

# ACUTE NECROTIZING PANCREATITIS COMPLICATED WITH ST ELEVATION ACUTE MYOCARDIAL INFARCTION: A CASE REPORT AND LITERATURE REVIEW

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Acute pancreatitis complicated with acute myocardial infarction has rarely been reported and the precise mechanisms of myocardial injury remain unclear. We report a 49-year-old man presenting with epigastralgia who had been hospitalized for acute necrotizing pancreatitis, and who subsequently developed ST elevation myocardial infarction. The patient eventually died because of severe heart failure and complications of progressive necrotizing pancreatitis. Although a standard management protocol for these patients has not yet been developed, administration of thrombolytic agents may cause severe complications based on the limited case reports already published. We suggest that coronary angiography and further interventions such as angioplasty and possibly stenting should be performed in these cases.

**Key Words:** acute coronary syndrome, acute myocardial infarction,  
acute pancreatitis, pancreas  
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Acute pancreatitis is an inflammatory disease of the pancreas clinically characterized by epigastralgia and elevated pancreatic enzymes, as revealed by blood tests [1]. Many complications of acute pancreatitis involve multiple systems. Cardiovascular complications include shock, hypovolemia, pericardial effusion, and even nonspecific ST–T changes in the electrocardiogram (ECG) mimicking acute myocardial infarction. However, acute pancreatitis with ECG signs of ST

elevation has rarely been reported and most of the cases were not true myocardial infarction according to cardiac enzyme levels and echocardiographic or angiographic findings. Here, we describe a 49-year-old patient who developed ST elevation myocardial infarction as a complication of severe acute pancreatitis, and review the associated literature.

## CASE PRESENTATION

A 49-year-old man was hospitalized with abdominal pain over the epigastric area. The abdominal pain was characterized as cramping without radiation to the back and no obvious aggravating or relieving factors were noted. Other associated symptoms prior to



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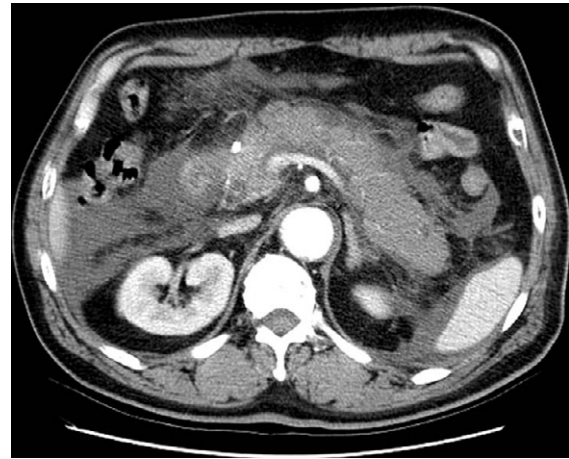
admission included bouts of nausea and vomiting, but there were no signs of fever or other toxicities.

On arrival at the emergency department, his pulse rate was 108 beats/min and systolic/diastolic blood pressure was 130/85 mmHg. Physical examination revealed tenderness over the epigastric area and crackles over bilateral lower lung fields. Biochemical studies showed sodium of 144 mmol/L, potassium of 4 mmol/L, amylase of 1,401 U/L, lipase of 935 U/L, and C-reactive protein level of 188 µg/mL in serum. Chest X-ray revealed cardiomegaly with pulmonary congestion and abdominal computed tomography (CT) revealed Grade E acute necrotizing pancreatitis and choledocholithiasis (Figure 1). The patient was then admitted to the hepatobiliary ward for the treatment of acute pancreatitis. Patient's medical history revealed that a myocardial infarction had occurred several years previously, but without receiving percutaneous coronary intervention. The patient had no history of hypertension, diabetes mellitus, or hyperlipidemia.

During his stay in the hepatobiliary ward, the patient initially received supportive care with intravenous fluids and nutrition. Forty-eight hours after admission, the Ranson score was 6, and the CT severity index was 10 (the highest score on the CT severity index). Both results indicated a high probability of morbidity and mortality and were compatible with the poor prognosis of Grade E necrotizing pancreatitis. Acute dyspnea developed during the hospitalization and ECG showed ST segment elevation, QS pattern in leads V1–V4 and pathologic Q wave in III, and aVF. Interestingly, an ECG performed approximately 6 months earlier showed only leads III, aVF with pathologic Q wave (Figures 2A and 2B).

Initial cardiac enzyme analysis revealed serial changes in creatinine kinase MB of 19.9 U/L and Troponin I of 0.5 ng/mL. Subsequent cardiac enzyme analysis revealed a peak creatinine kinase MB of 77.4 U/L and Troponin I of 82.2 ng/mL. Therefore, the patient was transferred to a cardiac care unit for further intensive care.

On arrival at the cardiac care unit, his pulse rate was 132 beats/min and systolic/diastolic blood pressure was 90/50 mmHg. Bedside echocardiography revealed poor left ventricle systolic function (ejection fraction: 36%) with septal and left ventricular posterior wall motion abnormalities. Additionally, after the patient developed hemodynamic instability and



**Figure 1.** Abdominal computed tomography showing Grade E acute necrotizing pancreatitis with a Computed Tomography severity index score of 10.

poor urine output, pulmonary artery catheterization was performed and revealed high pulmonary artery wedge pressure (25 mmHg), high systemic vascular resistance (2,766 dyn.sec/cm<sup>5</sup>) and low cardiac index (2.03 L/min).

