Lipemic Serum in Hypertriglyceridemia-Induced Pancreatitis

Michael A. Santos, MD[1,2,3], Nisarg B. Patel, MD[2], and Carolina Correa, MD[2]

[1]Department of Medicine, Penn State Milton S Hershey Medical Center, Hershey, PA, USA; [2]WellSpan Good Samaritan Hospital, Lebanon, PA, USA; [3]Department of Medicine, WellSpan Good Samaritan Hospital, Lebanon, PA, USA.

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A 47-year-old male with hypertension presented with epigastric pain. He did not have gallstones and rarely drank alcohol. Physical examination revealed tachycardia and epigastric tenderness. Abdominal CT scan showed pancreatic stranding. Serum triglycerides were 3568 mg/dl (range 0–150 mg/dl). He was diagnosed with hypertriglyceridemia-induced pancreatitis (HTGP). After 12 h of intravenous insulin at 0.1 units/kg/h, a reduction in triglycerides was visually apparent (Fig. 1).

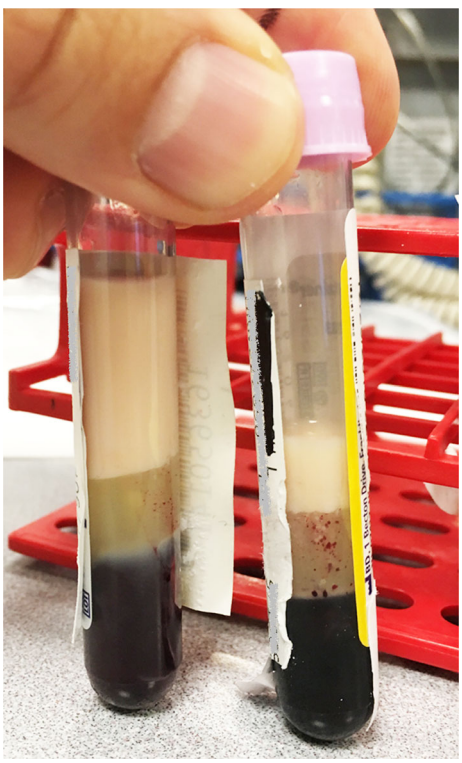

Figure 1 Unused blood samples showing a gross reduction in lipid volume from admission and after 12 h of IV insulin. After centrifugation, the lipemic layers settle above the gel separator layer

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Severe hypertriglyceridemia is the third most common cause of pancreatitis, after gallstones and alcohol.[1] Acute pancreatitis is rare when serum triglycerides are less than 1000 mg/dl. The pathophysiology of HTGP is unclear. Proposed mechanisms include increased blood viscosity from elevated levels of chylomicrons and the aggregation of free fatty acids, with subsequent pancreatic capillary occlusion leading to ischemic acinar cell injury.[2]

Acute management is supportive, with intravenous insulin, fluids, and dextrose until serum triglycerides are below 500 mg/dl. Insulin increases lipoprotein lipase activity, promoting chylomicron breakdown and clearance of serum triglycerides.[3] Heparin with insulin can be considered, but heparin monotherapy is not recommended.[4] When feasible, early plasmapheresis can reduce serum triglycerides by 65–70% in a single session.[5] Clinicians should also focus on dietary modification with early initiation of fibrates, niacin, and fish oil.

Corresponding Author: *Michael A. Santos, MD; Department of MedicineWellSpan Good Samaritan Hospital, Lebanon, PA, USA (e-mail: masantospitt@gmail.com).*

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