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A case of acute pancreatitis complicated by acute coronary syndrome

Alkame Akgümüş, Ali Duygu

Department of Cardiology, Bandırma Onyedi Eylul University, Faculty of Medicine, Balıkesir, Türkiye

ABSTRACT

An inflammatory condition affecting the pancreatic parenchyma is called acute pancreatitis. Alcohol consumption and gallstones are the most frequent etiological factors. Patients with acute pancreatitis can appear clinically in a variety of ways. While individuals with acute pancreatitis may experience ECG abnormalities that resemble acute myocardial infarction, it is uncommon for acute pancreatitis to coexist with actual acute myocardial infarction. Our goal was to present a case of acute pancreatitis exacerbated by acute coronary syndrome. The patient arrived at the emergency room with 60 minutes of epigastric abdominal pain that was prominent there and radiating to the back.

Keywords: Acute pancreatitis, acute coronary syndrome, anjioplasty

Acute pancreatitis is an inflammatory disease of the pancreas clinically characterised by abdominal pain accompanied by elevated serum levels of pancreatic enzymes (amylase and lipase). Pain is a prominent and distinctive feature of acute pancreatitis. It is localised in the epigastric region in more than 60% of patients [1]. Multiple systems are linked to a number of acute pancreatitis problems. Acute pancreatitis may result in systemic problems such as pulmonary, cardiovascular, haematological, renal, metabolic, and central nervous system abnormalities, as well as local complications such as pancreatic necrosis, abscess, or pseudocyst. Cardiovascular complications of acute pancreatitis include especially myocardial infarction (MI), shock, hypovolemia and pericardial effusion [2]. Here, we aimed to emphasise the effect of early coronary stenting on possible mortality and complications in a case of acute pancreatitis complicated with acute coronary syndrome (ACS).

CASE PRESENTATION

A 57-year-old woman was admitted to the emergency department with complaints of abdominal pain in the epigastric region for approximately 60 minutes. During hospitalisation, the temperature was 36.7 °C, blood pressure 110/70 mmHg and pulse rate 67/min. Physical examination was unremarkable except for tenderness in the epigastric region. The patient had a history of hypertension. Additionally, she had a 20-year history of smoking. The medical history obtained revealed no history of malignancy, autoimmune disease, hepatobiliary or gastrointestinal disease. Body mass index (BMI) was 33.2 (height: 1.55 m, weight: 80 kg). A prior myocardial infarction history was discovered. In the inferior leads, the ECG had abnormal Q waves. (Fig. 1).

Laboratory examination: leukocyte: 8.29/mm3, haemoglobin: 12.6 g/dL, hematocrit: 40.1%, platelet:

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Corresponding author: Ali Duygu, MD., Assist. Prof., Phone: +90 226 717 01 17, E-mail: ali.duygu1989@gmail.com, aduygu@bandirma.edu.tr

