Severe Hypertriglyceridemia - Induced Pancreatitis During Pregnancy

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We reported a case of acute pancreatitis associated with type V hyperlipoproteinemia. A 27-year-old woman was admitted at 25 weeks of gestation with persistent nausea, vomiting, progressive epigastric pain. She had very severe hypertriglyceridemia. Her serum amylase level was elevated. An ultrasound scan of abdomen was normal, with no evidence of cholelithiasis but there was perisplenic fluid accumulation. A diagnosis of acute pancreatitis secondary to hypertriglyceridemia was made. With early conservative treatment the patient’s condition was effectively improved and she was discharged from the hospital 8 days later. After 12 weeks, cesarean section was performed due to previous cesarean section. She was discharged home three days later with her baby.

Key Words: Pancreatitis, Pregnancy, Hyperlipidemia, Type V hyperlipidemia

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Introduction

Hyperlipidemic pancreatitis during pregnancy is rare but an important disorder which carries a high maternal and fetal morbidity and mortality.1 Most reports of such gestational hyperlipidemic pancreatitis suggest that pregnancy exacerbates preexisting hyperlipidemia.2,3 Type V hyperlipidemia is the most common type of lipid metabolic disorder related with acute gestational pancreatitis which is followed by type I hyperlipidemia. In pregnant women, hypertriglyceridemia should be ruled out as a cause of acute pancreatitis of unknown etiology.

We presented a case of acute pancreatitis due to type V hyperlipidemia in a pregnant woman and aimed to show that early diagnosis and treatment would have been life saving for the pregnant woman and her baby.

Case Report

The patient was a pregnant gravida 2, para 1, 27-year-old women who was admitted with persistent nausea, vomiting, progressive epigastric pain. Previously, she had an uncomplicated singleton pregnancy which was delivered by cesarean section for a breech presentation under general anesthesia. She had no history of diabetes mellitus, alcohol abuse, obesity, pancreatitis, gall stones, drug intake. The pregnancy proceeded without complication until 25 weeks’ gestation when she developed intermittent abdominal pain which became severe and localized to the epigastrium 48 hours before admission to hospital.

On physical examination, she had eruptive xanthomas. She had epigastric tenderness with rebound and uterus was soft but irritable. Blood samples taken resembled strawberry milkshake and were subsequently found to be grossly lipaemic. Laboratory examination revealed a serum triglyceride concentration of 1980 mg/dL (normal value 0-200 mg/dL), a serum total cholesterol concentration of 828 mg/dL (normal value 0-200 mg/dL). High density lipoprotein was found to be low, at 20 mg/dL (normal value 35-75 mg/dL). Lipoprotein electrophoresis led us to the diagnosis of type V hyperlipoproteinemia according to the WHO classification.4 Serum amylase was at 991 U/L (normal range 28-100U/L). Glycosilated hemoglobin was nearly normal, at 4.8 %, suggesting that hyperglycemia was a new onset. An ultrasound scan of abdomen was normal, with no evidence of cholelithiasis but there was perisplenic fluid accumulation. Obstetric sonography of the fetus showed normal growth with normal amniotic fluid index.

A diagnosis of acute pancreatitis secondary to type V hyperlipoproteinemia (familial hypertriglyceridemia) was made. The initial management was intravenous hydration, broad spectrum antibiotics and analgesia together with cessation of oral intake, nasogastric decompression and continuous glucose-insulin infusion. Treatment effectively improved the
patient’s condition. Serum amylase level decreased within 4 days from 991 U/L to 62 U/L. The patient was discharged as symptom-free with a triglyceride level of 769 mg/dL after 8 days. On discharge from the hospital, the patient was clinically stable; she was tolerating low fat diet. During 12 weeks following, her serum triglyceride and total cholesterol were higher than normal. She was admitted to our hospital in 37 weeks gestation with uterine contractions. Her serum triglyceride level was 1714 mg/dL. Serum amylase and lipase levels were normal. Because of uterine contractions, cesarean section was performed under spinal anesthesia and she delivered a female infant weighing 3240 gr. She was discharged home three days later with her baby. However, her serum triglyceride and cholesterol levels did not decline after the delivery and increased to 1487 mg/dL, 285 mg/dL respectively at postpartum two months. Fibrate therapy was started with low fat diet. The family members of patient were examined and serum triglycerides of her sister and mother were also found higher than normal. She has been still followed as an outpatient in gastroenterology department.

Discussion

Hypertriglyceridemia accounts for 4-6% of all cases of pancreatitis complicating pregnancy. The association between pancreatitis with hyperlipidemia in pregnancy was first reported in 1818. Gestational pancreatitis is probably caused by hyperlipidemia exceeding 22 mmol/L (2000 mg/dL). Miller et al published one of the largest retrospective studies wherein they followed 35 patients with hyperlipidemia. They found that triglyceride levels above 6000 mg/dL were associated with severe abdominal pain and pancreatitis. But unfortunately there is not absolute limit of hypertriglyceridemia to predict the development of pancreatitis. During pregnancy; low density lipoprotein, serum triglycerides and very low density lipoproteins increase physiologically. On the other hand, the metabolic changes of pregnancy may induce profound elevation of plasma triglycerides in women who have pre-existing disorder of lipoprotein metabolism. Type V hyperlipoproteinemia is the most common type of lipid metabolic disorder related with acute gestational pancreatitis which is followed by type I hyperlipoproteinemia. Bildirici et al reported a severe hyperlipidemic pancreatitis associated with type V hyperlipoproteinemia. Familial hypertriglyceridemia is inherited as autosomal-dominant trait and usually manifest in adulthood. The genetic mutations are unknown yet, and the diagnosis is a clinical one, mainly by demonstration of lipoprotein abnormalities by family history or family studies. A fasting lipid profile obtained in early pregnancy is recommended.

There is no specific treatment of pancreatitis. Supportive care remains the mainstay of therapy. If diagnosed early, intravenous hydration, dietary restriction of fat are adequate interventions. Gürsoy et al reported a patient with severe hypertriglyceridemia induced pancreatitis during pregnancy treated successfully with continuous intravenous insulin glucose infusion and cessation of oral intake.

Hemodialysis, plasma exchange, immunospecific apheresis have been used for treatment of resistant cases. In the rare patient, pancreatic necrosis may develop which necessitates operative management. Termination of pregnancy is rarely indicated and doesn’t seem to be an influence on maternal outcome.

Finally, one should be aware of that the incidence of pancreatitis during pregnancy is low, but related morbidity and mortality are high. Early and prompt treatment is the key factor to successful outcome. And the patients with hypertriglyceridemia related with genetic or familial origin must be followed carefully in their pregnancies because pregnancy exacerbates hyperlipidemia.

Gebelikte Hipertrigliserideriminin İndüklediği Pankreatit Olgusu

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References


