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A rare complication of necrotizing pancreatitis due to type 4 hyperlipidemia ıleus mimicking colon tumor

Sami Benli, Habip Sarı and Deniz Tikici

Abstract
Background: Acute pancreatitis develops as a result of the premature activation of pancreatic enzymes and manifests as abdominal pain and elevated pancreatic enzymes. The causes of acute pancreatitis are most frequently gallstones and alcohol, but another cause can be hypertriglyceridemia. Hypertriglyceridemia is responsible for approximately 1–4% of acute pancreatitis cases; serum triglyceride levels over 1000 mg/dL may trigger an attack of acute pancreatitis. Acute pancreatitis may develop when triglyceride levels reach 500–1000 mg/dL.
Case presentation: Colonic ileus was detected as a result of advanced radiological examinations in a 61-year-old male patient who presented to the emergency department with complaints of abdominal pain, abdominal swelling, gas, stool, and colonic tumor was considered as a preliminary diagnosis. There are familial episodes of pancreatitis due to type 4 hyperlipidemia in the patient's past medical history. As a result of expansion, it was observed that inflammation due to pacreatic necrosis constricted the transverse distal of the colon. Subtotal colectomy, end ileostomy and pancretatic debridement were performed on the patient. The patient was discharged on the 8th postoperative day with approximately 40 cc pancreatic fistula.
Conclusion: AP is a life-threatening inflammatory disorder of the pancreas which can be reversed. Although HTGP is the third most common form of pancreatitis after alcohol and gall stones, overall this disorder is quite rare. This results in the available information being sparse and the limited number of patients in the studies not allowing for generalization. Due to the absence of specific treatment guidelines for HTG AP, the HTGP patients are treated similarly to AP patients of other etiologies.

Keywords: hypertriglyceridemia, necrotizing pancreatitis, type 4 hyperlipidemia

Introduction
Background
Hyperthyglyceridemia (HTG) is the third most common cause of acute pancreatitis after gallstone disease and alcohol [1, 2]. It constitutes approximately 7% of the total acute pancreatitis cases [3]. The etiology of HTG is generally divided into 2 categories, primary and secondary. While primary one causes more severe HTG, it is the interplay of both primary and secondary factors that leads to severe HTG. HTG, usually in familial chylomicronemia syndrome, primary hypertriglyceridemia and mixed hypertriglyceridemia are also seen. These are Fredrickson Type I, IV and V respectively. Common genetic defects that lead to severe HTG include lipoprotein lipase deficiency, LPL gene mutation, and Apolipoprotein C II deficiency.
In pancreatitis (HTGP) caused by hyperthyglyceridemia, TGs are hydrolyzed by pancreatic lipase, resulting in free fatty acids that cause damage. The prevalence of developing acute pancreatitis among dyslipidemic patients is 5% if the TG level is greater than 1000, and 20% if it is greater than 2000. National cholesterol Education Program ATP III categorizes triglyceride (TGs) level as normal (<150), borderline high (150-199), high (200-499), and very high (>500 mg/dL) (1 mmol= 88.5736 mg/dL)Thereare various epidemiological studies that tried to determine the appropriate cut-off for TG level to cause AP. In a study of 129 patients with severe HTG, 26/129 (20.2%) patients experienced at least one episode of AP [4]. Current recommendation that TG < 1000 mg/dL is unlikely to cause acute pancreatitis.
Although HTGP manifests clinically like other pancreatitis, complication, morbidity and mortality rates are higher in HTGP [5]. Deng *et al.* reported severe HTG-AP (TGs > 500 mg/dL) was associatedwith higher 24 hr APACHE II score, increased incidence of renal failure, shock, infection, and overall mortality (31.1 versus 9.1, P < 0.01)when comparedwith severeAP from other causes [6].
Diabetes is the most common secondary factor physicians would encounter in patients with HTG pancreatitis. TG levels are higher in patients with poorly controlled diabetes and in the

setting of diabetic ketoacidosis, thereby increasing the risk of pancreatitis. The prevalence of diabetes in HTG pancreatitis is much higher than the general population and patients with HTG but no pancreatitis.

