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Case Report

Hyperemesis Gravidarum: Looking Beyond Pregnancy

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Abstract

Hyperemesis gravidarum is defined variably as vomiting being sufficiently severe enough to produce weight loss, dehydration, ketosis, alkalosis from loss of hydrochloric acid and hypokalemia. It is not common for a patient affected with hyperemesis gravidarum to present with idiopathic acute pancreatitis in the same time period. This case pertains to a 22-year-old pregnant woman, admitted to our hospital with complaint of severe and frequent episodes of vomiting and severe epigastric pain at 14 weeks of gestation. After treatment with parenteral fluid and antiemetics, the patient's condition failed to improve. Further investigations confirmed a diagnosis of idiopathic acute pancreatitis. Patient was managed conservatively. Early and rapid diagnosis and supportive treatment play a vital role in the management of such cases.

Keywords: Hyperemesis Gravidarum; Pregnancy

1. Introduction

Hyperemesis gravidarum refers to intractable vomiting during pregnancy that leads to weight loss and volume depletion resulting in ketonuria and/or ketonemia [1, 2]. Etiopathogenesis of hyperemesis gravidarum is unknown. This condition is probably due to high serum levels of pregnancy related hormones such as human chorionic gonadotropin, estrogen, progesterone, leptin, placental growth hormone, prolactin, thyroxine and adrenocortical hormones. Rarely it may be caused by

systemic pathology which needs prompt diagnosis and treatment to decrease the morbidity.

2. Case Report

A 22yr old, primigravida with 14-week period of gestation, resident of New Delhi, initially presented to our hospital in the month of May 2018, with complaint of severe vomiting since 10^{th} week of gestation. The patient was admitted and was conservatively managed with parenteral fluids, antiemetics and nutritional supplementation. Medical, personal and family history was insignificant. General and systemic examination was normal. The hematological and biochemical investigations such as complete blood count, liver function tests, renal function tests, serum electrolyte, random blood glucose and thyroid profile were done at first visit which revealed no abnormality. Ultra-sonography showed a live intrauterine pregnancy of 14 weeks 4 days. Patient was discharged after 4 days but she was again with the same complaints of severe vomiting and epigastric pain 5 days later. She was started on parenteral nutrition and antiemetics but there was no significant improvement in her condition. Further laboratory investigations were done which revealed hypokalemia with serum potassium of 3.1 millimoles/lt. liver enzymes aspartate and alanine aminotransferase were raised to 484 and 518 I. U/l respectively. In view of severe unrelenting pain, serum amylase and serum lipase were done with values of 202 and 332 I. U/l respectively.

Ultrasound whole abdomen was done which showed signs of early pancreatitis. There was no evidence of any intra, or extra hepatic duct dilatation and the gallbladder was unremarkable. In the absence of evidence of any organic causes of acute pancreatitis, diagnosis of acute idiopathic pancreatitis was confirmed. Patient was kept nil orally with complete bowel rest. Along with Parenteral fluids and nutrition thiamine and potassium supplementations were given. Patient's condition gradually improved with reduction in the number of episodes of vomiting. Patient was advised to be discharged after 2 weeks. Her further antenatal course was uneventful till 30 weeks of gestation when she developed fetal growth retardation. Although hyperemesis by itself does not increase the risk of fetal growth restriction, the condition is significantly associated with multiple risk factors like psychological stress and poor nutrition through which hyperemesis indirectly increases the risk of fetal growth restriction. Patient was managed conservatively and was followed with monthly ultrasonography with doppler. Patient had preterm onset of labor at 36 weeks. She delivered a 1.8 kg male healthy baby. Postnatal course was uneventful and both the mother and the baby were discharged on day 3.

3. Discussion

Hyperemesis gravidarum has an incidence of 0.3-3%. Geographically, it is more commonly seen to occur in western countries [3]. It occurs in a very small proportion of pregnant women and very rarely it can be due to other medical complications such as acute pancreatitis. Acute pancreatitis in pregnancy is most often associated with gallstone disease or hypertriglyceridemia but rarely is idiopathic [4]. In such case, diagnosis is difficult due to overlapping symptoms of acute pancreatitis and severe hyperemesis of pregnancy. Hence, this condition is often misdiagnosed or undiagnosed resulting in delayed treatment, which may prove detrimental to the health of both the mother and the baby. Investigations like serum amylase, lipase, complete blood count, serum triglycerides, calcium and liver function tests are required for confirmation of diagnosis of acute pancreatitis. An elevated serum amylase level has a diagnostic sensitivity of 81% which further enhances to 94% by adding serum lipase [5]. Abdominal ultrasound can be considered an ideal

imaging technique for diagnosis of acute pancreatitis as it has no associated radiation risk, and it is useful in detecting dilated pancreatic ducts and pseudocysts. However, it poses a difficulty in diagnosis because an enlarged gravid uterus and combined ileus makes pancreas shadow invisible. Acute pancreatitis is generally managed conservatively (intravenous fluids, antibiotics, antispasmodic drugs and gastric decompression). However, surgical treatment may be done if there is pancreatic enlargement, necrosis and gastrointestinal perforation. Very few cases in literature are reported that shows association of hyperemesis of pregnancy with acute pancreatitis.

