

## Lipemic sample: Is it worth our attention?

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### ABSTRACT

A 27-year-old male presented to the emergency room with abdominal pain and vomiting. While investigating, the blood sample collected was densely lipemic and repeatedly rejected by the analyzer. On deliberation, the sample was subjected to dilution and results showed hyperglycemia, hypertriglyceridemia (HTG) (>4000 mg/dL), and hyperamylasemia. Acute pancreatitis (AP) was confirmed on computed tomography scan of the abdomen. Lipemic sample while investigating abdominal pain in either pregnant women or patients with diabetes mellitus, hormone disorders, or chronic alcoholism, must prompt the diagnosis of AP (HTG induced pancreatitis). The laboratory should be aware of sample artifacts and efforts should be taken to convey the findings (both physical and biochemical) to the clinician. This case stresses the need for good communication between treating clinical and diagnostic faculties.

**Keywords:** Alcoholism, Diabetes, Hypertriglyceridemia, Lipemic, Pancreatitis

Approaching a case of the abdominal pain in an emergency is always challenging. Few are the conditions which can be objectively diagnosed like Alvarado scoring system for acute appendicitis [1]. It is the subtle signs on examination and clues taken from history taking that come to our rescue. Acute pancreatitis (AP) is often included in the differential diagnosis of abdominal pain. Gallstones and alcoholism are the most common causes of AP. Other causes include trauma, post (Endoscopic retrograde cholangiopancreatography), drugs, infections, hypercalcemia, tumors, ductal anomalies, autoimmune, and systemic causes [2].

Hyperlipidemia in the form of hypertriglyceridemia (HTG) is a rare but well-known cause and can result in episodes of recurrent AP. HTG may be primary or secondary to alcohol, diabetes mellitus, hormone intake, or pregnancy. The management includes supportive therapy as in any case of AP. However, unlike other causes, HTG-induced -AP can be suspected earlier by the lipemic nature of serum collected from patients. This case report describes the diagnostic journey in a case of abdominal pain arriving at HTG induced-AP, a well-established entity, although still a rarity in India [3].

### CASE REPORT

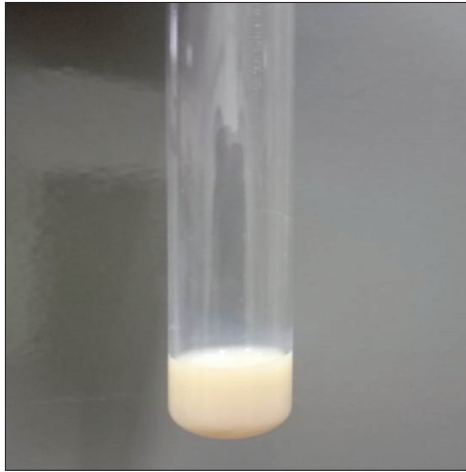
A 27-year-old male presented with complaints of abdominal pain and 2 episodes of vomiting over 10 h duration. The patient had taken meals 10 h ago. There was no history of fever, loose motions, constipation, obstipation, burning micturition, history of pain in the abdomen, or any significant comorbidity. Furthermore,

there was no history of tuberculosis or tuberculosis contact and alcohol consumption. On examination, there was tenderness in the epigastric region, but no guarding or rigidity. Rest of the systemic examination including vitals was unremarkable.

Considering the history of vomiting and epigastric abdominal pain, the patient was initially treated as a case of acute gastritis with proton pump inhibitors (injectable pantoprazole 40 mg). However, the pain was not relieved, and this prompted further investigations. Ultrasonography was scheduled, and blood was collected for laboratory testing. Complete blood count showed hemoglobin: 15.8 g%, total leukocyte count: 12400/cmm, and platelet count: 83000/ $\mu$ L. Interestingly, the serum, separated on standing, was observed to be milky-white in appearance (Fig. 1) and it was advised that a repeat fasting sample is collected, since, the machine was not able to analyze the "lipemic sample." As previously mentioned, the sample was, in fact, a fasting one. Another blood sample was collected but to no avail. It was dense lipemic. After much deliberation, the sample was subjected to dilution (1:8) and processed (Erba Chem  $\times$ 5). The results were as follows: Random blood glucose - 178 mg/dL, total cholesterol - 758 mg/dL, triglycerides (TG) - 4578 mg/dL, and serum amylase - >140 U/L.

