Acute pancreatitis with eclampsia-preeclampsia syndrome and poor maternal outcome: two case reports and review of literature

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Abstract

Acute pancreatitis in pregnancy is a rare entity and has been reported to be associated with preeclampsia in the literature. Fulminant pancreatitis may have a guarded prognosis despite intensive multidisciplinary management. Two cases of maternal mortality in women with acute pancreatitis noted in the setting of preeclampsia-eclampsia syndrome are reported here.

Keywords

Pancreatitis, pregnancy, preeclampsia, eclampsia

Introduction

Acute pancreatitis complicates 1 in 1000–3000 pregnancies.¹ It usually occurs during the third trimester¹ or early postpartum period and is most commonly caused by cholelithiasis.^{2,3} Maternal and fetal mortality have shown a declining trend over the years with early diagnosis and intensive care management. Acute pancreatitis can be managed conservatively but surgery may be considered in unresponsive cases. Mild acute pancreatitis has a good prognosis with conservative management but fulminant acute pancreatitis carries a poor prognosis despite good supportive care. We present two cases managed at Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh, a teaching and a tertiary referral hospital of north India where both women illustrate the acute presentation of pancreatitis associated with eclampsia, requiring intensive care support, multidisciplinary team and poor maternal outcome.

Case 1

A 20-year-old primigravida presented at 37 weeks gestation with a two-day history of acute epigastric pain with associated vomiting and high-grade fever with chills. On examination she was found to be febrile, with tachycardia, blood pressure of 160/114 mmHg and extreme tenderness in epigastrium with abdominal distension. The fetus was live at admission with fundal height corresponding to 36 weeks. A clinical diagnosis of severe preeclampsia with acute abdomen was made. Blood results are shown in Table 1. Anuria was noticed after admission with deranged renal function tests and metabolic acidosis. Opening CVP was 3 cm. She had a generalised tonic clonic convulsion 4 hours after admission. An LSCS was performed in view of eclampsia with suspected surgical cause of acute abdomen. Serum amylase was 3810 IU/L making a diagnosis of acute pancreatitis. Intraoperatively, around 500 mL of serous fluid was noted in the abdominal cavity with chalky white calcifications over the omentum. Mesentery was chalky and thickened, suggesting pancreatitis (Figure 1). A live born girl (2.5 kg with APGAR 2,5,7) was delivered. Gall bladder and common bile duct were normal on palpation. A feeding jejunostomy was created with drains placed in the lesser sac. She required haemodialysis in the immediate postoperative period. She remained in ICU on supportive ventilation. Anuria with metabolic acidosis and fever persisted in the postoperative course and she received nine cycles of haemodialysis over a period of 16 days. Computerised tomography (CT) revealed an oedematous pancreas with extensive peripancreatic inflammation and ascites. Fever persisted in the postoperative period

with total leucocyte count up to 32,000 c/mm³ despite antibiotic cover with Imepenam, Vancomycin and Flucanazole. There was no improvement in the renal function and anuria persisted despite dialysis. She required ionotropes on postoperative day 9. Chest X-ray showed bibasal atelectasis and bilateral pleural effusions. She succumbed to refractory acidosis. Histopathology of the omental biopsy revealed fat necrosis with inflammation.

Case 2

A 26-year-old primiparous woman presented at postpartum day 2 following antepartum eclampsia with acute renal failure. She had been diagnosed with preeclampsia at 28 weeks gestation and had eclampsia at 31 weeks. Labour was induced with misoprostol followed by oxytocin and she delivered a still born baby of 1.3 kg. On day 2 postpartum, she had anuria with abdominal distension; renal function tests were deranged (Table 2). Ultrasonogram revealed ascites with bulky and heterogenous pancreas with distended gall bladder and postpartum uterus. Serum amylase was 1200 IU/L and lipase was 1600 IU/L. She required ventilatory support because of metabolic acidosis 6 hours after admission and received intensive care monitoring. CT revealed gross ascites with bilateral pleural effusions and around 30% necrosis of the parenchyma in the proximal body of pancreas with an ill-defined acute peripancreatic necrotic collection extending into the lesser sac. Conservative management with supportive treatment was continued. Thromboprophylaxis was not instituted in view of her deranged coagulation. On day 10, fever was persistent with temperatures up to 40 degrees despite administration of Imepenam, she had a sudden collapse and succumbed to suspected pulmonary thromboembolism.

