Severe gestational hypertriglyceridemia, a rare cause of pancreatitis in pregnancy: a case report

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Background

Acute pancreatitis is a rare but serious cause of maternal and fetal mortality in pregnancy. The most common occurrence is in the third trimester or in the postpartum period.[1] Normal pregnancy involves increased glucose production, progesterone synthesis and lipogenesis while causing decreased lipolysis to ensure requirements of a growing fetus.[2] Serum triglycerides often peak in the third trimester but rarely exceed 3.3mmol/L or 300 mg/dL which is not sufficient to cause acute pancreatitis. Abnormal lipid metabolism in pregnancy women may lead to severe gestational hypertriglyceridemia is defined as triglycerides greater than 11.4mmol/L or >1,000 mg/dL.[3] Levels greater than 11.4mmol/L significantly increases the risk of acute pancreatitis in pregnancy.

Case

The case is of a G2P1 25 year old female who presented with severe upper abdominal pain at 35 weeks gestation. She had a previous uncomplicated cesarean delivery, appendectomy, a body mass index of 35 and a history of unmedicated gestational hypertension. On presentation she reported a gradual onset of constant severe epigastric pain and fevers. Her temperature was 37.8, she was tachycardic and required four litres of oxygen by nasal prongs. On examination she had generalised abdominal tenderness and no peritonism. Investigations including ultrasound, cardiotocography and blood work was completed. Ultrasound showed no evidence of gallstones, no placental abruption, normal fetal growth and dopplers.

The diagnosis of pancreatitis secondary to hypertriglyceridemia became a differential diagnosis when the blood test was unable to be processed by the laboratory repeatedly. During recollection we noted that the blood tube showed lipid content in the blood. A recollection including a lipid profile was ordered A lipid profile revealed triglyceride levels greater than 40mmol/L and cholesterol 18.6mmol/L. She was commenced on an insulin-dextrose infusion for hypertriglyceridemia.

The patient had unmanaged pain requiring morphine. A multidisciplinary team discussion occurred including discussion regarding stillbirth risks, maternal wellbeing with unmanaged pain and the pregnancy impact on treatment decision for pancreatitis. A decision was made for an emergency caesarean section and diagnostic laparoscopy.
Intraoperatively oedema was noted adjacent to the descending portion of the duodenum and distal stomach suggesting pancreatic swelling. A chylous effusion was noted during diagnostic laparoscopy. She had an uncomplicated cesarean delivery and there was no neonatal concerns. She was commenced on piperacillin/tazobactam post surgery for concerns of intrabdominal sepsis.

Post delivery her triglyceride levels significant decreased to normal levels. Day 1 post operative her triglycerides reduced to 8.5mmol/L. The insulin infusion was ceased when triglycerides were below 11 mmol/L.Post surgery she had a postoperative ileus that resolved post operative day four and a PICC line was inserted for total parenteral nutrition. A computed tomography scan confirmed pancreatitis. She was discharged day five post operatively and made a full recovery.

In hospital she had a dietician review and was recommended a low fat diet. She was counselled regarding recurrence in future pregnancies, risk of adhesions due to concurrent inflammation from pancreatitis, life long cardiovascular risk, contraception and was referred to the endocrine and lipid clinic. Recommendations included commencement of omega 3 fatty acid tablets three times daily and fenofibrate 145 mg daily. She was recommended to repeat profile in 3 months postoperatively and prior to her next pregnancy.

Discussion

The pathogenesis of pancreatitis caused by hypertriglyceridemia is not completely understood. It is theorised that triglycerides accumulating around the pancreas are hydrolysed by pancreatic lipase causing high levels of free fatty acids that inflame acinar cells and the capillary endothelium. There are also increased chylomicron concentrations, which lead to capillary plugging, acidosis, and ischemia. In this acidic environment, free fatty acids activate trypsinogen and trigger acute pancreatitis. [4]
The diagnosis of acute pancreatitis in pregnancy can be a diagnostic dilemma as symptoms can mimic other diseases presenting with abdominal pain or labour. Acute pancreatitis can also trigger labour secondary to to peritoneal irritation. A differential diagnosis including myocardial infarction, peptic ulcer, appendicitis, cholecystitis, acute mesenteric ischemia, gastrointestinal or pancreatic cancer and pyelonephritis was ruled out. In addition, obstetric causes including preeclampsia, HELLP syndrome, placental abruption, acute fatty liver of pregnancy, uterine rupture was also ruled out. [5]

Management in this case included insulin infusion as it activates lipoprotein lipase which works rapidly to increase removal of triglycerides from the plasma and hence used to treat severe hypertriglyceridemia. However there is limited data on the use in patients without diabetes. Generally due to risk of hypoglycaemia it is not recommended in non diabetic patients. [5] Omega-3 fatty acids have a rapid onset of action in decreasing maternal triglyceride level by downregulating hepatic lipogenesis and it stimulates fatty acid oxidation in skeletal muscle and liver. Omega-3 fatty acids are safe in pregnancy for monotherapy but have only moderate effects. [4] Nicotinic acids have been used in case reports in first trimester of pregnancy without adverse effects to the foetus however there are limited studies later in pregnancy. Fibrates have limited use in pregnancy due to limited data and statins have potentially teratogenic effects.

This case highlights the importance of a multidisciplinary team approach and inclusion of severe hypertriglyceridemia causing pancreatitis as an important differential in pregnant women presenting with abdominal pain.

References

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