibiotics. She started improving symptomatically after 24 hours of conservative management. Serum amylase level dropped to 149 IU/L on day four of supportive treatment. The foetus was monitored by biophysical profile which was satisfactory. As it was a precious pregnancy, she underwent elective lower segment cesarean section at 37 weeks and delivered a healthy baby. Mother and baby were discharged in a stable condition on postpartum day five.

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Table 1- Laboratory data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day-1</th>
<th>Day-4</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>15,500</td>
<td>12,000</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>79</td>
<td>78</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>20</td>
<td>17</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Hb (g%)</td>
<td>15.4</td>
<td>12.8</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>45.8</td>
<td>40.4</td>
</tr>
<tr>
<td>Platelet (Lakhs)</td>
<td>1.21</td>
<td>1.01</td>
</tr>
<tr>
<td>S. amylase (IU/L)</td>
<td>1496</td>
<td>149</td>
</tr>
<tr>
<td>Fasting Blood Sugar (mg/dL)</td>
<td>101</td>
<td>83</td>
</tr>
<tr>
<td>T.P/Alb (g/dL)</td>
<td>6.2/3</td>
<td>6/2.9</td>
</tr>
<tr>
<td>AST/ALT (IU/L)</td>
<td>135/212</td>
<td>64/81</td>
</tr>
<tr>
<td>ALP (IU/L)</td>
<td>578</td>
<td>321</td>
</tr>
<tr>
<td>T.Bil /D.Bil (mg/dL)</td>
<td>0.7/0.2</td>
<td>0.7/0.1</td>
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<tr>
<td>BUN/Crt (mg/dL)</td>
<td>22/0.9</td>
<td>24/0.9</td>
</tr>
</tbody>
</table>

Discussion

The etiology of AP during pregnancy are similar as seen in the general population, most common being gallstone disease or hypertriglyceridemia. It was reported that 38.9% cases of acute pancreatitis in pregnancy were caused by biliary tract diseases, 27.8% were due to hypertriglyceridemia and 11.1% were idiopathic. The incidence of gallstone related diseases such as acute cholecystitis and biliary pancreatitis complicating pregnancy is 0.05%-0.8%. Pregnancy does not primarily predispose to pancreatitis, but it increases the risk of cholelithiasis and biliary sludge formation as there is decreased gall bladder motility attributed to hormones of pregnancy. Cholelithiasis often precedes the symptoms and clinical picture of pancreatitis as seen in our case. During second and third trimesters, there is increased cholesterol secretion in the hepatic bile as compared to bile acids and phospholipids, leading to formation of supersaturated bile. The reduced rate and volume of emptying of gall bladder leads to large residual volume of supersaturated bile. This leads to the retention of cholesterol crystals and gallstones formation. There is strong association between the formation of biliary sludge and stones with the frequency and number of pregnancies.

During pregnancy, triglycerides are increased by 2-4 times the non-pregnant levels which are usually seen in third trimester. Hypertriglyceridemia is a second commonest cause of AP and is seen to be an independent risk factor for AP if the levels are more than 1000 mg/dL. Reduction in the triglyceride levels below 1000 mg/dL can effectively prevent further episodes of pancreatitis. The other common causes are alcohol and diabetes mellitus. Rarely seen causes are hyperparathyroidism, connective tissue diseases, idiopathic or drug induced (N-Acetyl-Cysteine, Furosemide, Somatostatin and Valproate etc.).

There are numerous diagnostic challenges due to the physiological and anatomical changes seen in pregnancy. Presenting symptoms of AP such as nausea, vomiting, abdominal discomfort, or pain, are frequently seen in pregnancy. Clinical evaluation becomes difficult due to the following reasons:

1. Gravid uterus,
2. Stretching and lifting of the anterior abdominal wall away from the area of inflammation,
3. Hampering of movement of the omentum to an area of inflammation.

