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Acute Pancreatitis Concomitant Acute Coronary Syndrome

Akut Koroner Sendrom ve Akut Pankreatit Birlikteliği

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ABSTRACT

Acute pancreatitis is an inflammatory syndrome with unpredictable progression to systemic inflammation and multi-organ dysfunction. As in our case rarely, acute pancreatitis can be presented with the coexistance of acute coronary syndrome. To prevent a misdiagnosis of acute situation presented with chest or abdominal pain, physicians must be aware for coexisting pathophysiologies and take into account the differential diagnosis of all life-threatening causes such as cardiac ischemia or acute abdominal situations.

Key Words: *Acute coronary syndrome; pancreatitis, acute necrotizing.*

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ÖZET

Akut pankreatit progresyonu çoğu zaman öngörülemeyen sistemik inflamasyon ve multiorgan disfonksiyonuna neden olabilen bir durumdur. Olgumuzda olduğu gibi nadiren akut pankreatit akut koroner sendrom birlikteliği görülebilmektedir. Göğüs veya karın ağrısı şikayeti nedeniyle başvuran hastaların tanısında yanılgıya düşmemek için hayati önem taşıyan kardiyak iskemi veya akut batın nedenlerinin birlikte görülebileceği ve birbirlerinin etyopatogenezinde rol oynayabileceği akılda tutulmalıdır.

Anahtar Kelimeler: *Akut koroner sendrom; pankreatit, akut nekrotizing.*

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INTRODUCTION

Acute pancreatitis is a complex inflammatory syndrome with unpredictable progression to systemic inflammation and multi-organ dysfunction. In spite of modern treatment, rates of death with acute coronary syndrom remain high. Herein we report a patient with acute pancreatitis and acute coronary syndrome.

CASE REPORT

A 51-year-old man with a medical history of gall-stone presented severe epigastric pain and retrosternal chest pain associated with nausea, vomiting, and diaphoresis for

eight hours. On admission to our emergency department, he was afebril, with blood pressure of 115/88 mmHg and respiratory rate of 13 /min. Oxygen saturation was 98% on inhaled room air. Her lung sounds were clear bilaterally and cardiovascular examination was normal, without murmurs, rubs or gallops. A 12-lead electrocardiography (ECG) revealed sinus rhythm with ST-segment depression in leads V5-V6. A diagnosis of acute coronary syndrom was initially suspected and the patient was admitted to the coronary care unit. He received acetilsalicilic acid, intravenous nitroglycerin and metoprolol. His chest pain improved with these treatment but epigastric pain persisted. Abdominal examination showed tenderness in the epigastrium and right lower quadrant. Computed tomographic scan of the abdomen revealed inflammatory changes within the adjacent peripancreatic fat, consistent with pancreatitis (Figure 1). Serum biochemistry results were available after catheterization and showed creatinine 0.9 mg/dL (0.6-1.1), amylase 2578 U/L (35-115), lipase 845 U/L (5-85), alanine aminotransferase 63 U/L (5-45), aspartate aminotransferase 57 U/L (5-45), total bilirubin 1.0 mg/dL (0.1-1.1), alkaline phosphatase 352 U/L (98-295), γ-glutamic transpeptidase U/L (3-52), albumin 3.76 g/dL (3.2-5.5), triglycerides 130 mg/dL (850-170), and calcium 8.1 mg/dL (8.4-10.2). Serum electrolytes and creatinine kinase were normal, and serum troponin T was positive (1.2 ng/mL). The patient was treated with adequate analgesia, intravenous fluid, electrolyte repletion, and cessation of oral intake. He symptomatically improved, his chemistries returned to normal levels, and he was discharged after 10 days. After discharge, treadmill test was performed that revealed ST segment deviatiaon. He was taken for cardiac catheterization, which revealed 90% stenosis of proximal segment of the left descending coronary artery and treated by percutaneous coronary intervention (PCI) (Figure 2).

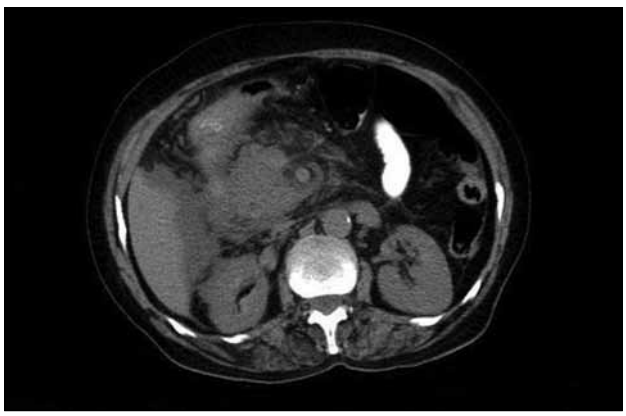

Figure 1. Abdominal contrast-enhanced computed tomography showing irregularity of the pancreas and pancreatic diffuse edema.

