Hypertriglyceridaemic pancreatitis with eruptive xanthomas

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DESCRIPTION

A 26-year-old man presented with severe upper abdominal pain radiating to the back for the past 2 days. He also had multiple episodes of non-bilious vomiting. He was a chronic smoker and an alcoholic for the past 8 years. He had no history of diabetes mellitus or any drug abuse. On examination, the patient was hyperventilating and in severe respiratory distress. There were multiple yellowish papular lesions over the extensor surface of bilateral forearms, arms (figure 1A) and dorsum of hands (figure 1B). He was febrile and blood pressure was 92/68 mm Hg and pulse rate was 112/min. When the blood was drawn for initial lab investigations, white cloudy opaque (lactescent) serum was observed (figure 1C). Total leucocyte count ($15 \times 10^9/L$), Serum amylase levels (546 mg/dL) and lipase levels (346 mg/dL) were elevated. Lipid profile of the patient showed elevated triglycerides (TG, 2928 mg/dL), total cholesterol (601 mg/dL), high-density lipo protein (HDL) cholesterol (105 mg/dL). He was initially treated with aggressive intravenous fluid resuscitation and was haemodynamically stabilised. Intravenous analgesics were administered and he was kept on nil per oral and was started on intravenous fluid. A contrast-enhanced CT of the abdomen showed bulky pancreas with multiple intrapancreatic and peripancreatic collections with no evidence of necrosis, abscess or pseudocyst formation (figure 2), which confirmed the diagnosis of hypertriglyceridaemic pancreatitis (HTGP) with eruptive xanthomas. Patient was started on intravenous heparin and insulin for reducing the lipid level. He was started on orals the next day and was escalated to a normal diet and was discharged in a stable condition. Dietary modification was advised and he had no further episodes even after 1-year follow-up.

Figure 1 (A) Yellowish papular lesions over bilateral forearms; (B) Yellowish papular lesions over the dorsum of both the hands; (C) A plain sample collection tube containing lactescent serum.

Figure 2 Transverse section of contrast-enhanced CT of abdomen showing bulky pancreas with multiple peripancreatic and intrapancreatic collections. (A - Anterior, P -Posterior)

Severe hypertriglyceridaemia (HTG) (levels greater than 2000 mg/dL) accounts for up to 10% of all the acute pancreatitis cases.¹ High levels of TG are hydrolyzed by pancreatic lipases into glycerol and free fatty acids. These free fatty acids were thought to cause cytotoxic reaction, also damage to the vascular endothelium culminating in pancreatic inflammation and ischaemic injury.² Although the clinical presentation is similar to pancreatitis caused by other causes, clinical signs

Patient's perspective

I had an episode of severe abdominal pain after I had an alcohol binge. It was a burning type of pain and I was unable to stand still due to its severity. I took an antacid but I felt nauseous and vomited thrice. I never had that severe pain ever before. We immediately went to a hospital where the doctors examined me and told me that my pancreas might have been the cause for my pain. They gave me some pain medication and inserted a tube into me through my nose. I was shocked and surprised when the doctor had told me that I had milky serum and might've been due to elevated lipids. They took a CT scan and told me that the problem was indeed with my pancreas. Doctors strictly asked me not to take anything by mouth and I complied. Pain was better the following day and I was started on water. I was instructed not to take alcohol, exercise regularly and to have a healthy lifestyle. I judiciously followed the doctor's instructions and it's been an year since the episode. I am quite healthy now.

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such as eruptive xanthomas that occur on the extensor surfaces of extremities should raise a clinical suspicion of HTGP. These lesions occur in 10% of the cases with HTG and usually point to a familial aetiology which is absent in our case.³ The serum levels of pancreatic enzymes can be falsely low when TG levels are >500mg/mL which is likely due to the interference of TG with the assay.⁴ The diagnosis of HTGP may be challenging as sometimes the TG levels may be normal due the nil oral intake by the patient because of severe pain abdomen, and in the absence of a clear aetiology patients should have a new test of TG levels after the acute attack has resolved and the patients has restored oral intake.⁵ Management is similar to acute pancreatitis due to other causes. The optimal treatment of HTG-associated pancreatitis is not clear at present. In many cases, the TG levels will likely normalise following nil per mouth and fluid therapy

Learning points

- ▶ Serum triglycerides (TG) level of ≥ 500 mg/dL in patients with acute pancreatitis (AP) should raise a high degree of suspicion of hypertriglyceridaemic pancreatitis (HTGP), especially when no other aetiology of AP seems apparent.
- ▶ Presentation of HTGP is similar to AP due to other aetiologies but due to high serum levels of circulating TG, the serum amylase levels may not be markedly elevated.
- ▶ Clinical course of patients with HTGP depends on the control of TG levels and so an early diagnosis, followed by quick initiation of lipid-lowering treatment is of paramount importance in the management of HTGP.
- ▶ Dietary and lifestyle modification, abstinence from alcohol along with lipid-lowering agents (statins+fibrates) are required in these patients for the control of TG levels and to prevent recurrences.

without need of insulin/heparin or plasmapheresis. But if it is not normalised then additionally, intravenous heparin and insulin has to given as in the present case. Alternatively, plasmapheresis can be considered but its efficacy over medical management is not fully proven.¹ TG levels of <500mg/dL are desirable to prevent recurrences which can be achieved by dietary modifications and antihyperlipidaemic drugs.⁴

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