Ultrasonography of the abdomen showed bulky pancreas while computed tomography scan (Fig. 2) showed edematous pancreas with loss of lobulation and minimal peri-pancreatic fluid predominantly around the pancreatic tail suggestive of acute interstitial pancreatitis.

The patient was treated conservatively with intravenous analgesia, fluids, and anti-emetics. He was referred to a



**Figure 1:** The sample separated was dense lipemic (milky white in appearance)



**Figure 2:** Axial sections of computed tomography scan of the abdomen showing edematous pancreas with loss of lobulation and minimal peri-pancreatic fluid predominantly around the pancreatic tail

higher center for expert opinion and evaluation of HTG and hyperglycemia. The patient was classified as 1 (bedside index of severity in AP) and monitored for 10 days. He was advised to avoid alcohol and hepatotoxic drugs and regular monthly follow-up. On follow-up (2 months after acute episode), ultrasonography was normal, and all the serum markers were unremarkable (serum amylase - 40 IU/L and total cholesterol/TG/low-density lipoprotein/high-density lipoprotein -253/334/155/31 mg/dL).

**DISCUSSION**

AP may be induced by HTG ([TG] levels >1000 mg/dL) and is responsible for approximately 9–10% cases [4]. It is commonly associated with diabetes mellitus or diabetic ketoacidosis, pregnancy and increased alcohol intake [5-7]. HTG may also be due to disorders of lipid metabolism and HTG-AP may be the heralding event, where no other coexisting cause could be elicited.

The exact mechanism is unknown, but the action of pancreatic lipase on TG and formation of free fatty acids leading to inflammation has been speculated. The clinical course of HTG-AP is no different than AP of other etiologies. The

management also does not differ much. Few studies have advised insulin (in diabetics), dextrose insulin infusion (in non-diabetics), and plasmapheresis [5]. However, the ultimate aim is reducing TG levels with dietary restrictions and lipid-lowering medication (fibrates preferred) [8].

Viral hepatitis, peptic ulcer disease, pancreatic pseudocysts, cholelithiasis, and cholecystitis are some of the conditions with the presentation similar to pancreatitis. The biochemical analysis is an important adjunct to diagnosing pancreatitis. At the onset, it helps to confirm the diagnosis as well as find the etiology. The diagnostic criteria for AP include raised serum amylase and/or lipase levels associated with characteristic imaging findings. Liver function test with enzyme levels can help rule out biliary tract disorders and hepatitis.

Assessing the appearance of serum before processing is good laboratory practice and mentioning it in the report is important because this cannot be detected during blood collection (serum has not separated) and the clinician may not be aware of such serum morphology. Apart from lipemic, the sample may also be hemolyzed [9] or viscous. In all these cases, the automated analyzer is bound to give error readings, especially for liver function tests, lipid profile, and serum analysis for amylase and lipase. All these analyses are performed by spectrophotometric method and lipoproteins in lipemic sample cause physical interference. Moreover, they may lead to reduced uniformity of sample and hence, erratically low values [10]. If it is absolutely essential to process the sample, like for amylase levels in our case, the sample can be diluted sufficiently and readings reported. However, a note about dilution and possibility of false low values due to TG interference must be added in the report. In general, for amylase levels, the interference limit for TG is 2000 mg/dL.

However, the diagnosis requires raised serum amylase and/or lipase levels (>3 times the upper limit of normal) along with characteristic imaging findings of AP.

**CONCLUSION**

Lipemic sample while investigating abdominal pain in either pregnant women or patients with diabetes mellitus, hormone disorders, or chronic alcoholism, must prompt the diagnosis of HTG-AP. The laboratory should be aware of sample artifacts and efforts should be taken to convey the findings (both physical and biochemical) to the clinician. This case also stresses the need for good communication between treating clinical and diagnostic faculties.

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