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Table 1. Laboratory parameters on postop day 0 and postop day 18 (Case 1).

	Postop D1	Postop D18
Hb (g%)	12.0	7.8
TLC (cells/mm ³)	16,200	18,400
Platelets (cells/mm ³)	47,000	72,000
PTI	91%	58%
APTT (sec)	34	52
Urea (mg %)	49	121
Creatinine (mg %)	1.7	4.8
K ⁺ (mMol/L)	5.5	6.6
Ca ²⁺ (mMol/L)	5.6	8.2
Total bilirubin (mg %)	0.9	1.8
SGPT/SGOT/ALP (IU/mL)	56/76/342	67/82/435

TLC: Total Leucocyte Count; PTI: Prothrombin Index; APTT: Activated Partial Prothrombin Time; SGPT: Serum Glutamic Pyruvic Transaminase; SGOT: Serum Glutamic Oxaloacetic Transaminase; ALP: Alkaline Phosphatase.

Table 2. Laboratory parameters on postnatal day 0 and postnatal day 11 (Case 2).

	Postnatal D0	Postnatal D11
Hb (g%)	12.0	6.8
TLC (cells/mm ³)	15200	21700
Platelet count (cells/mm ³)	1.72L	1.5L
PTI	100%	50%
APTT (sec)	32	58
Urea (mg %)	45	119
Creatinine (mg %)	1.5	5.8
K ⁺	4.4	5.2
Ca ²⁺ (mMol/L)	7.2	9.4
Total bilirubin (mg %)	1.2	4.2
SGPT/SGOT/ALP (IU/mL)	56/74/298	109/88/423

TLC: Total Leucocyte Count; PTI: Prothrombin Index; APTT: Activated Partial Prothrombin Time; SGPT: Serum Glutamic Pyruvic Transaminase; SGOT: Serum Glutamic Oxaloacetic Transaminase; ALP: Alkaline Phosphatase.

Figure 1. Intraoperative pictures showing calcific deposits over omentum and saponification associated with pancreatitis.

Discussion

Acute pancreatitis is commonly associated with gall stone disease, hypertriglyceridaemia and alcohol. In pregnancy it appears to be more prevalent with advanced gestational age¹; postpartum occurrence has also been reported as in our case.⁴⁻⁶

Pancreatitis has rarely been reported in association with preeclampsia-eclampsia syndrome.⁵⁻¹⁰

Possible explanations of such an association could be attributed to the microvascular changes of preeclampsia: Microthrombi, intravascular coagulation and vasculitis, possibly affecting the pancreatic vessels. Badja et al.⁷ have reported a case of acute pancreatitis presenting with eclampsia and attributed it to the vascular changes and hypoxia due to preeclampsia. Both cases reported here had similar presentations of eclampsia with renal failure and acute pancreatitis. The association of these together complicates management and leads to poor maternal and fetal outcome.

These women usually require conservative ICU care which includes providing pancreatic 'rest' with intravenous fluid supplementation and antibiotics. Intravenous fluid supplementation corrects the

fluid losses and provides bowel rest and suppresses the function of the exocrine pancreas. The role of antibiotics is controversial; prophylaxis is advocated to prevent the infection of pancreatic necrosis which is a leading cause of morbidity and mortality. If initial conservative management fails then surgical intervention may be indicated. Surgical treatment of pancreatitis has two aspects, which include operative intervention for the disease itself and surgical treatment of associated biliary tract disease after the acute inflammation subsides. Intensive care monitoring with multidisciplinary approach improves outcomes.

Mild acute pancreatitis is conservatively managed and carries a good prognosis. Acute severe pancreatitis is associated with multi-organ dysfunction. Necrotising pancreatitis complicates approximately 10% of acute severe pancreatitis and is associated with mortality rates up to 80% despite the best possible care. Mortality in acute severe pancreatitis has been reported in the literature from developed countries^{4,7,11,12} and the incidence has been reduced with better understanding and management. However in developing countries maternal and perinatal outcomes are guarded and therefore it should be considered an absolute emergency.

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Guarantor

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Contributorship

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