

Case Report

Hyperlipidaemia as a Cause of Acute Pancreatitis: Report of Two Cases and Review of the Literature

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ABSTRACT

Hyperlipidaemia is known to cause acute pancreatitis. Recently, we have encountered two such cases in one

month. In this report, we present the two cases with review of relevant literature.

KEY WORDS: acute pancreatitis, hyperlipidaemia

CASE 1

A 33-year-old man was admitted with acute epigastric pain radiating to the back. It was associated with nausea, vomiting and anorexia. The patient was a known diabetic on oral hypoglycemic agents. He had no other relevant medical history.

On examination, he was febrile but haemodynamically stable. Systemic examination revealed no abnormality. The abdomen was lax but there was localized tenderness and guarding in the epigastric region. The only significant finding was the presence of eruptive xanthomas over the extensor aspect of the forearm and knee joints.

The laboratory could not perform the routine investigations as the blood sample was viscous and highly lipaemic. Only serum amylase (587 i.u.) and urinary amylase (2602 i.u.) results were obtained.

A few hours later, the patient became tachypnoeic. Blood gas analysis (BGA) revealed severe respiratory and metabolic acidosis. At this point, the patient was transferred to the ICU for further management. A central venous line was secured and supportive treatment was started with sodium bicarbonate and intravenous (IV) fluids. He continued to breathe spontaneously via a ventimask and maintained a SPO₂ of 98-99%. However, he had a HR of 140 bpm, a BP of 107/75 mmHg and a RR of 50 bpm. CVP measured was 6 cm of water.

Despite supportive treatment, the patient deteriorated. It was decided to intubate and ventilate him. BGA after intubation revealed a pH of 7.34, PCO₂ of 11 mmHg and PO₂ of 96.7 mmHg with a base excess of minus 15.8 mmol/L. The patient was maintained on sedation, analgesia and relaxant in appropriate doses.

One of the greatest difficulties in the management of this case was to maintain IV access. All lines were getting repeatedly blocked due to high viscosity of blood. Heparin was started after consultation with the hematologist.

Despite therapy with appropriate doses of insulin and normal saline, the urine continued to show glycosuria and ketonuria. Urine output was 1300 ml in six hours. The patient continued to have tachycardia (170 bpm) inspite of all aggressive measures.

Fifteen hours after admission to the ICU, he suddenly developed bradycardia and cardiac arrest. He did not respond to CPR and died.

CASE 2

A 42-year-old lady was admitted to the medical department with history of shortness of breath and vomiting since morning. She was a known case of non-insulin dependent diabetes mellitus (NIDDM) on oral hypoglycemic agents. She was not hypertensive.

On examination, she was conscious, coherent. Abdominal examination revealed guarding and tenderness in the upper abdomen. Bowel sounds were sluggish. Other systems were normal. She had a HR of 130 bpm, BP of 160/80 mmHg, RR of 50 bpm and CVP of 14 cm of H₂O.

The blood sample was lipaemic but some laboratory results could be obtained. Her blood sugar level was 29.7 mmol/L, albumin 25.5 g/L, calcium 1.02 mmol/L, total bilirubin 40.37 µmol/L, direct bilirubin 21.6 µmol/L, sodium 121 mmol/L, potassium 3.9 mmol/L, bicarbonate 13.4 mmol/L and LDH 282 i.u. Urine was positive for glucose

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and ketones. BGA revealed a pH of 7.28, PCO₂ of 28 mmHg, PO₂ of 146 mmHg and a base excess of minus 11 mmols.

An ultrasound examination of the abdomen showed markedly dilated bowel loops and a bulky pancreas. A working diagnosis of severe acute pancreatitis with diabetic ketoacidosis with hyperlipidemia and hypocalcaemia was made. Treatment consisted of nasogastric aspiration, antibiotics, H₂ receptor blockers, insulin, heparin, potassium chloride, analgesics (pethidine) and albumin.

The patient showed clinical improvement on the second day. But CT abdomen showed increased oedema of the head of the pancreas. She became tachypnoeic in the evening but still maintained acceptable oxygenation. The lipid profile however showed a marked increase in the cholesterol and triglyceride levels.

On the third day, IV lines began to get blocked due to increased viscosity of blood. She became febrile, tachycardic and tachypnoeic. Chest auscultation revealed right-sided crepitations. BGA revealed compensated metabolic acidosis.

