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October Introduction

Anand Swaminathan, MD and Jan Shoenberger, MD

Take Home Points

- **While lipase elevation is not specific for pancreatitis, a level elevated more than 3 times above the upper limit of normal is more specific.**
- **Hypertriglyceridemia is a fairly common cause of pancreatitis.**
- **Patients with pancreatitis due to hypertriglyceridemia may be treated with plasmapheresis or insulin drip.**

- A 44-year-old male presented with 3 days of constant sharp epigastric pain radiating to his back. He had nausea and vomiting, but denied fevers. There was no history of similar symptoms in the past. He had no past medical history and was not on medications. He didn't smoke or drink. He was uncomfortable. He was tachycardic with a heart rate of 115 bpm and was tachypneic with a respiratory rate of 22 breaths per minute. His blood pressure was 118/65 mmHg. Oxygen saturation was 97% and he was afebrile. He had epigastric tenderness with guarding.
- **What is your differential diagnosis?** This presentation has a broad differential diagnosis. Perforated ulcer or viscus. Gallbladder pathology such as cholecystitis, choledocholithiasis, or gallstone pancreatitis. Pancreatitis. Myocardial infarction (although these patients are not usually tender to palpation).
- **They got an ECG.** They sent labs. His white blood cell count was elevated at 13. Platelets were elevated. LFTs were normal. The lipase was 1083.
- **Lipase elevation is not specific for pancreatitis.** However, when the lipase is elevated more than 3 times above the upper limit of normal, it is very specific for pancreatitis.
- **Should you get a CT?** Shoenberger doesn't get a CT on all patients with pancreatitis. She is more likely to get imaging if the patient is sicker.
- **Why does the patient have pancreatitis?** It can change the management and disposition. Patients with gallstone pancreatitis and necrotizing pancreatitis may be admitted to the surgical service.
- **The patient swore he didn't drink alcohol.** His wife confirmed he was not a drinker.
- **There were no gallstones on ultrasound.** He was not on medications. There was no report of scorpion bite. Hypercalcemia and hypertriglyceridemia can be an etiology.
- **Hypertriglyceridemia leading to pancreatitis is fairly common.** You can make this diagnosis at the bedside by looking at the separation of lipids in the tube. The triglyceride level was 2500 mg/dL (28.2 mmol/L).
- **What are you going to do about it?** There is increasing evidence that judicious fluid hydration is better than drowning the patient. Pain control.
- **There are two options for hypertriglyceridemia.** Plasmapheresis and insulin infusion. The insulin infusion is easier to arrange in the ED. Insulin infusions reduce the triglyceride levels and the synthesis of the triglycerides by accelerating their metabolism. This can rapidly lower the triglycerides. The insulin is usually given at a fixed rate of 0.25 units/kg/hr. You may have to infuse dextrose as well. The patient was transferred to the ICU and did well.
- **It is important to get the insulin drip started early to stop the process.**

Critical Care Mailbag: Acute Liver Failure

Anand Swaminathan, MD and Scott Weingart, MD

Take Home Points

- **Although patients with liver failure may have an elevated INR, it doesn't mean they are coagulopathic. Thromboelastography (TEG) or rotational thromboelastometry (ROTEM) might provide a more accurate assessment of coagulopathy.**
- **The role of prophylactic antibiotics in acute liver failure patients is unclear.**
- **Consider vasopressin when giving vasopressors.**
- **Contact your transplant center for transfer if you do not have these resources in-house.**

CASE

A 24-year-old woman presented with fever, nausea, vomiting, and abdominal pain. She had an extensive work-up and her liver function testing was markedly elevated. ALT was approximately 30000. AST approximately 20000. Bilirubin was 9. The INR was 2.1. The acetaminophen level was undetectable. The patient likely had acute liver failure from a viral hepatitis.

- **Is there a role for N-acetylcysteine (NAC) in the absence of acetaminophen overdose?** Yes. It is a free-radical scavenger and replenishes the stores of glutathione. Although literature is limited, it does seem to be effective for any cause of acute liver failure. There is little downside aside from cost.
  - **Is the regimen the same as acetaminophen overdose?** Yes, although you can continue the infusion beyond 23 hours.
  - **What are the indications for NAC?** It depends on the chronicity. If the patient has chronic liver failure with a bump in the LFTs, it is your usual cirrhotic patient and you do not have to give NAC. However, if the patient had normal liver function and is now in acute liver failure, give it empirically.
- **Do you treat the coagulopathy if they have bleeding from their puncture sites?**
  - Puncture site bleeding is arbitrary. INR elevation does not necessarily mean the patient will have increased bleeding.
  - INR measures Factor VII and is only supposed to be used for warfarin monitoring. Warfarin affects all of the vitamin K dependent clotting factors (II, VII, IX and X) and measuring just one of these factors is sufficient to determine the medication effect. However, in liver failure, the coagulopathy is not distributed evenly. Factor VII tends to be affected more than the other factors, which may be okay.
  - TEG or ROTEM may provide a better idea of the coagulation status.
  - If the patient has overt bleeding, you may treat potential coagulopathy with FFP or PCC.
- **Patients with cirrhotic liver failure have high risk of infection and there is benefit to prophylactic antibiotics in patients with GI bleeds. Is there any role for antibiotics in acute liver failure?** Antibiotics have a role in patients who could be infected or are infected. Weingart would not give antibiotics just for liver failure alone. You should give antibiotics to cirrhotic patients with GI bleeds.
- **What else should you consider in the management of acute liver failure?**
  - **Are you a liver transplant center?** If you are not, you should consider transferring the patient. Make sure you are speaking to the GI attending or transplant attending if the transfer is refused.
  - **The degree of encephalopathy may be worse in acute fulminant hepatic failure patients than in cirrhotics.** The relationship of ammonia levels to hepatic encephalopathy in cirrhotic patients is not well-defined. Encephalopathy in acute hepatic failure patients may be caused by acute increased intracranial pressure. Obtain a head CT. Evaluate the optic nerve sheath on ultrasound or CT.
    - If they have evidence of increased intracranial pressure, they may need hypertonic saline or mannitol. Avoid mannitol in a hypotensive patient. Mannitol itself has minimal effect on blood pressure but it can be difficult to keep up with fluid losses. If you give mannitol, you need to monitor urinary output and match any losses. You can give hypertonic saline to hypotensive or hypotonic patients.
    - You are likely going to intubate these patients. The patients may need ICP monitoring.
    - How do you monitor response to these agents if you don't have ICP monitoring? Most of these patients will be transferred to a facility with capabilities for ICP monitoring. You can use ultrasound to track changes in intracranial pressure with the optic nerve sheath diameter. Your goal is optic nerve sheath diameter less than 5-6 mm.
  - **Do they need additional fluids or keep them dry?** There isn't much evidence available. If Weingart is going to give more fluid than a little bit of crystalloid, he is quick to reach for albumin. Albumin has multiple benefits for liver failure patients. Cirrhotics do better with albumin with spontaneous bacterial peritonitis. Albumin has less potential for edema formation. Weingart may give 50-100g of the higher percentage albumin.
  - **These patients may develop renal failure.** They may need continuous renal replacement therapies.
  - **There is some enthusiasm for liver dialysis although not much supporting data.**
  - **If you need vasopressor support, you can use standard pressors.** Weingart likes vasopressin in liver failure patients. Chronic liver failure patients are vasopressin-deficient in general. It is unclear if acute liver failure patients are vasopressin-deficient. In general, these patients don't need much inotropic support, although they do tend to have low blood pressures. You don't need to go crazy to reach high normal in these patients.