Such alterations can lead to misdiagnosis or unnecessary non-obstetric surgical interventions which are associated with a higher premature labour rate. Leukocytosis and raised hematocrit are usual features of pregnancy. Elevated amylase and/or lipase remains the diagnostic hallmarks of AP; yet, in hypertriglyceridemia-induced acute pancreatitis, amylase levels may be reported as normal or even low in more than 50% patients.

Imaging plays an important role in diagnosing of AP in establishing underlying etiology and grading the severity of disease. Abdominal ultrasound is an ideal imaging as it has no radiation risk. It is also useful for detecting dilated pancreatic ducts and pseudocysts. Endoscopic ultrasound (EUS) is done when common bile duct stone is suspected and has got a high positive predictive value nearly 100% even for small stones ≤2 mm or sludge. Endoscopic retrograde cholangiopancreatography (ERCP) or contrast-enhanced computed tomography (CECT) (a gold standard for diagnosing common bile duct stones and pancreatitis) or MRI is preferred if USG fails to find out a cause of an acute abdomen. Serum amylase levels are raised within hours of onset of pain and can remain high for 3 to 5 days. An elevated serum amylase level has a diagnostic sensitivity of 81% and adding serum lipase increases the sensitivity to 94%. Increased levels are also seen in intestinal obstruction, visceral perforation, tubo-ovarian abscess, renal failure and salivary gland disease. Serum lipase has higher specificity for pancreatic disease, but it may be elevated in other conditions as well. The severity of pancreatitis does not correlate well with the serum levels of amylase and lipase. Hence daily trends need not be looked for because it does not correlate with either recovery or prognosis. The other laboratory abnormalities seen in acute pancreatitis are hyperglycemia, hypocalcaemia, leukocytosis, elevated blood urea nitrogen (BUN) >20mg/dL and mild derangement of liver function test. Elevation of serum alanine
aminotransferase more than 80 U/mL is highly specific but poorly sensitive for biliary pancreatitis. The diagnosis of acute pancreatitis requires at least two criteria, such as 1) classical abdominal pain 2) radiographic evidence of acute pancreatitis 3) elevation of amylase and of lipase three times the upper limit of normal.

A multidisciplinary approach including surgeons and gastroenterologist should be considered in managing AP in pregnancy more so in severe cases. The first line of management is conservative and supportive which includes gastric decompression, antispasmodic drugs and antibiotics. Surgical treatment could be considered in case of worsening condition after 2-3 days of conservative management, pancreatic enlargement, necrosis and gastrointestinal perforation. Endoscopic Retrograde Cholangiography (ERCP) sphincterotomy and laparoscopic cholecystectomy can be performed preferably in second trimester, when technical conditions are optimal and risks are minimized. AP alone is not an indication for termination of pregnancy but may be considered in full term gestation, deteriorating maternal condition after 24-48 hours of conservative treatment, no improvement of paralytic ileus, stillbirth, foetal malformation, and severe pancreatitis. AP in pregnancy can lead to preterm labour and preterm birth, placental abruption and profound metabolic complications and acidosis. Hence, regular foetal monitoring should be emphasized during conservative management.

In the past decade higher maternal and foetal mortality rates were seen, 20% and 50% respectively.15,16 However, era of ERCP and laparoscopic cholecystectomy has resulted in improved feto-maternal outcome and reported foetal loss is 4.7 % with no maternal deaths.15,16

Conclusion

Although rare, acute pancreatitis should be suspected in all pregnant patients admitted for non-obstetric abdominal pain as early diagnosis, classification of severity of AP and appropriate management can result in successful outcome.

Editor’s Comments

Acute pancreatitis (AP) although rare event in pregnancy is of great concern as it deals with two lives rather than one. Pregnancy related hematological and biochemical alterations influence the interpretation of diagnostic tests. Advances in clinical gastroenterology has improved outcome of pregnant patients with AP. This case emphasizes the importance of early diagnostic studies and proper therapeutic management to help reduce maternal mortality and foetal loss.

References