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Case Report

Acute pancreatitis in pregnancy: a rare case report

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ABSTRACT

Acute pancreatitis (AP) during pregnancy is a rare condition characterized by acute inflammation of pancreas due to premature activation of enzymes resulting in local pancreatic destruction and activation of an inflammatory cascade. This condition is most commonly caused by gall stones, hypertriglyceridemia, idiopathic factors. It's very rare, incidence being 1 in 1000 to 1 in 12,000 pregnancies. Its more common in multigravida (75%) and relatively uncommon in first trimester. Patient usually present with acute severe pain abdomen radiating to back, nausea, vomiting, fever and anorexia. On examination there can be tachycardia, jaundice, epigastric tenderness, abdominal guarding and rigidity in severe cases. Serum amylase and lipase levels three times upper limit of normal value is diagnostic. Ultrasound remains imaging modality of choice. Vigorous fluid replacement is recommended. Prompt diagnosis and early treatment can prevent maternal and fetal morbidity and mortality. 23 years primigravida with 14 weeks of pregnancy came to outpatient with nausea and vomiting (10-15 episodes per day) and pain abdomen for 3 days. On abdomen examination epigastric tenderness was present. Serum amylase was 1246 Units/litre and serum lipase was 507.3 units/litre. She was given supportive care with IV fluids. Analgesics, antiemetics and antihistaminics were given. Gradually liquids were started and she recovered in 5 days.

Keywords: Acute pancreatitis, Pregnancy, Serum amylase, Serum lipase, Gall stones

INTRODUCTION

Acute pancreatitis (AP) is a condition where there is acute inflammation of pancreas due to premature activation of enzymes zymogen and trypsinogen. This triggers local pancreatic destruction and activation of inflammatory cascade leading to systemic inflammatory response syndrome (SIRS) and multiorgan dysfunction syndrome (MODS). AP during pregnancy is considered a very rare condition, with incidence ranging from 1 in 1000 to 1 in 12000 pregnancies.[1] Diagnosing AP during pregnancy may be challenging due to physiological changes that occur during pregnancy. Earlier maternal and fatal mortality was high (20% and 50%) but now because of technical advances in imaging and therapeutic endoscopy the mortality rate has reduced to 0-5%. Correct and timely diagnosis with early intervention may improve the prognosis.

CASE REPORT

A 23 years primigravida with 14 weeks of pregnancy came to out-patient with nausea and vomiting (10-15 episodes per day) and pain abdomen for 3 days. There was no history of bleeding per vagina.

On admission she was conscious coherent with BMI-21.2, pulse rate of 100/min, blood pressure of 100/70. On per abdomen examination epigastric tenderness was present. There was no guarding, rigidity, bowel sounds were sluggish. On per vagina examination bilateral fornices were free, there was no cervical motion tenderness, no bleeding per vagina.

At admission blood tests were done in which complete blood count, liver function tests, renal function tests, serum electrolytes, lipid profile, blood sugar and serum calcium were normal. Serum amylase was 1246 units/litre

and serum lipase was 507.3 units/litre. HIV, HBsAg, HCV and covid antigen were negative. USG showed single live intrauterine fetus of 13 weeks 5 days gestation, NT of 1.3, normal nasal bone with no gall bladder calculi, normal common bile duct and pancreas were bulky with thin peri pancreatic fluid and minimal free fluid in peritoneal cavity suggestive of pancreatitis.

She was given supportive care with IV fluids. Analgesics, antiemetics and antihistaminics were given. On second day sips of liquids were given. On 3rd day all blood tests were repeated again and were normal. Serum amylase was 452 units/litre. Epigastric pain reduced and vomiting stopped on third day. On 4th day oral fluids were commenced. On 5th day soft diet was started and patient was discharged on 6th day. After conservative management patient didn't have any recurrences. TIFFA was done at 21 weeks and growth scan at 28, 32 and 36 weeks. She went in labour spontaneously and delivered a female child of 2.86 kg.

DISCUSSION

Diagnosis of AP is based on revised Atlanta criteria which requires presence of 2 of following 3 criteria[2]: Acute pain abdomen or tenderness in upper abdomen, elevated levels of pancreatic enzymes (serum amylase and/or lipase ≥3 times upper limit of normal value and presence of abnormal imaging findings in pancreas that are associated with AP.

Table 1: Causes of AP in pregnant women.[4]

S. no.	Causes of AP in pregnant women
1	Gall stones (65-100%)
2	Alcohol induced (5-10%)
3	Familial hypertriglyceridemia-induced pancreatitis (5%)
4	Idiopathic (15%)
5	Drugs-induced AP (thiazide diuretics)
6	Pancreatitis associated with pregnancy-induced hypertension
7	Acute fatty liver of pregnancy
8	Hyperparathyroidism (hypercalcemia)
9	Gene mutations-cationic trypsinogen (PRSS1)
10	Cystic fibrosis transmembrane conductance regulator (CFTR)
11	Pancreatic secretory trypsin inhibitor (PSTI)
12	Peroxisome proliferator-activated receptor gamma (PPARG)

Incidence

AP during pregnancy is considered a very rare condition, with incidence ranging from 1 in 1000 to 1 in 12000 pregnancies. Its more common in multigravida (75%) and relatively uncommon in 1st trimester (12%). Incidence increases with increasing gestational age. Incidence during 1st, 2nd and 3rd trimester is 19, 26 and 50% respectively and accounts for 38% during post-partum period.[3]

Pathophysiology

GB disease[5]

During pregnancy estrogen causes increase in cholesterol secretion and increase in percentage of cholic acid during second and third trimesters leading to supersaturation of bile.