We present a case in which we performed subtotal colectomy end ileostomy as a result of complete splenic flexure obstruction due to pancreatic inflammation, as a result of the investigations of a 61-year-old male patient admitted to the emergency department with the complaints of abdominal pain, inability to expel gas-stool and abdominal swelling.

Case presentation

A 61-year-old male patient is admitted to the emergency room with abdominal pain, swelling in the abdomen, inability to pass gas and stool and nausea and vomiting. At admission, the patient has sinus tachycardia (122 / min), tachypnea (22 / min). Controlled diabetes and hypertension were present as comorbidities. On physical examination, the abdomen was extremely distended, there was no rebound, but there was tenderness. The patient had a history of previous cholecystectomy, ercp history and hypertriglyceridemia in his past medical history. The abdominal CT showed also has a dilated appearance compatible with the colonic loops with ileus. The dilatation of the colonic loops reached up to the splenic flexure, and the loops distal to the splenic flexure were in collapsed appearance. In the patient who did not have a transition from splenic flexure to distal, tumoral formation was considered in the first stage, but in detailed examination, it was seen that the cause of ileus was secondary to pancratic inflammation and the patient was taken into an emergency operation with the diagnosis of ileus.

At the time of operation, secondary to inflammation, the patient was observed to have a narrowed necrotizing pancreas, including the splenic flexure. The distal of the splenic flexure was collapsed and the proximal part of it was dilated. There was no mass formation to suggest a tumor. Serosal openings were seen in the transverse colon and cecum. Subtotal colectomy and end ileostomy were performed on the patient. Pancreatic abscess was drained. Pancreatic necrosectomy was performed. The patient was taken to the service from the intensive care unit on the second postoperative day. He was discharged on the 8th postoperative day with a daily 40 cc pancreatic fistula.

