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Case report

Acute pancreatitis secondary to severe hypertriglyceridemia: Clinical case

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Abstract

Introduction: Acute pancreatitis is considered an inflammatory process of the pancreas, which results from the activation of digestive enzymes released by this gland. This pathological entity is associated with multiple etiologies. **Case:** 37-year-old male with hypogastrium pain irradiated to the back. On physical examination, the patient was tachycardic, hypertensive, with abdominal pain on deep palpation without signs of peritoneal irritation. Laboratory tests showed an elevation of acute phase reactants and pancreatic amylase, and imaging studies showed signs of acute pancreatitis. **Discussion:** Pancreatitis associated with hypertriglyceridemia occurs in 0.5 to 1% of cases. The initial management triad is a clear liquid diet, intravenous hydration, and analgesics. Hypertriglyceridemia can be managed with oral hypolipidemic drugs. When levels are higher than 1 000 mg/dL, plasma exchange can be used due to its good effectiveness, decreasing the values to normal ranges in 80% of the cases with the first session.

Keywords

Pancreatitis, Dyslipidemia, Hypertriglyceridemia, Triglycerides.

INTRODUCTION

Acute pancreatitis is an inflammatory process that occurs in the pancreas as a result of the activation of digestive enzymes released by this gland, primarily trypsinogen, which causes self-digestion of acinar cells (1), local lesions, systemic inflammatory response syndrome (SIRS) and, in the worst-case scenario, multiple organ failure (2).

This is one of the most common gastrointestinal disorders requiring hospitalization, with an annual incidence of 5 to 30 cases per 100 000 inhabitants (3) and an overall mortality rate of 5 % to 7 % that can reach up to 30 % in cases of severe pancreatitis (4).

The main causes of acute pancreatitis include biliary disorders (5), alcoholism (6), genetic alterations (7), smoking, and use of drugs such as azathioprine, estrogens, furosemide/thiazides, sulfonamides, isotretinoin, tetracycline, valproic acid, angiotensin converting enzyme inhibitors (ACE inhibitors), antidepressants, steroids, β-blockers, among others (8). Severe hypertriglyceridemia occurs in 2 % to 5 % of cases and is defined as triglyceride concentrations in blood ≥1000 mg/dL (9), although its pathophysiological mechanism is still under study; however, there are several hypotheses such as the prolonged release of intracellular calcium, which facilitates cell apoptosis, inhibition of mitochondrial function in the pancreatic acinus, toxicity due to

free radical release and formation, and facilitation of the inflammatory response. Furthermore, it has been described that excess fatty acids can obstruct arterial circulation, making pancreatic ischemia more likely (10).

The aim of this paper is to present the case of a young patient who developed acute pancreatitis as a result of severe hypertriglyceridemia, as well as the clinical management provided and his clinical response to treatment.

CLINICAL CASE

37-year-old male with a 4-day history of severe hypogastric pain radiating to the lumbar area, hyporexia, unquantified fever with presence of diaphoresis, and multiple emetic episodes that started after consuming a high fat meal. The patient had no relevant medical history.

On physical examination the following information on his vital signs was reported: heart rate (HR): 115 beats per minute (bpm), blood pressure (BP): 142/83 mm Hg, respiration rate (RR): 22 breaths per minute (rpm), oxygen saturation (SaO_2) 81 % at room air, temperature: 40°, height: 168 cm, weight: 78 kg, body mass index (BMI) 27.6 (overweight), Glasgow score: 15/15, distended abdomen, soft on palpation, and no signs of peritoneal irritation; the rest of the examination was normal.

In view of a suspected pancreatic disease (pancreatitis with possible overinfection), further tests were requested. Lab test results on admission showed pancreatic amylase at 1229 mg/dL, leukocytosis (21 000/mm^3), neutrophilia (92%) and hypertriglyceridemia (3980 mg/dL), elevated very low-density lipoprotein (VLDL) (80 mg/dL), with total cholesterol (120 mg/dL), and high-density lipoprotein (HDL), electrolytes, kidney function, liver function and procalcitonin within normal values, thus the probability of an infection. Additionally, an abdominal computed tomography (CT) scan was requested, which revealed bilateral pleural effusion, signs of interstitial edematous pancreatitis associated with multiple acute peripancreatic fluid collections and free abdominal fluid, leading to the diagnosis of Balthazar C-D pancreatitis (**Figure 1**), with an APACHE clinical severity score of 8 points and a Marshall score of 2 points.

Initial management was given in the emergency room with Ringer's lactate solution 1000 mL IV bolus, meperidine 30 mg every 8 hours, ranitidine 50 mg every 12 hours, metoclopramide 10 mg every 12 hours, atorvastatin 20 mg every 12 hours, and complete restriction of oral intake.

The patient was assessed by the general surgery department, which determined he did not have pancreatic collection overinfection and was not a candidate for surgery. He was then evaluated by the internal medicine service and his treatment was modified to include dipyrone 1.5 g every 8 hours (instead of meperidine), ciprofibrate 100 mg every day (instead of atorvastatin), and a soft hypofat and hyperprotein diet.