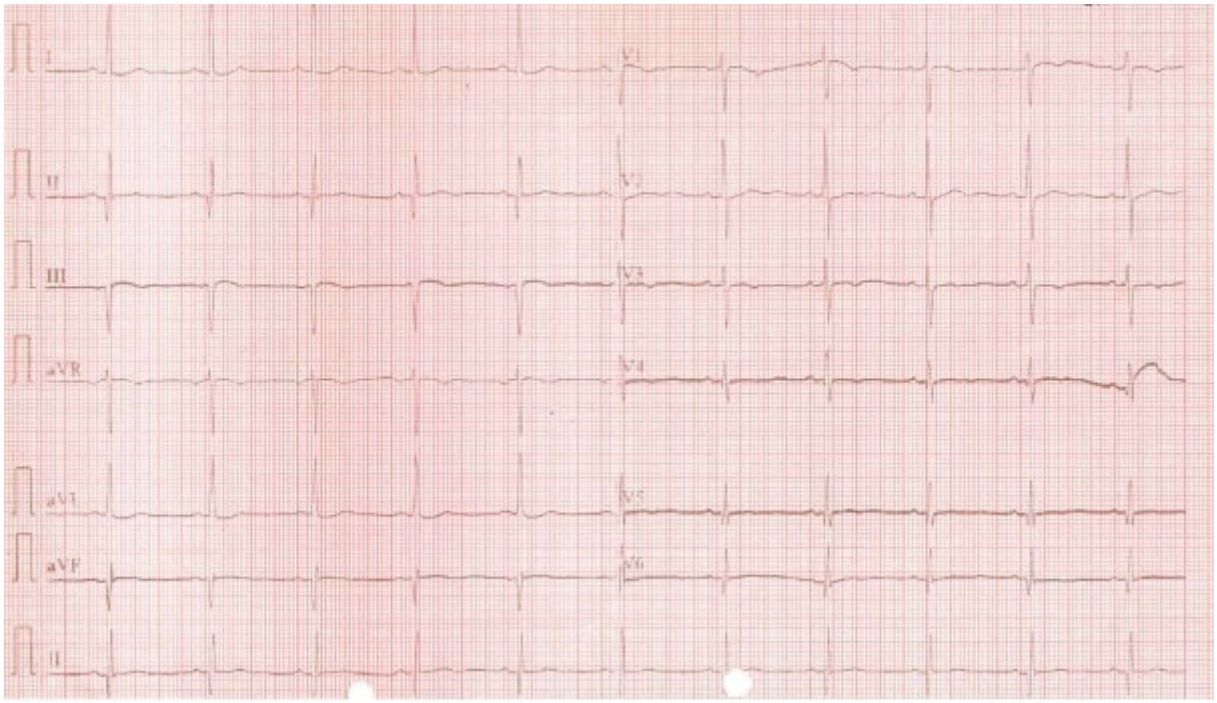

Fig. 1. ECG at the time of admission.

283.000/mm3, glucose: 144 mg/dl, urea: 30 mg/dl, creatinine: 0.98 mg/dl, alanine aminotransferase: 38 IU/L, aspartate aminotransferase: 142 IU/L, sodium: 141 mmol/L, potassium: 3.9 mmol/L, alkaline phosphatase 176 U/L and troponin 300 pg/mL (0.1-15.6). The patient was diagnosed as ACS and emergency coronary angiography (CAG) was performed. Plaque in the left anterior descending coronary artery (LAD) and circumflex coronary artery and thrombus image in the mid right coronary artery (RCA) were detected and stent placement and complete patency was achieved (Fig. 2). During coronary intensive care unit follow-up, the patient's complaints and troponin values increased and control CAG was performed and the stent in the RCA was found to be patent (Fig. 3).

Noncardiac pathologies were thought to be the cause of the patient's complaints. Abdominal examination revealed tenderness in the epigastric region.

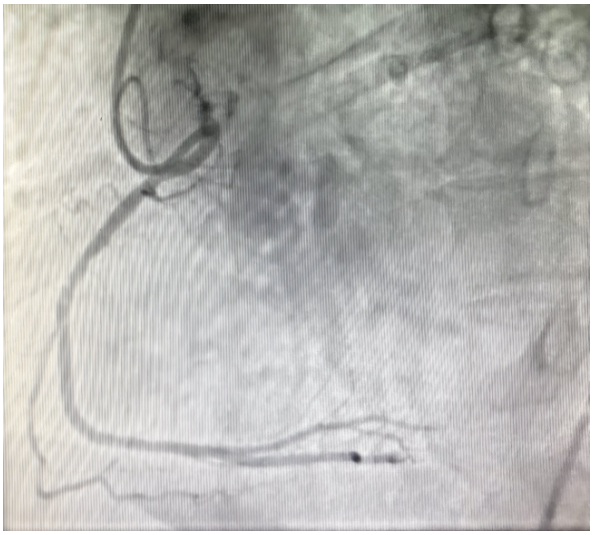

Fig. 2. RCA before stent implantation.

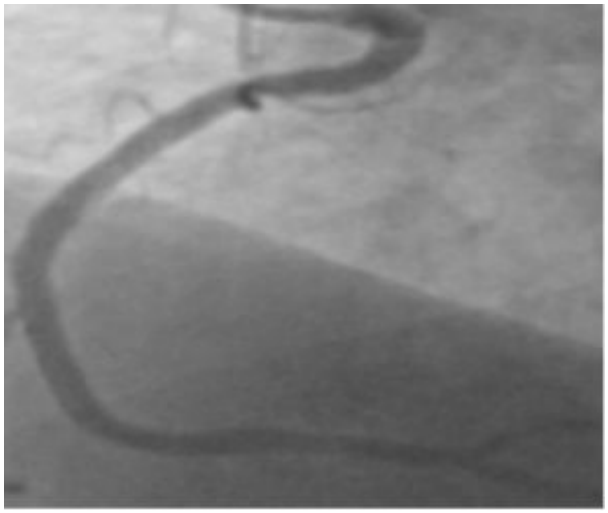

Fig. 3. Image of patent RCA after control CAG and implanted stent.

