Abstract: Acute pancreatitis is a rare event in pregnancy, which can have a high maternal mortality and fetal loss. Gallstone disease is thought to be responsible for about 70% of these cases. Here, we present two cases of acute pancreatitis with preeclampsia-eclampsia in the third trimester of pregnancy which was initially not diagnosed as pancreatitis and as a result we lost one patient. Investigation undertaken led to the diagnosis of acute pancreatitis.

Keywords: Acute pancreatitis, Pregnancy induced hypertension, Pregnancy complications, Intensive care unit

1. Introduction

Acute pancreatitis in pregnancy (AIP) is a rare life-threatening condition which poses a real challenge for clinician to diagnose it early. The annual incidence of acute pancreatitis in general population is 5 to 80 per 100,000. However in pregnancy, it varies and is approximately 1 in 1000 to 1 in 10,000[3,4]. Older reviews of AIP in pregnancy reported maternal and fetal mortality rates as high as 20% and 50% respectively. More than 50% of cases in pregnancy are diagnosed in third trimester demonstrating that acute pancreatitis is more common with advancing gestational age, paralleling the frequency of gallstones in pregnancy[1,5,6]. The commonest reasons of AIP are biliary disease and congenital or acquired hypertriglyceridemia, and very rarely AIP is associated with preeclampsia-eclampsia or HELLP syndrome[7]. Acute pancreatitis rarely complicates pregnancy, can occur during any trimester but over half (52%) occur during the third trimester and rarely during post-partum period. Although the diagnostic criteria for AP are not specific for pregnant patients, it’s very useful for the prediction to have an early diagnosis and treatment for AIP patients. Preeclampsia is associated with microvascular abnormalities that may involve cerebral, placental, hepatic, renal and splanchnic circulation and rarely can cause acute pancreatitis. Pancreatitis often presents as an acute abdomen and can have lethal effect on the mother and the fetus. It is important to be aware that AIP may be more severe, posing a survival threat. Early diagnosis and good supportive care by multidisciplinary team are crucial to ensure good maternal and fetal outcomes.

2. Case 1

A 25-year-old primigravida was admitted in Intensive care unit (ICU) at 32 weeks of gestation because of headache, vomiting, massive abdominal distension and unconsciousness. On admission in ICU, her vitals were: Pulse rate 106/min, blood pressure 220/140mmHg, respiratory rate 32/min. On Per Abdomen examination she was 32 weeks with cephalic presentation with fetal heart rate not localized. On Per vaginal examination os was closed, cervix uneffaced, firm in consistency and was posteriorly directed. Laboratory investigations were: Hemoglobin (Hb) 11g%, Total leukocyte counts (TLC) 13,800/mm², proteinuria (3+), Platelets 1,64khs/ mm², Bleeding time 5mins, Clotting time 8mins, prothrombin time 13.5s, Blood urea 22mg/dL, S.creatinine 1.4mg/dL, S. Sodium 135meq/L, S. Potassium 5.0meq/L and low serum albumin of 24 g/L. She had low serum calcium of 1.43 mmol/L, and elevated liver enzymes (aspartate and alanine aminotransferase values of 131 and 170 units/L). Fundus examination showed optic disc edema. She was induced with 50 micrograms of vaginal misoprostol 4hrly maximum upto 200 micrograms. She underwent a Cesarean section for nonprogress of labour and delivered an intrauterine dead female fetus of 2.1kg, no retroplacental clot was present. 2.5 litres of peritoneal fluid was present Postoperatively she could not be extubated and shifted to ICU. Two hours later her Blood pressure falls to 80/50 mmHg and was treated with intravenous noradrenaline, dopamine, hydrocortisone. Ideally Pre-eclampsia and eclampsia had a better prognosis once the fetus is delivered, but this patient deteriorates and the cause of shock could not be made so USG abdomen and CT Scan was advised by physician. Ultrasonography (USG) abdomen showed pancreatic swelling involving body and head and fluid collection in the pancreatic area and free fluid in abdomen and pelvis. Serum amylase and lipase were 320U/L and 68U/L respectively. Cerebral CT scan showed cerebral edema whereas abdominal CT Scan showed hepatic hypodensities (2-day after admission). Alkaline phosphatase, Aspartate and Alanine aminotransferase were 468, 131 and 170 units/L respectively. Pancreatitis was not suspected initially and serum amylase or lipase was not performed at the time of admission. Marked hypoalbuminemia and hypocalcemia were more likely secondary to severe acute pancreatitis that was not suspected initially. She could not be resuscitated and was expired on 3rd day due to severe pre-eclampsia with acute pancreatitis with refractory shock with cardiopulmonary arrest.