Although the above findings clearly indicated acute myocardial infarction complicated by cardiogenic shock, the family refused further invasive coronary intervention. Heparinization was done for anticoagulation but was discontinued after coffee ground substances were noted in the nasogastric tube.

Empirical antibiotics with meropenem were administered to treat the severe Grade E necrotizing pancreatitis, but the inflammation, as evaluated by the C-reactive protein level, did not improve and gradually became worse over time. An abdominal CT confirmed worsening of the acute pancreatitis with pseudocyst formation. The patient died of severe heart failure complicated with severe acute pancreatitis several days later.

## DISCUSSION

Local complications of acute pancreatitis include pancreatic necrosis, abscess or pseudocyst, and systemic complications such as pulmonary, cardiovascular, hematologic, renal, metabolic, and central nervous system abnormalities. In terms of cardiovascular events, ST segment elevation is relatively rare whereas other ECG findings such as arrhythmia, conduction



**Figure 2.** (A) An electrocardiogram performed in April 2006 showed III, aVF with pathologic Q wave. (B) An electrocardiogram performed in December 2006 showed ST segment elevation and QS pattern in leads V1–V4 as well as pathologic Q wave in lead III and aVF.

abnormalities, and duration change in the T wave and QT period are relatively common [2]. Although complications involving pseudo or true myocardial infarction are very rare, ECG changes mimicking acute myocardial infarction in patients with acute pancreatitis have been documented before now. In the few reports of pseudo myocardial infarction, cardiac enzymes, ECG data, coronary angiographic findings and, sometimes, postmortem examinations were generally normal, despite an ST–T change mimicking

myocardial infarction [3–11]. Nevertheless, cases of acute pancreatitis complicated with true acute myocardial infarction are very rare [12,13]. In 2005, Korantzopoulos et al reported a case which they claimed was the third report of myocardial infarction coexisting with acute pancreatitis [12].

However, the precise mechanisms of myocardial injury during the course of acute pancreatitis remain unclear. The hypothesized relationships between ECG abnormalities or myocardial injury and acute

pancreatitis [14–17] include the following: (1) vagally mediated reflexes (cardiobiliary reflex); (2) metabolic and electrolytic abnormalities; (3) toxic effects of pancreatic enzymes on myocardium; (4) coronary artery spasm; (5) hemodynamic instability and/or systemic inflammatory response induces cardiac damage such as severe sepsis or septic shock; and (6) prothrombotic derangement and other hypothesized causes.

The presence of ECG signs for ST elevation in patients with acute pancreatitis is a rare phenomenon and most of the previously reported cases were actually pseudo myocardial infarction [3–11]. The patient reported here represents true myocardial infarction based on the changes in cardiac enzyme levels, and echocardiographic and ECG findings. Because our case had prior history of myocardial infarction, the underlying acute pancreatitis might make the patient more prone to myocardial damage. It has been reported that trypsin can change platelet adhesiveness, influence the coagulation system, and lead to coronary thrombosis [14]. In addition, unstable hemodynamic status may represent another mechanism that may reduce coronary flow and cause myocardial ischemia, particularly in patients with coronary artery disease.

The initial differential diagnosis of pseudo or true myocardial infarction is important because their treatment strategies differ markedly. Erroneously administering thrombolytic agents after incorrectly diagnosing acute myocardial infarction may result in a disastrous patient outcome. For example, Cafri et al reported a 54-year-old man who underwent thrombolytic therapy after being misdiagnosed with myocardial infarction [17]. Meanwhile, Main et al reported a 47-year-old male with alcohol-related acute pancreatitis who died of severe retroperitoneal hematoma apparently related to thrombolytic therapy [18].

Although the time to onset of acute myocardial infarction could not be precisely determined in our patient, coronary angiography for patients with cardiogenic shock who are candidates for revascularization or persistent hemodynamic instability was still the class I indication according to current American College of Cardiology/American Heart Association guidelines. In addition, the opportunity and indications for operation in patients with acute necrotizing pancreatitis are still controversial according to the medical literature [19]. Some reports have shown that early surgery for pancreatic necrosis could result in a worse prognosis than late surgery. However, some

reports have confirmed the efficacy of non-surgical management. Therefore, intensive non-surgical treatment might be considered as an initial treatment for patients with severe necrotizing pancreatitis to delay surgery [19].

A standard management protocol has not yet been defined because of the paucity of reports of pancreatitis complicated with acute myocardial infarction. However, coronary angiography is essential to avoid the potentially lethal consequences of thrombolytic therapy in patients with acute myocardial infarction. Angioplasty and coronary stenting could also be performed immediately if needed.

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# 急性壞死性胰臟炎併發 ST 段上升之急性心肌梗塞： 個案報告及文獻回顧

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急性胰臟炎併發急性心肌梗塞在過去的文獻中是相當罕見的，而且造成此心肌受損的詳細機轉目前仍然不明確。我們報告一位以上腹痛為表現之 49 歲男性，他因為急性壞死性胰臟炎而住院，接著發生了 ST 段上升之急性心肌梗塞。病人最後因為嚴重之心衰竭以及逐漸惡化之壞死性胰臟炎而死亡。雖然針對此類病患之標準治療方法尚未被確立，根據過去有限的病例，使用血栓溶解劑治療可能會導致嚴重之合併症。因此我們建議冠狀動脈攝影術應該是較佳的選擇。在這一類的患者，若有需要，進一步的介入性治療，如氣球擴張術以及支架置放術也應該被施行。

**關鍵詞：**急性冠心症，急性心肌梗塞，急性胰臟炎，胰臟  
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