Laboratory tests were re-studied more comprehensively. Amylase value was found to be 1,218 U/L. Abdominal tomography showed heterogeneous and oedematous pancreatic head corpus and bile sludge in the gallbladder (Fig. 4). When assessed according to Ranson criteria, the diagnosis was made with a score of 3, indicating severe pancreatitis. After 48 hours of follow-up, the hematocrit was measured at 32.1 (decrease >%10), and due to fluid deficit, the score was 5, with a mortality risk of 40%.

In the coronary intensive care unit, acute pancreatitis treatment was organised simultaneously with ACS treatment. Oral intake was stopped and IV hydration treatment was started. In daily follow-up, amylase values normalised and complaints regressed. Vital values were stable, medical treatment was organised and the patient was discharged with recommendations.

DISCUSSION

Acute pancreatitis is a clinical picture with high mortality and morbidity that develops as a result of activation of inactive enzymes in the pancreas due to various reasons and autodigestion of the pancreas, manifested by severe abdominal pain and may lead to local and systemic complications. Although many factors play a role in the etiology of acute pancreatitis, biliary gallstones and excessive alcohol consumption are responsible for 70% of cases. Drugs, hyperlipidaemia, pregnancy, endoscopic retrograde cholangiopancreatography (ERCP) and trauma are among other causes. Mortality rate ranging from 1% to 9% is influenced by the severity of the disease and several prognostic factors [3, 4].

Many complications of acute pancreatitis are related to multiple systems. Local complications such as pancreatic necrosis, abscess or pseudocyst and systemic complications such as pulmonary, cardiovascular, haematological, renal, metabolic and central nervous system abnormalities may occur in the course of acute pancreatitis. While ST segment elevation is rare among cardiovascular events, other ECG findings including arrhythmia, conduction anomalies and duration changes in T wave or QT period are more common [2]. Although patients with acute pancreatitis may

