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Hypertrygliceridemia-induced Acute Pancreatitis Following Hyperlipidemic Abdominal Crisis

Hipertrigliseridemi ile İnduklenen Akut Pankreatit Sonrası Hiperlipidemik Abdominal Kriz

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Abstract

Hypertriglyceridemia is a well established cause of acute pancreatitis (AP). Multiple mechanism are proposed to explain this phenomenon, but the exact mechanism is unknown. Clinical manifestations are similar to other forms of AP. Although amylase and lipase levels exclude the diagnosis of AP in normolipidemic patients, they may be normal in hypertriglyceridemia-induced AP. Further evaluation with imaging studies are needed for diagnosis. A less known entity "hyperlipidemic abdominal crisis" is a prior clinical state before development to AP. We describe a young male patient without any previously diagnosed metabolic disorder presenting to emergency department with abdominal pain and vomiting where normal amylase in lactescent serum was detected. His abdomen computed tomography (CT) was reported as normal. His symptoms were relieved with antiemetic and histamine-2 blocker and he was diagnosed with hypertriglyceridemia and dyspepsia. Readmission with recurrence of symptoms after 3 hours resulted in hospitalization where the second CT showed edematous AP.

Key Words: Computed tomography, Hypertriglyceridemia, Pancreatitis

Özet

Hipertrigliserideminin akut pankreatite (AP) neden olduğu bilinmektedir. Bu duruma neden olabilecek bazı mekanizmalar açıklanmaya çalışılsa da tam nedeni tam olarak bilinmemektedir. Klinik tablo, AP'in diğer formlarına benzemektedir. Nonlipidemik hastalarda normal sınırlardaki amilaz ve lipaz değerleri AP tanısını dışlamaya yardımcı olsa da hipertrigliseridemi ilişkili AP hastalarında normal sınırlarda olabilirler. Görüntüleme yöntemlerini içeren ileri incelemeler tanı için gerekli olabilir. Daha az tanımlanmış olan "Hiperlipidemik Abdominal Kriz", AP gelişimi öncesi klinik durumdur. Olgu sunumumuzda önceden herhangi bir metabolik bozukluğu olmayan genç erkek bir hastanın üt görünüşlü serumunda normal amilaz seviyesine sahip olup acil servise karın ağrısı ve kusma nedeniyle geldiğini anlattık. Batın bilgisayarlı tomografisi (BT) normal olarak raporlanmıştı. Semptomları antiemetik ve histamin-2 bloker tedavisi ile gerilemiş ve hipertrigliseridemi ve dispepsi tanılarıyla taburcu oldu. 3 saat sonra semptomlarının tekrarlaması sonucu hospitalize edilen hastanın çekilen 2. BT'sinde ödematöz pankreatit saptandı.

Anahtar Kelimeler: Bilgisayarlı tomografi, Hipertrigliseridemi, Pankreatit

Introduction

Since Speck identified the relationship between hyperlipidemia and acute pancreatitis (AP) in 1865, no specific test has been available to establish the diagnosis, predict the severity and identify the etiology [1]. In the diagnosis of AP, serum amylase and lipase levels remain important tests; however, simultaneous estimations of both do not improve accuracy [2]. Although normal serum amylase levels exclude the diagnosis in normolipidemic AP, this is not the case in acute exacerbations of chronic pancreatitis or hypertriglyceridemia-induced acute pancreatitis (HIAP). The diagnosis of AP is mainly confirmed with imaging studies.

We present a young male patient presenting to the emergency department (ED) with abdominal pain, misdiagnosed as dyspepsia and treated with a histamine 2 blocker, with relief of symptoms. AP was excluded with normal contrast-enhanced computed tomography of the abdomen and a general surgery consultation before he was discharged from the ED. He was readmitted with recurrence of symptoms and was diagnosed with HIAP.

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Case Report

A 28-year-old male presented to the training and research hospital ED with a history of 2 hours of constant epigastric pain radiating to the right upper quadrant and flanks. It was described as a dull pain accompanied by nausea and vomiting. He did not have any relevant past medical history. He denied any history of alcohol abuse or a family history of metabolic disorders. He did not smoke and denied any medication usage.

His vital signs were within the normal range on admission. His physical examination revealed epigastric pain aggravated by deep palpation of the epigastric region without any acute abdominal signs, while the remaining examination was normal.

He was first treated with the histamin-2 blocker famotidine and the antiemetic metoclopramide while waiting for laboratory results. His serum was noted to be lactescent. Liver enzyme levels were not measured due to high lipidemia, and the amylase level was 47 U/l. His leucocyte count was 11,000/ml. His blood work was repeated with additional cholesterol levels, and his symptoms persisted. The triglyceride level was 1689 mg/dl (normal range 40-150 mg/dl), and the high-density lipoprotein level was 9 mg/dl (normal range 35-60 mg/dl); the low-density lipoprotein level was within the normal range. Abdominal ultrasonography appeared normal. Abdominal computed tomography (CT) with intravenous and oral contrast was performed (Figure 1A). It was reported as normal by the radiologist. The patient's symptoms resolved, and he was discharged following a general surgery consultation.

Three hours after discharge, the patient was readmitted with a recurrence of symptoms. He reconsulted with general surgery and was hospitalized for treatment. He was treated with intravenous fluids and analgesics and given nothing by mouth. His second abdominal CT, taken after hospitalization, was consistent with acute edematous pancreatitis (Figure 1B). His Ranson score was 0. In the days following surgery, his triglyceride levels were normalized gradually with relief of symptoms, and he was discharged after 6 days of treatment. He was followed by endocrinology as an outpatient.

