

## Deceived by Acute Pancreatitis Masquerading as Acute Inferior Myocardial Infarction

### Dear Editor,

A 48-year-old man presented to the emergency department with acute onset “crushing” chest pain of approximately 15 minutes duration, associated with diaphoresis and dyspnoea. His cardiovascular risk factors included diabetes mellitus and hypertension. He also underwent living-related renal transplantation in 1990 and 2003 (failure of the first allograft) for end-stage renal failure secondary to IgA nephropathy. He was a non-drinker and did not have a history of gallstone disease. On presentation, the patient was in distress and diaphoretic, but haemodynamic condition was stable. Twelve-lead electrocardiogram (ECG) showed a 1-mm ST-segment elevation in leads II, III, AVF, with reciprocal changes in leads I, AVL (Fig. 1, arrows). A diagnosis of acute inferior ST-segment elevation myocardial infarction was made. In view of the early presentation, fibrinolytic therapy with recombinant tissue plasminogen activator was administered in the emergency department.

The patient was transferred to the coronary care unit for observation. However, the chest pain persisted and extended to the epigastric region over the subsequent few hours. Serial measurements of the serum level of cardiac enzymes were normal. Repeated physical examination showed that the abdomen was slightly guarded, and there was epigastric tenderness on palpation. A diagnosis of acute abdomen was then suspected. Additional laboratory investigations revealed an elevated white cell count ( $23 \times 10^9/L$ , normal:  $3.2-8.9 \times 10^9/L$ ), raised serum lipase level (2427 U/L, normal: 5-50 U/L), amylase level (1427 U/L, normal: <110 U/L), alkaline phosphatase level (154 U/L, normal 40-130 U/L) and bilirubin level (48  $\mu\text{mol/L}$ , normal 5-30  $\mu\text{mol/L}$ ). The diagnosis was hence re-adjusted to acute

pancreatitis; and a referral to the gastroenterologist was made. The patient soon developed severe septicaemia with altered mental state, and the condition was complicated by disseminated intravascular coagulopathy as well as acute renal failure. Magnetic resonance imaging (MRI) scan of the abdomen confirmed the absence of necrotising or haemorrhagic pancreatitis. The patient was treated with aggressive antibiotic therapy including vancomycin and meropenam. A myocardial perfusion scan performed 10 days later showed normal coronary perfusion and a post-stress left ventricular ejection fraction of 61%. He recovered gradually, and was discharged 2 weeks after admission. He remains well and there have been no symptoms suggestive of coronary artery disease in the subsequent 2 years.

We present a case of acute pancreatitis masquerading as acute inferior myocardial infarction. Given the presentation of chest pain with typical ST-segment elevation on twelve-lead ECG, the diagnosis of an acute myocardial infarction is indubitably the first that comes to mind. This case illustrates that to assume a single diagnosis with such conviction can be a blunder. Chest pain with ST-segment elevation on ECG is an uncommon presentation for acute pancreatitis. A high index of suspicion is required to diagnose this condition. When suspected, emergency coronary angiography with the view of angioplasty, which obviate the bleeding risk from fibrinolytic agent and facilitate early referral to a gastroenterologist if needed, might be the preferred treatment option.

There were 2 reasons why fibrinolytic therapy, rather than percutaneous revascularisation, was chosen as the reperfusion strategy for this patient. First, the patient presented within 1 hour after symptom onset. Within this golden-hour, efficacy between pharmacological and percutaneous reperfusion strategies is similar. Second, this patient has received living-related renal transplantation twice, and there was concern about the risk of contrast-induced nephropathy. However, as the correct diagnosis turned out to be acute pancreatitis, fibrinolytic therapy might have increased the risk of developing haemorrhagic pancreatitis, which is a potentially fatal complication. In fact, acute pancreatitis is considered a relative contraindication for fibrinolytic therapy.<sup>1</sup> Apart from exposing the patient to unwarranted fibrinolytic therapy, the mis-diagnosis also deprived the patient of prompt antibiotic therapy that may have contributed to the development of profound septicaemia and disseminated intravascular coagulopathy.

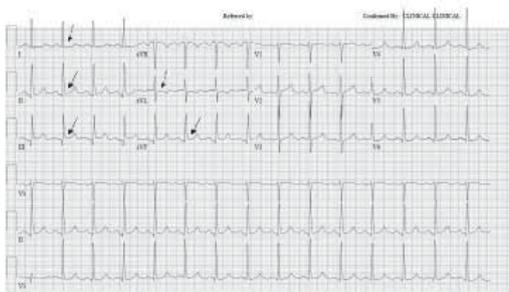


Fig. 1. Twelve-lead electrocardiogram showing 1-mm ST-segment elevation over leads II, III, aVF ( —→ ) and reciprocal ST-segment depression over leads I and aVL ( ---→ ).

Under appropriate clinical setting, ST-segment elevation on ECG is a hallmark feature for acute myocardial infarction, and prompt administration of reperfusion therapy is recommended. However, the phenomenon of ST-segment elevation on twelve-lead ECG caused by conditions other than acute myocardial ischaemia is well recognised.<sup>2</sup> Among these, cardio-respiratory conditions remain the most common underlying reasons. Acute pancreatitis has been reported to be associated with electrocardiographic changes imitating acute myocardial infarction.<sup>3-5</sup> Apart from ST-segment elevation, other associated ECG abnormalities include non-specific T wave changes, sinus tachycardia, QT prolongation and intraventricular conduction disturbances.<sup>3,4</sup> The underlying mechanism behind acute pancreatitis presenting with ST-segment elevation remains obscure. Suggested mechanisms include metabolite disturbances, cardio-biliary reflex, release of pancreatic proteolytic enzymes causing myonecrosis, and transient coronary vasospasm.<sup>3,5,6</sup>

In this case, the physician was misled by what appeared initially to be a case of ST-segment elevation myocardial infarction. Early administration of fibrinolytic therapy aimed to improve outcomes, has inadvertently delayed appropriate treatment and exposed the patient to the risk of haemorrhagic pancreatitis.

## REFERENCES

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