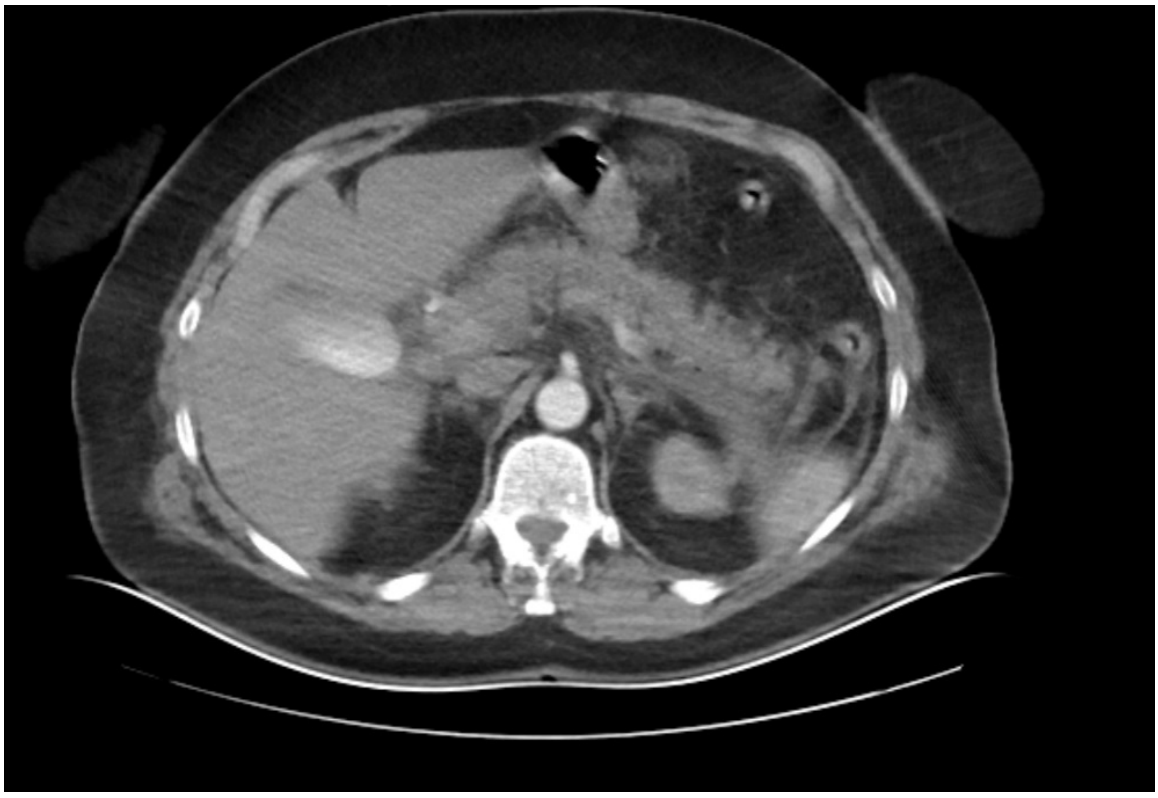

Fig. 4. Computed tomography image of acute pancreatitis.

exhibit ECG abnormalities that resemble acute myocardial infarction, it is extremely uncommon for acute pancreatitis to coexist with acute myocardial infarction [5]. Cases of acute infarction with elevated troponin level detected with chest pain despite a normal ECG have also been described [6]. The mechanism of myocardial involvement observed in the course of acute pancreatitis is not very clear. According to clinical research, intravenous injections of pancreatic proteolytic enzymes into a rabbit model have been demonstrated to induce acute myocardial necrosis, which resolves after two weeks [7]. The existence of a cardiobiliary reflex is another mechanism that might harm the heart by changing the flow of blood to the coronary arteries. Patients with underlying coronary artery disease should pay particular attention to this mechanism [8]. Furthermore, a third fluid gap is known to be caused by acute pancreatitis, and this intravascular loss might have played a role in coronary hypoperfusion. More research is still needed in this area. In our study, we tried to contribute to this aim by presenting this rare case to the literature.

There are many hypotheses proposed between myocardial disease and ECG abnormalities seen in acute pancreatitis. These include vagally mediated reflexes (cardiobiliary reflex), metabolic and electrolyte abnormalities, toxic effects of pancreatic enzymes on the myocardium, coronary artery spasm, haemodynamic dysregulation and/or systemic inflammatory response and prothrombotic conditions. It has been reported that trypsin, which has an important role in the pathophysiology of pancreatitis, may alter platelet adhesion, affect the coagulation system and lead to coronary thrombosis [9]. Under appropriate clinical conditions, ST-segment elevation on ECG is a prominent feature of acute myocardial infarction and urgent reperfusion therapy is recommended. However, other conditions in which ST-segment elevation on ECG may occur for reasons other than acute myocardial ischaemia should be well known [9]. Acute pancreatitis is both an emergency and a clinical entity that must be taken into consideration.

Acute pancreatitis is a challenging disease that can lead to many complications. Its association with ACS is rare and our case is an extreme example. Previously published studies have reported cases of ACS as a complication of acute pancreatitis. However, cases of acute pancreatitis secondary to ACS are extremely rare and there are few examples in the literature. Despite all these studies, the association of acute pancreatitis and ACS is still awaiting clarification. In our case, we think that multidisciplinary evaluation of the patients can broaden the physician's perspective and it is extremely important in terms of patient survival by making the diagnosis of diseases with similar symptoms quickly.

CONCLUSION

In conclusion, it should be kept in mind that patients presenting with ACS may have concurrent cardiac and/or noncardiac pathologies. It should be kept in mind that various ECG changes may also occur in noncardiac patients such as acute pancreatitis. The diagnosis of true myocardial infarction and the application of appropriate treatment approaches are extremely important in terms of morbidity and mortality of patients.

Informed Consent

Patient was informed about the purpose of the case report, and informed consent was obtained from the patient for this publication.

Ethical Statement

The authors confirm that written consent for the submission and publication of this case, including images, was obtained from the patient in line with COPE guidance.

Authors' Contribution

Study Conception: AD; Study Design: AA; Supervision: AA; Funding: AA; Materials: AD; Data Collection and/or Processing: AA; Statistical Analysis and/or Data Interpretation: AD; Literature Review: AA; Manuscript Preparation: AD and Critical Review: AA.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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Editor's note

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