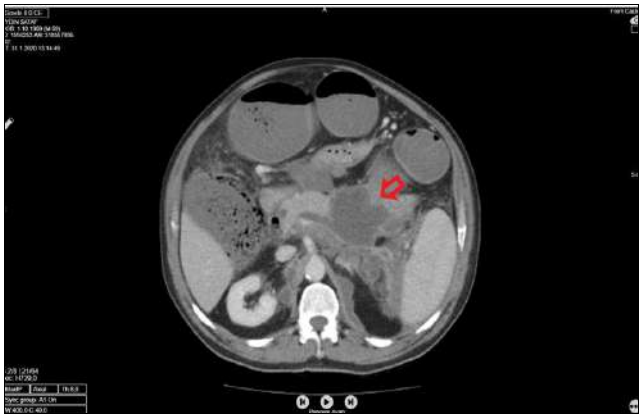

Fig 1: Collection due to pancreatic abscess causing narrowing of splenic flexure

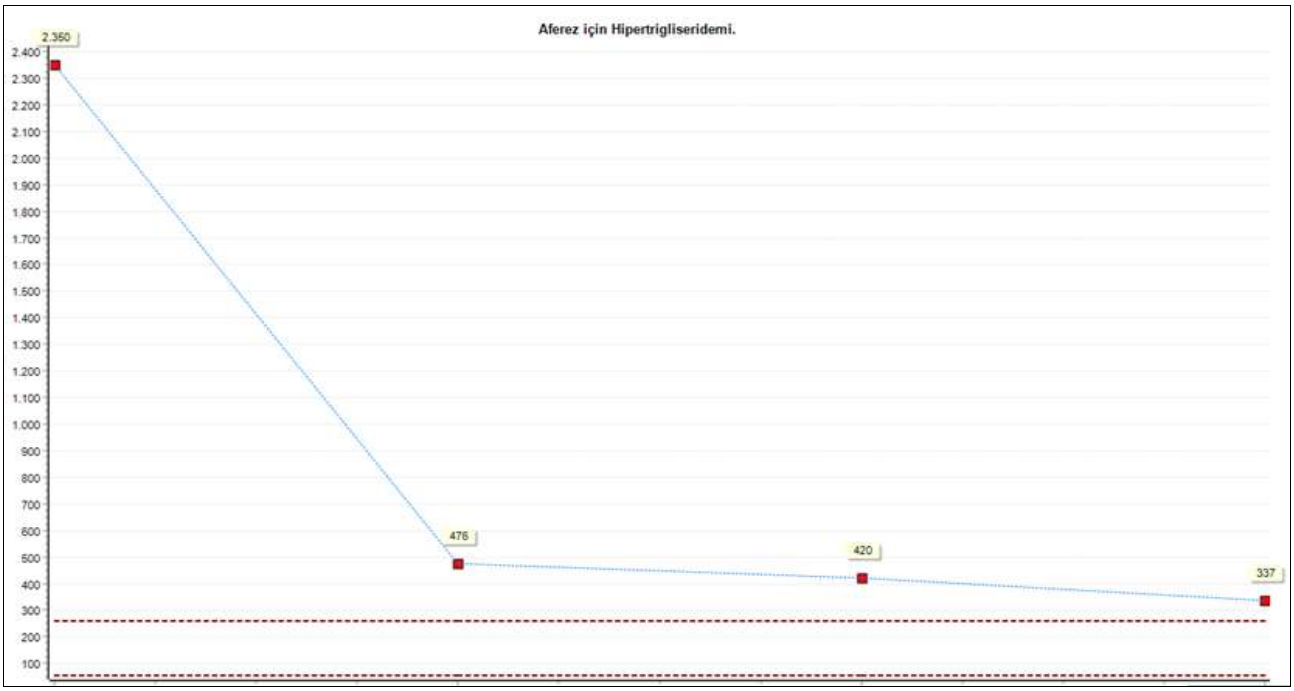

Fig 2: The patient's triglyceride levels

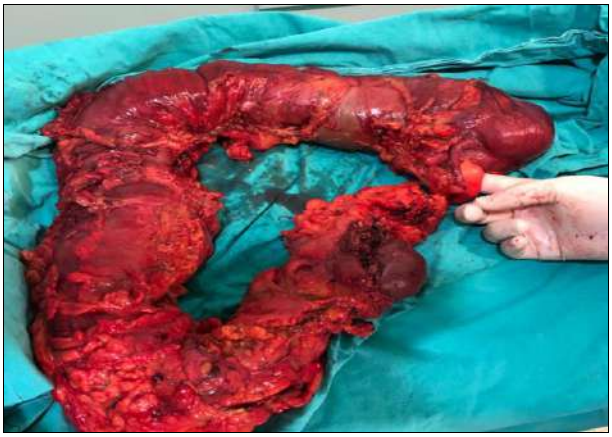

Image 1: Subtotal colectomy material

Discussion

Hyperlipidemia is an important problem that should be kept in mind in the differential diagnosis of acute non-biliary pancreatitis. Another detail that should be known is that, despite severe acute pancreatitis, measurement of serum pancreatic enzymes in hyperlipemic plasma may give false low results and therefore enzyme levels may be found normal during pancreatitis in these cases.

Although there is no standardized treatment strategy in acute pancreatitis cases developing on the basis of hypertriglyceridemia, protocols consisting of insulin and heparin combination based on increasing LPL activity have been used successfully [7].

Alipogene tiparvovec is a promising gene therapy in the treatment of severe pancreatitis due to LPL deficiency [8]. Plasmapheresis was used as a lipid-lowering method for acute pancreatitis caused by hypertriglyceridemia and treatment regimen AG133, and complete recovery was observed in three quarters of the patients [9].

A multidisciplinary approach is required to prevent recurrences in patients with HTG pancreatitis. Lifestyle changes aimed at losing weight, limiting fat intake and avoiding simple carbohydrates and alcohol, and controlling secondary risk factors such as diabetes are an integral part of patient management. Patients usually need lipid lowering agents during their follow-up. In addition, they should be closely monitored by the endocrinologist and dietician in blood sugar regulation. Although near-normal TG levels may be preferred, levels <500 mg / dl represent a safe therapeutic target for the prevention of relapses.

Conclusion

HTG is more likely to be associated with severe pancreatitis as compared to other causes but no mortality difference has been reported. Recent data points to severe course of disease with higher TGs level on admission. Currently, there are no standardized treatment guidelines.

It should be kept in mind that hyperlipidemia is one of the causes of acute pancreatitis. In the treatment, routine acute pancreatitis treatment should be done in the first stage, and lipid-lowering treatments should be started in the ongoing process. Patients should be followed up by a multidisciplinary team.

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