3.1 Management

Appropriate intravenous fluid and electrolyte replacement is the mainstay of therapy for hyperemesis gravidarum. Antiemetic drugs can also be given with vitamin supplementation. In patients unresponsive to conservative management systemic pathologies such as gastroenteritis, cholecystitis, pancreatitis, hepatitis, peptic ulcer disease, pyelonephritis, and the fatty liver of pregnancy must be excluded. The importance of psychological support cannot be underestimated. The patient must avoid foul smells and undesired foods that may trigger nausea and vomiting [6]. Besides antiemetic medications, pyridoxine appears to be more effective in reducing the severity of nausea [7]. Ondansetron, a category B drug, is a 5-hydroxytryptamine receptor antagonist which is found useful in treating cases with severe symptoms. Although serotonin is not implicated in the pathogenesis of hyperemesis gravidarum, ondansetron may be reserved for refractory cases [8]. In refractory patients, steroids may be used. In one such study, intravenous hydrocortisone was followed with oral prednisolone in seven patients with intractable hyperemesis gravidarum [9]. All patients responded to the treatment. In very severe cases parenteral nutrition maybe required [10]. After acute symptoms subside with initial therapy, enteral feeding may be considered an alternative approach in patients who cannot tolerate oral feeding despite antiemetic treatment [11, 12].

Aggressive intravenous hydration is suggested for the first 12 - 24 hours at the rate of 250 - 500 ml/ hour of isotonic crystalloid solution [13]. More aggressive hydration is recommended in patients with circulatory manifestations of severe dehydration such as hypotension and tachycardia. Fluid requirements should be monitored at frequent intervals by measuring blood urea nitrogen at least for next 48 hours [13]. Discontinuation of oral feeding results in cessation of pancreatic exocrine secretion and on the other hand patient's nutrition is supported either by total parenteral nutrition through central venous catheters or by enteral nutrition through a naso-jejunal catheter. The enteral feeding is preferred to total par enteric nutrition as central venous catheters' complications are avoided and the bowel continues to function which means that the gut flora maintains the enteric mucosal immunity [14]. Acute pancreatitis with mild symptoms usually resolves within a week and the patient can feed immediately as long as the nausea, vomiting and abdominal pain subsides [5, 13]. A low-fat solid diet appears as safe as a clear liquid diet [13]. Antibiotics which are classified on FDA pregnancy category B or C may be administered if required for extrapancreatitic infection (such as cholangitis, catheter-acquired infections, bacteremia, urinary tract infections, pneumonia) and infected necrosis [13].

3.2 Complications

Recurrent pancreatitis, pancreatic pseudocyst, diabetes, generalized peritonitis, adult respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC) multiple organ failure and death are well recognized complications of acute pancreatitis in pregnant patients. This may also lead to preterm

delivery and fetal loss. Pancreatic hemorrhage following pancreatic necrosis was also reported during pregnancy [14-21]. It is pertinent to note that serum lipase elevations may occur in hyperemesis gravidarum without pancreatitis also as observed by Amanda Johnson et al [22]. They postulated that disorders from obstructive and inflammatory bowels, intestinal infarction, duodenal ulcer, liver disease, abdominal trauma and probably hyperemesis can trigger production of lipase enzymes from these nonpancreatic sources. Thus, several disease processes other than acute pancreatitis can cause isolated elevation of serum lipase. Further all patients with hyperemesis should be vigilantly followed during pregnancy as they are prone to develop complications as reflected in the present case.

4. Conclusion

A high index of suspicion for other disease processes should be kept if patient is having intractable vomiting in pregnancy and is not relieved by traditional conservative measures. Patient should have a detailed workup with blood investigations along with radiological examination of the gastrointestinal tract and diagnosis of acute pancreatitis should always be confirmed by radiological evidence. Patients with other comorbid conditions should be followed throughout the pregnancy as they are susceptible for other complications like fetal growth restriction and preterm delivery.

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