3. Case 2

A 25 years old woman was admitted at 32 weeks of gestation. Her pregnancy was uneventful until two weeks before admission when she noticed increasing distension of abdomen, severe epigastric pain, headache and swelling over lower limbs. She was G2P0+1L0 with the history of one delivery. She had a normal delivery 3 years back. On admission her vitals were: Pulse rate 90/min, blood pressure 170/110 mmHg, respiratory rate 18/min and was afebrile. On general
examination pallor and gross pedal oedema were present. On Per Abdomen examination she was 32 weeks with cephalic presentation with fetal heart rate of 134/min and fluid thrill was present. Local examination reveals gross vulval oedema. On Per vaginal examination os was closed and cervix uneffaced and pulled up with fullness in all fornices. Laboratory investigations were: Hemoglobin(Hb) 12.4g%, Total leucocyte counts ( TLC ) 8300/mm3, proteinuria (3+) Platelets 1.8lakhs/mm3, Bleeding time 5mints, Clotting time 5mints, prothrombin time 13.5s, Blood urea 22mg/dL, S.creatinine 1.4mg/dL, S. Sodium 131meq/L, S. Potassium 3.8meq/L,S.calcium 6.3mmol/L and low serum albumin of 20 g/L. Total bilirubin was 0.6, alkaline phosphatase was 367 with elevated liver enzymes (aspartate and alanine aminotransferase values of 109 and 110 units/L). Serum amylase and lipase values were 41units/L and 361units/L respectively. Fundus examination showed optic disc edema. Ultrasonography (USG) abdomen showed gestation of 31 weeks with features of intrauterine growth retardation and maternal pancreatitis with ascites and bilateral pleural effusion. There was no evidence of gall stones or biliary disease. Decision of delivery was taken and patient was induced with 50 micrograms of sublingual misoprostol 4 hrly and after 3 doses she was taken up for emergency caesarean section in view of non progress of labour and fetal distress. Per-operatively 3.0 litres of peritoneal fluid was present and she delivered a low birth weight female fetus of 1.1kg with low APGAR score. Postoperatively she was given injection calcium gluconate, intravenous antibiotics, Magnesium sulphate and antihypertensives. She recovered fully with resolution of pancreatitis with conservative management and was discharged in a satisfactory condition on 10th postoperative day.

4. Discussion

Acute pancreatitis complicates one in 1,000 to 3,000 deliveries [3]. Preeclampsia is associated with microvascular abnormalities that may involve cerebral, placental, hepatic, renal and splanchnic circulation. It is likely that pancreatic vasculature was also altered and caused acute pancreatitis that resulted in organized pancreatic necrosis in our patient. Pregnancy increases serum cholesterol and triglyceride levels, increases bile stasis, and thus may induce gallstone formation. However, hyperlipidemia may directly induce acute pancreatitis. Preeclampsia-associated pancreatitis can occur but is very rare [4,10]. In the patient described the diagnosis of pancreatitis was missed initially and first made by CT scan that was performed 2-days after the onset of symptoms and amylase levels checked on 4th and 7th day after the symptoms started were within normal limits. Serum lipase was not performed. However, the presence of organized pancreatic necrosis [11] highly suggests that symptoms, marked hypoalbuminemia, hypocalcemia occurring in the immediate peri-partum period were secondary to severe episode of acute pancreatitis. It is possible that the pancreatitis started before delivery but there were no symptoms of nausea, vomiting, abdominal pain and her initial calcium level was normal. There were no gallstones, her cholesterol and triglycerides levels were normal and there was no episode of hypotension or evidence of ischemia or vasculitis and no diuretics were used. The severe hypoalbuminemia out of proportion to the degree of proteinuria, hypocalcemia and generalized anasarca - as seen in this patient, should alert the physician to look for other inflammatory conditions that can result in capillary leak syndrome, including acute pancreatitis so that early and effective management be considered and complications could be avoided.

5. Conclusion

A case of pancreatitis associated with preeclampsia-eclampsia is presented where initial diagnosis was missed and resulted in fatal prognosis. Thus, it is mandatory to keep in mind, the rarest complication while managing the common diseases in order to give the best possible patient care.

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7. Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