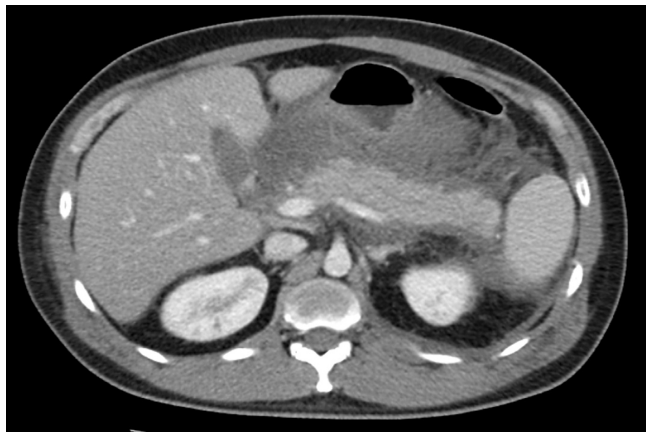

Figura 1. CT scan of the abdomen. Edematous interstitial pancreatitis associated with multiple apical peripancreatic collections.

Finally, the patient was transferred to the intensive care unit (ICU) for plasmapheresis, which is considered the first line of treatment for hypertriglyceridemia, resulting in a satisfactory response, as evidenced by the substantial decrease in triglyceride values (410 mg/dL) after the first session. The patient stayed in the intensive care unit for 48 hours. Then, on the fourth day of hospital stay, and since the patient's clinical condition progressed satisfactorily and his lab test results were normal, he was discharged with medical instructions for therapeutic management, outpatient follow-up, and recommendations.

ETHICAL CONSIDERATIONS

Informed consent was obtained in accordance with the provisions of Resolution 8430 of 1993, and only sociodemographic, clinical, and laboratory tests data relevant to the study were used.

DISCUSSION

Severe hypertriglyceridemia is the third most common cause of acute pancreatitis in young patients (9) with ages similar to that of the patient described in this case report. This pathophysiological association has been reported to be low (11). Pancreatitis secondary to severe hypertriglyceridemia has been identified in 0.5 % to 1 % of cases (12), with other incidences ranging from 1% to 7% (17). Similarly, since serum triglyceride levels of 500 mg/dL are associated with a high risk of acute pancreatitis (9), details of these events, effective clinical treatment, and the results of outpatient studies are needed.

In this way, hypertriglyceridemia is classified depending on factors such as cause, pattern of lipid elevation, and severity, among others. Depending on the etiology, hypertriglyceridemia can be primary, including cases associated with genetic disorders related to triglyceride synthesis and metabolism (8), or secondary, resulting from inadequate control of endocrine disorders (diabetes, obesity, metabolic syndrome, hypothyroidism), intake of medications, and excessive alcohol consumption (13). The patient described here developed acute pancreatitis caused by hypertriglyceridemia, overweight and most likely low alcohol intake.

There are several hypotheses about the pathophysiological mechanisms associated with the elevation of triglycerides as a cause of pancreatitis. The most compelling theory is that excess plasma triglycerides, together with the action of pancreatic lipase, cause an accumulation of fatty acids in the pancreas, which generates a large amount of free radicals and, as a result, irrigation is altered by blood hyperviscosity, which, in turn, affects pancreatic acini (10).

In-hospital management should include close monitoring, pain control, the administration of antiemetics, and common therapeutic measures such as restricted, low or no fat diet to stabilize the patient's metabolism and, if necessary, the use of nasojejunal tubes (14, 15). An effective pillar of treatment is fluid replacement with Ringer's lactate, which reduces the risk of systemic inflammatory response syndrome (SIRS) by 84% ($p = 0.035$) compared to normal saline solution. Proper intravenous fluid control is accomplished by ensuring adequate urinary output, and electrolyte and glucose stability; however, other measures should be taken to prevent hypoxemia using supplemental oxygen in case of patient stabilization (16, 17).

There are a variety of treatment options for hypertriglyceridemia. The first is fasting, which lowers triglyceride levels during the initial management period; the second is the administration of oral lipid-lowering agents, which keep triglyceride levels within acceptable limits, and finally, drastic steps such as plasmapheresis, which separates plasma from harmful elements, in this case, excess triglycerides (17). Plasmapheresis has the advantage of lowering triglyceride values to normal levels after a single session in 50 % to 80 % of cases, which is why its use is recommended when triglycerides concentrations are above 1000 mg/dL and are associated with complications such as that developed by our patient (acute pancreatitis) (18-20). Some studies have shown that mortality rates decrease by 24% when plasmapheresis is used, but these results are not statistically significant (21, 22). Another therapeutic option that can be used is the infusion of insulin or heparin, which favors the degradation of chylomicrons and reduces the activity of plasma lipases (23, 24).

To date, the percentage of pancreatitis recurrence due to hypertriglyceridemia is unknown, but carrying out regular check-ups, prescribing fibric acid derivatives, treating the secondary cause and controlling the risk factors associated with the metabolic disorder is fundamental (24-26).

In conclusion, pancreatitis associated with hypertriglyceridemia has a low prevalence and its clinical manifestations and diagnostic methods are similar to those related to other etiologies; however, additional laboratory tests are required to detect the type of alterations associated with lipid metabolism. Treatment is divided into three stages. In the first phase, it is treated similarly to other types of pancreatitis; the second phase is concerned with the acute control of elevated serum triglyceride values, for which plasmapheresis is used; and the third phase involves strict diet control and the long-term use of oral lipid-lowering agents.

Conflict of interest

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