Since the patient was not showing signs of improvement, we decided to ventilate her for 48 hours. Propofol was avoided as a sedative in view of the existing hyperlipidemia. On day four, the patient was stable but febrile. BGA was satisfactory. Abdominal girth was increasing and bowel sounds were absent. CT abdomen with contrast showed a bulky head of pancreas with minimal pleural effusion.

On day five, she was weaned from respiratory support and extubated. She was observed in the ICU for the next two days. She improved clinically, maintained stable vital signs with a progressively decreasing cholesterol and triglyceride profile.

She was then moved to the ward and discharged from the hospital four days later.

DISCUSSION

We present here, two cases of severe acute pancreatitis probably caused by hyperlipidemia. Both were aggressively managed in the ICU. However, the first patient succumbed to the disease due to coronary ischaemia shortly after admission but the second patient responded to therapy and recovered. Repeated blockage of IV access due to high viscosity of blood and inability to get laboratory result because of lipaemic samples were common problems encountered. Both patients showed metabolic acidosis, hyperglycemia, and elevated levels of triglycerides and cholesterol.

Acute pancreatitis is a known complication of hyperlipidemia. Type I, IV and V of hyperlipidemia is likely to cause pancreatic

inflammation, if the triglyceride level increases to more than 11.3 mmol/dl^[1].

Hyperlipidemia is defined as elevated concentrations of triglycerides (> 2.2 mmol/L) or cholesterol (> 5.2 mmol/L) in fasting plasma. The latter are transported in the plasma by lipoproteins. There is accumulating evidence linking elevated total cholesterol, elevated low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C) with the development of coronary artery disease. Elevated TG may cause pancreatitis.

Familial type V hyperlipoproteinaemia characterized by elevated TG, cholesterol, chylomicrons and very low density lipoproteins (VLDL) levels and decreased HDL levels is the most common genetic disorder associated with pancreatitis in adults. Patients with this disorder can have severe recurrent pancreatitis that can progress to pancreatic insufficiency or failure.

Secondary triglyceridemia resulting from treatment with estrogen or isotretinoin has been associated with pancreatitis. Estrogen induced pancreatitis usually occurs during the initial months of therapy. Isotretinoin-induced pancreatitis results from dose-related hypertriglyceridemia within the first month of therapy.

Estrogen replacement therapy has been reported to induce pancreatitis in women with familial type V hyperlipoproteinemia. It has been suggested that pancreatitis in these cases is secondary to the combined effects of estrogens on triglyceride cleaning enzymes, carbohydrate metabolism and increased TG production. It has been recommended that estrogen therapy be relatively contra-indicated in women with TG levels greater than 3.39 mmol/dl and absolutely contra-indicated in women with TG levels greater than 8.8 mmol/dl, to prevent TG-induced pancreatitis^[1].

As early as 1972, clinicians were alerted to cases of marked hyperlipidemia and pancreatitis associated with the use of birth control pills, although no genetic testing was available at that time^[2]. Apoprotein E (apo E), particularly apo E allele 2, has been found in association with higher triglyceride levels in pregnant patients with chylomicronemia^[3]. Although it is known that pregnancy results in an increase in plasma triglyceride levels, how this increase occurs is not totally understood.

Approximately 15 cases of pancreatitis associated with pregnancy and hyperlipidemia have been described since 1956^[4]. The specific genetic defect in lipid metabolism has yet to be fully explained, although it is phenotypically associated with familial hyperlipidemias I, III, and V^[5,6,7]. A derangement in lipoprotein lipase (LPL) has been found in association with pregnancy-induced

hypertriglyceridemia and pancreatitis for a patient homozygous for a missense ser172→cys mutation^{8]}.

Although it is known that pregnancy results in an increase in plasma triglyceride levels^{9]}, how this increase occurs is not totally understood. In addition, certain persons express profound elevations in triglyceride values during pregnancy^{14,6]}. No physiologic explanation for this unique population of pregnant patients with hypertriglyceridemia has been found. It has been found that the profound increase in triglyceride level in the presence of a lipolytic defect does not resolve whether triglyceride synthesis or LPL-induced catabolism is primarily affected. However, given the role of LPL in the triglyceride rich particle catabolism, the finding in some cases indicates a primary cause of a defect in LPL.

In conclusion, these two cases of acute pancreatitis caused by hyperlipidemia illustrate the special problems associated with management, particularly the repeated blockage of IV access and difficulty in obtaining reliable laboratory results. These cases are critical and may have a high mortality rate as demonstrated by our first case. They need close monitoring and aggressive therapy in the ICU in order for them to survive the episode of severe acute pancreatitis.

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