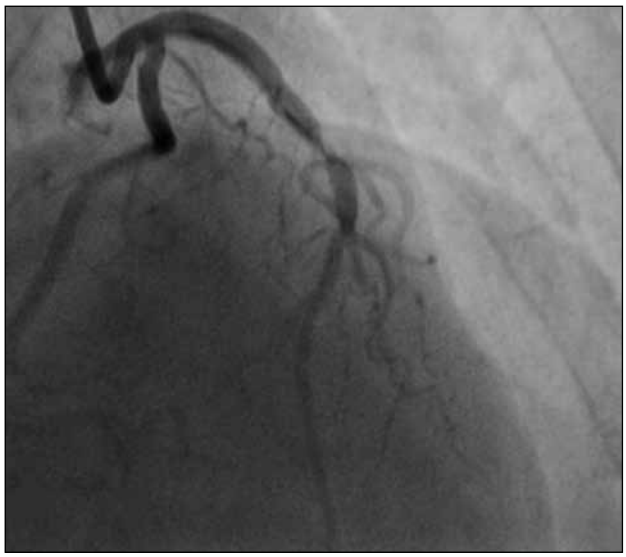

Figure 2. 90% stenosis of proximal segment of the left descending coronary artery.

DISCUSSION

Acute pancreatitis is a potentially lethal disorder of the exocrine pancreas associated with acinar cell injury with local and systemic inflammation may range from mild edema to pancreatic necrosis[1]. Its incidence varies from 4.5 to 79.8 per 100.000 per year in different countries due to different diagnostic criteria,geographic factors and changes over time[2]. Mortality rate ranging from 1% to 9% is influenced by the severity of the disease and several prognostic factors[3]. Several etiologic factors have been described for acute pancreatitis but most commonly as in our case gallstones followed by alcohol intake are responsible for 80% to 90% of cases of acute pancreatitis[4]. Although the exact mechanism of acute pancreatitis is unknown several pathophysiologic processes have been described that ultimately lead to intrapancreatic zymogen activation and autodigestion with destruction of the acinar cell. Pancreatic ductal obstruction, hypersecretion, ischemia/reperfusion injury and enzymatic colocalization have been mentioned as factors that contribute to the initiation of the inflammatory process[5,6]. Intra-acinar activation of trypsinogen still plays a central role in the pathogenesis of acute pancreatitis, resulting in activation of proteases that ultimately causes cell damage. In consequences of acute pancreatitis can mimic or co exist with other pathophysiologies, the diagnosis of acute pancreatitis can be a source of confusion.As in our case rarely, acute pancreatitis can be presented with the coexistance of acute coronary syndromes. Several hypotheses have been proposed to

explain concomitance of acute pancreatits and acute coronary syndromes. Pancreatic proteolytic enzymes such as trypsin may have resulted in direct injury to the paricardium or myocyte membrane leading to changes in cell permeability with possible necrosis and consequent electrical changes seen on ECG such as T wave inversion, ST segment depression or ST segment elevation[7]. Another mechanism is the existance of a cardiobiliary reflex, which may cause cardiac damage by direct action on the myocardium or by altering coronary blood flow[8,9]. This mechanism is especially important in patients with the presence of underlying coronary artery disease[10]. Other possible mechanisms such as coronary vasospasm, exacerbation of underlying coronary artery disease or coronary thrombus formation secondary to increased platelet adhesiveness or pancreatic enzyme-induced coagulopathy[11-13]. Moreover acute pancreatittis is known to cause fluid third spacing, and this intravascular loss also may have contributed to coronary hypoperfusion.

Our case is an extreme example of acute pancreatitis coexisting with acute coronary syndrome with clinical, ECG and cardiac enzymes derangements accompanying anatomical and functional evidence of obstructive coronary disease. Although we postulate a combination of all these mechanisms may have contributed the coexistance of acute coronary syndrome with acute pancreatitis the precise reason of this coexistance remains to be elucidated fully. To prevent a misdiagnosis of acute situation presented with chest or abdominal pain, all primary care physicians must be aware for coexisting pathophysiologies and take into account the differential diagnosis of all life-threatening causes such as cardiac ischemia or acute abdominal situations.

CONFLICT of INTEREST

None declared.

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