Discussion

Hypertriglyceridemia is a well-established cause of AP. It is generally believed that triglyceride levels of more than 1000 mg/dl precipitate an episode of AP [3]. HIAP accounts for 1-4% of all cases of AP [4]. Hypertriglyceridemia can present with acute pancreatitis, recurrent pancreatitis or chronic pancreatitis, although it is rare.

The exact pathogenesis of HIAP still remains unclear. One proposed mechanism for this phenomenon is that triglyceride-rich chylomicrons impair the circulatory flow in the pancreas, causing edema, hemorrhages and elevated amylase levels [5]. It is also believed that high levels of triglycerides are hydrolyzed and accumulate to free fatty acids in high concentrations [6]. These toxic molecules cause damage while activating trypsinogen and causing AP.

The common clinical scenarios of HIAP include poorly controlled diabetic patients with or without hypertriglyceridemia, young patients with hypertriglyceridemia secondary to alcohol or drug abuse or patients with familial hypercholesterolemia. Secondary causes of hypertriglyceridemia are alcohol, diabetes, pregnancy and medications including estrogens. The symptomatology of HIAP is similar to other forms of AP, mainly abdominal pain, nausea and vomiting. The clinical course of AP secondary to HTG is no different than with other causes of AP, and HIAP should be treated in the same manner as other causes of AP [7].

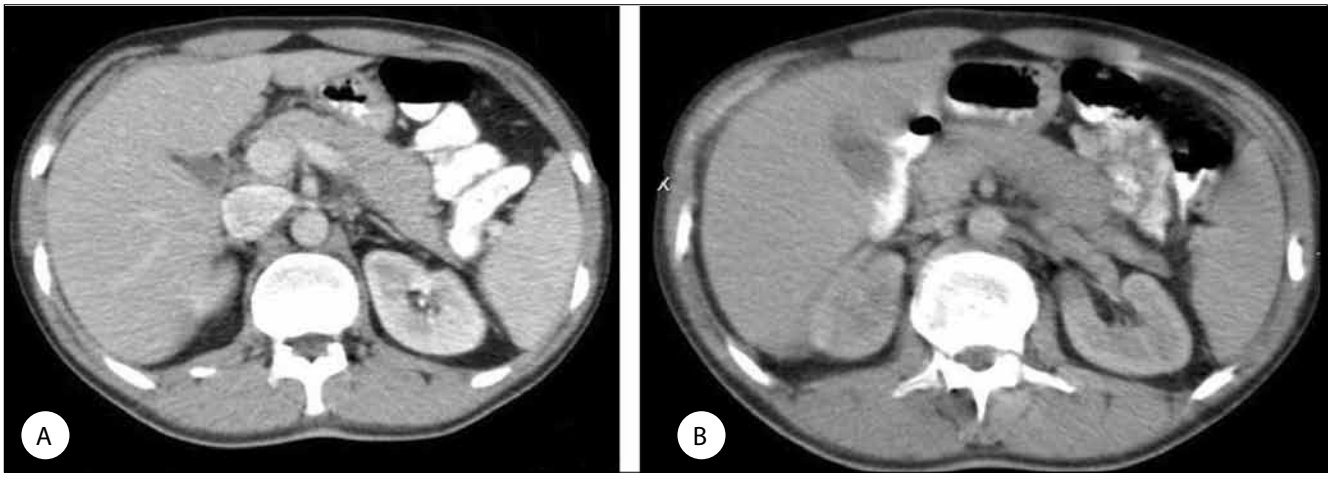

Figure 1. *Computed tomography of abdomen A. At the time of first admission to emergency department (reported as normal) B. After hospitalization (consistent with edematous acute pancreatitis) .*

Initial treatment of AP in cases with HTG is similar to treatment for other causes of AP, including bowel rest, analgesics, intravenous hydration and cessation of oral intake [2]. Fasting lowers triglyceride levels as quickly as 24 to 48 hours after onset in the majority of patients. If total parenteral nutrition is required, lipid infusions must be avoided, as they increase triglyceride levels. Although triglyceride levels fall rapidly after the cessation of oral intake, utilization of insulin therapy or efforts for the removal of lipoproteins, such as plasmapheresis, have been considered in case reports [4].

Elevated levels of serum amylase and lipase are frequently used in diagnoses of AP, but the lower cost and wider availability of amylase make it the preferred method. A serum triglyceride level greater than 500 mg/dl tends to show falsely normal levels of amylase [4]. This is thought to be caused by an in-vitro interaction with high levels of lipid particles in lactescent serum. Serial dilutions of the serum can reduce this phenomenon and reveal abnormal levels of amylase [8]. Moreover, there are fewer available data interpreting the levels of lipase in HIAP [2, 4]. Navarro et al. reported that serum amylase and lipase levels supported the diagnoses of HIAP in 26% and 58% of cases, respectively [9].

Some patients with hypertriglyceridemia suffer from episodes of mild pancreatitis, known as "hyperlipidemic abdominal crisis" [10]. It is characterized by abdominal pain and vomiting. Serum amylase and lipase levels are frequently within the normal range with a normal ultrasonic evaluation. Despite normal laboratory findings and radiologic imaging, these patients are at risk for developing AP. We believe that when the CT imaging showed normal pancreatic imaging at the first admission, our patient was suffering from a hyperlipidemic abdominal crisis; however, in the following hours, he developed AP, with typical findings with CT recorded after the hospitalization.

The diagnosis of HIAP can be missed with normal levels of amylase and lipase, and in many cases, ultrasonographic evidence may be absent [2]. The resolution of symptoms in overcrowded EDs may lead to discharge of patients. ED physicians must keep in mind that patients with abdominal pain consistent with AP and lactescent serum, but with normal amylase levels and normal radiologic findings, may have HIAP and should be observed with hospitalization, although the CT imaging may appear normal.

Conflict of interest statement: The authors declare that they have no conflict of interest to the publication of this article.

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