Progesterone on the other hand , causes smooth muscle relaxation, leading to delayed gall bladder emptying and increased pressure in sphincter of oddi leading to bile stasis. Gallstones getting lodged in constricted distal end of Ampulla of Vater, block flow of both bile and pancreatic juice in duedenum.

Hypertriglyceridemia[6]

Certain pre-existing factors predidpose an individual to dyslipidemia. They include excessive weight gain, diabetes, alcohol consumption, drugs (steroids, diuretics and beta-blockers) and ceratin latent genetic abnormalities (lipoprotein lipase, apoC2 or apoE). Women having history of familial hyperlipidemia develop hypertriglyceridemia especially in third trimester during pregnancy. Some may develop hypertriglyceridemia without any predisposing factor as well. Due to effect of estrogen, triglyceride concentration rises 2.5-3 times above pre-pregnancy levels specially during third trimester. The usual total serum triglyceride level during pregnancy is less than 300 mg/dl while the levels required to induce AP is between 750-1000 mg/dl, with levels usually exceeding 1000 mg/dl.

Presentation

Patient usually present with acute severe pain abdomen radiating to back. Additional symptoms include nausea, vomiting, fever and anorexia. Physical examination findings include tachycardia, jaundice, epigastric tenderness, sluggish bowel sounds. In severe cases there can be abdominal guarding and rigidity, patient can be in shock and coma as well. Specific physical findings point towards a particular cause for instance jaundice suggests biliary origin, spider angiomas point towards alcoholism, Xanthomas and lipemia retinal suggest hyperlipidemic pancreatitis, Cullen sign (periumbilical bruising) and grey-turner sign (flank discoloration) suggest hemoperitoneum.

Diagnosis

Diagnosing AP during pregnancy may be challenging due to physiological changes that occur during pregnancy.

Workup includes complete blood count to assess for leukocytosis which may suggest infection.

Liver function test where ALT >500 U/L may suggest gallstone pancreatitis and fulminant disease. Renal function test including serum electrolytes, serum calcium,

blood sugar, serum lipid profile to exclude triglyceridemia, serum lipase and serum amylase raised 3 times the upper limit of normal value. Amylase to creatinine clearance ratio >5% suggests pancreatitis.[7]

Serum lipase-rises 4-8 hours after the onset of symptoms and remains elevated for days. It has better sensitivity (94% vs 83%) and specificity (96% vs 88%) then serum amylase because of longer persistence after an attack.

Serum amylase-rises immediately and peaks in a few hours and remains elevated for 3-5 days. Elevated levels are seen in other conditions like cholecystitis, cholangitis, peptic ulcer, ectopic pregnancy, kidney stones, intestinal obstruction. It may be normal in severe attacks and falsely negative values may be seen in hyperlipidemic patients.

Imaging

Remains a controversial issue because of effect of radiation on developing fetus.

Ultrasound

Ultrasound remains the preferred modality during pregnancy due to concerns of effect of radiation on developing fetus. It can differentiate normal appearing pancreas from the one that is enlarged and also identify areas of necrosis which is visible as hypoechoic regions.

It can identify other causes of acute abdomen like gall stones, renal calculi, appendicitis.

Findings suggestive of AP are increased pancreatic volume and decrease echogenicity due to fluid exudation leading to heterogenicity of parenchyma

***Magnetic resonance cholangiopancreatography* (MRCP)**

It is indicated when other non ionizing forms of imaging studies are dubious. In pregnant patients it should be done without contrast medium(gadolinium). It studies pancreatic parenchyma and duct in detail and rules out pseudocysts, inflammation. It can also rule out common bile duct stones and can also be used to remove them.

Endoscopic ultrasound

It has better diagnostic ability for common bile duct stones and in same sitting stone removal along with sphincterotomy can be done with ERCP. Drawback of this is that it's an invasive procedure and general anaesthesia is required.

Endoscopic retrograde cholangiopancreatography (ERCP)

Initially it was introduced as a diagnostic procedure but over the years it has evolved into a predominantly therapeutic tool. It is not very frequently used in pregnancy because of risk of radiation exposure to fetus.

Severity of AP

AP is classified into mild and severe forms based on its severity. Almost 80% of patients develop mild disease (Interstitial pancreatitis) and 20% severe disease (necrotizing disease). Severe pancreatitis necessitates intensive care and is associate with higher mortality while mild pancreatitis generally improves within 5-7 days.

