



Hypertriglyceridemia-Induced Acute Pancreatitis in Pregnancy

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Background:

- The incidence of acute pancreatitis in pregnancy is as low as about 3:10,000 – the most commonly attributable cause is gallstones; and hypertriglyceridemia implicated in an estimated 1-7% of those cases [1]
- Normally, elevated estrogen levels in pregnancy enhance lipogenesis and suppress hepatic lipase activity, while pregnancy-related insulin resistance contributes to increased lipolysis and decreased LPL activity [2]
- Severe hypertriglyceridemia in pregnancy (triglycerides >11.2 mmol/L) is uncommon and usually points toward an underlying genetic abnormality for example, monogenic disorders in genes affecting LPL levels or activity [2]
- Secondary factors such as insulin resistance, hypothyroidism, and glucocorticoids also play an important role

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Case Presentation:

- 35yo G6P5, no reported PMHx, prior CDx3, presented at 26-weeks GA for PPROM and found to have chronic abruption.
- Admitted to antepartum for expectant management, intermittently endorsing generalized abdominal pain and cramping.
- After a meal on HD#5, had intractable vomiting episodes and reported “burning” epigastric pain with radiation to back.
- Initial lab workup had to be resent because specimens noted to be lipemic. 
- Subsequently ruled in for acute pancreatitis (AP) with elevated amylase (201) and lipase (277). LFTs normal, abdominal U/S only showed fatty liver, small gallbladder polyp, and normal appearing pancreas. Triglyceride(TG) and LDL-cholesterol levels were **>5680mg/dl** and >600, respectively, both outside reportable range.
- Though patient was euglycemic, an insulin drip (with concurrent D10) was initiated.
- Patient admitted to ICU on HD#8 as became toxic-appearing, noted increased maternal and fetal tachycardia, and lack of response to escalating doses of IV morphine PCA.
- Continuous EFM and a C-section tray were at ICU bedside.
- A RIJ trialysis catheter was placed, and a single round of plasmapheresis was performed. There was subsequently a rapid decrease in TG level and improved pain scores. 
- She was initiated on fenofibrate, omega-3 fatty acids, and a strict low-fat diet once TG levels were <1000.
- As clinical symptoms improved, she was transferred back to antepartum service for continued PPROM surveillance.
- Ultimately, delivery was expedited as MCA dopplers worsened shortly thereafter (PUBS not indicated in setting of chronic abruption) and she had quaternary CD at 29-weeks GA performed under combined spinal-epidural anesthesia (procedure uncomplicated).
- Postpartum course was significant for development of a LUE DVT provoked by a midline catheter, and she was started on therapeutic lovenox. Chest CT was negative for PE, however a pancreatic pseudocyst was incidentally found (GI did not recommend drainage while inpatient).



Discussion

- Hypertriglyceridemia-induced acute pancreatitis (HTGAP) is a rare complication in pregnancy (and in the general population) that can portend maternal and/or fetal mortality in severe cases.
- Diagnosis can be difficult in pregnancy because of confounding obstetric etiologies (i.e. labor, uterine rupture, placental abruption, acute fatty liver of pregnancy) and other acute abdomen presentations.
- There are no strict guidelines for managing this condition in pregnancy, and most sources emphasize NPO, fluid resuscitation, pain control, and reduction of triglyceride levels (numerous case reports point to conflicting risks/benefits in utilizing insulin drip versus plasmapheresis).
- Clinicians must use best judgment in more severe presentations, ideally delaying delivery until triglyceride levels have returned to normal range and maternal condition has stabilized.