Several clinical indices can help assess the severity of disease such as Ranson's criteria, BISAP criteria, ATLANTA, APACHE and bedside assessment of severity according to which disease is mild if there is no rebound tenderness, guarding of the abdomen, hematocrit and creatinine levels are normal. The Ransons criteria consists of total 11 parameters with a maximum score of 11. Five are assessed at admission and other six at 48 hours of admission. Patients with higher Ransons score have higher mortality rate. The modified criteria have a max score of 10.[6,8,9]

Management

AP during pregnancy requires a multidisciplinary approach. Combined care from gastroenterologist, surgeon, intensivist and obstetrician is essential. Admission is compulsory for all patients. Worsening parameters may necessitate ICU care. Supportive care is essential including vigorous fluid resuscitation, pain relief and early nutrition therapy. In severe cases ICU care may be necessary. There is no role of the prophylactic antibiotics.

For mild pancreatitis patients are kept nil by mouth to give rest to bowels. Intravenous fluids, analgesics, antiemetics are given. Correction of electrolyte and metabolic abnormalities is necessary. Once pain and tenderness decrease oral intake is resumed. Most patients recover within 3-7 days and need no further therapy.

Severe AP necessitates ICU care, where pulmonary, renal, circulatory and hepatobiliary support is given. Patients are kept nil by mouth and are fed through naso jejunal tube. Naso jejunal feeding is preferred to Total Parental Nutrition because it induces pancreatic secretion and total parental nutrition causes gastric mucosal atrophy and bacterial translocation.

Fluid resuscitation

Vigorous fluid resuscitation remains cornerstone of therapy due to substantial amount of fluid loss to third space.[8] It is started early, immediately after admission and is given as bolus dose followed by continuous infusion. Both crystalloids and colloids can be used but according to American gastroenterologist association crystalloids are preferred. Among crystalloids Ringer lactate is preferred

over normal saline.[9] Colloids are used when hematocrit and serum albumin is low.[10] Urine output of 40-50 ml/hr is used as a marker of adequate hydration. Attention should be paid to not overhydrate the patient which might lead to pulmonary edema and hypoxia.

Nutrition

AP is a hypercatabolic state. Therefore, nutrition plays a vital role in management. Early nutrition therapy modulates stress response, decreases risk of bacterial translocation and infection and aids in early resolution of disease. Enteral route is preferred. Nutrition should be started early and increased gradually. Enteral feeding is started with liquids then patients are allowed to have soft diet and then regular diet. For severe disease naso jejunal feeding with low fat formulation is recommended.

Interventions in AP

Cholecystectomy

Gall stone are the most common cause for AP. Definitive treatment is surgery. Choice of procedure either laparoscopic or open cholecystectomy depends on surgeon. However, laparoscopic cholecystectomy is preferred. It's safe in the second trimester as by this time fetal organogenesis will be completed and size of the uterus will not be too large so as to interfere with the procedure.

Table 2: Material complications.

S. no.	Maternal complications
1.	Mortality-Earlier maternal and fatal mortality was high (20% and 50%) but now because of technical advances in imaging and therapeutic endoscopy the mortality rate has reduced to 0-5%
2.	Recurrent pancreatitis
3.	Pancreatic pseudocyst diabetes
4.	Generalized peritonitis
5.	Adult respiratory distress syndrome (ARDS)

Table 3: Fetal complications.

S. no.	Fetal complications
1.	Preterm labor
2.	Intrauterine growth retardation
3.	Intrauterine death

ERCP

It is a combination of endoscopic and fluoroscopic procedures. Endoscope is introduced into second part of the duodenum and through major duedenal papilla instruments are passed into biliary and pancreatic duct through which abnormalities of bile duct, pancreatic duct and ampulla are investigated. It removes impacted stones in CBD, prevents cholestasis, bacterial infection and improves symptoms. Drawback of this procedure is radiation exposure to baby; therefore, fetus and pelvis should be covered with lead shield.

Complications[7]

Pregnancy outcome with AP

There are no standard guidelines regarding the mode of delivery with AP complicating pregnancy. It is not a contraindication to vaginal delivery and delivery is not routinely indicated in all patients with pancreatitis. Mode of delivery should be based on obstetric factors. Steroids should be given for fetal lung maturity and $MgSO_4$ for fetal neuroprotection, when indicated.

Prevention and precaution

Table 4: Prevention and precaution.

S. no.	Prevention and precaution
1.	Healthy lifestyle
	Balanced diet and avoiding high fat meals
	Regular exercise
	Avoiding excessive alcohol consumption and smoking
2.	Maintaining healthy weight (obesity and high levels of triglycerides are known risk factors for pancreatitis)
3.	Adequate hydration (prevents formation of gallstones)
4.	Regular prenatal care
5.	Proper control of blood sugar in patients with gestational diabetes. High blood sugar levels can increase the risk of developing pancreatitis.
6.	Medication review in prenatal period
7.	Genetic counselling in patients with women with genetic mutations

CONCLUSION

AP is an important clinical entity in pregnancy with diverse causes. A multidisciplinary approach along with correct diagnosis and proper treatment can prevent maternal and fetal morbidity and mortality.

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