

## Acute ST segment elevation myocardial infarction complicated by acute pancreatitis: A case report

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### Summary

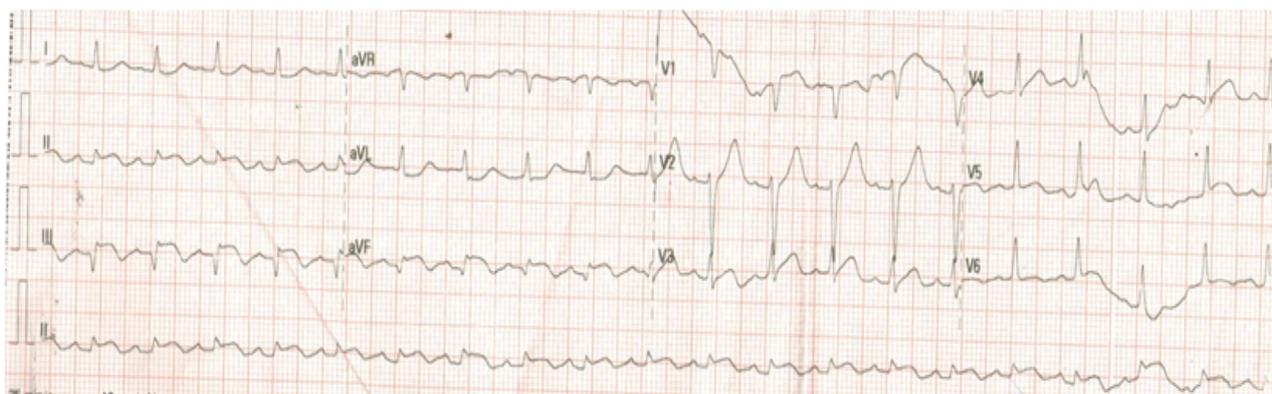
Acute myocardial infarction with acute pancreatitis is a rare association. Both have inflammation in common. But the exact mechanism of this association is unknown but systemic inflammatory response syndrome (SIRS) might be contributory. Here we report a case of acute inferior myocardial infarction treated with primary percutaneous intervention subsequently diagnosed to have acute pancreatitis on evaluation of persistent nausea and retching.

**Key words:** Acute Myocardial Infarction; Acute Pancreatitis; Inflammation

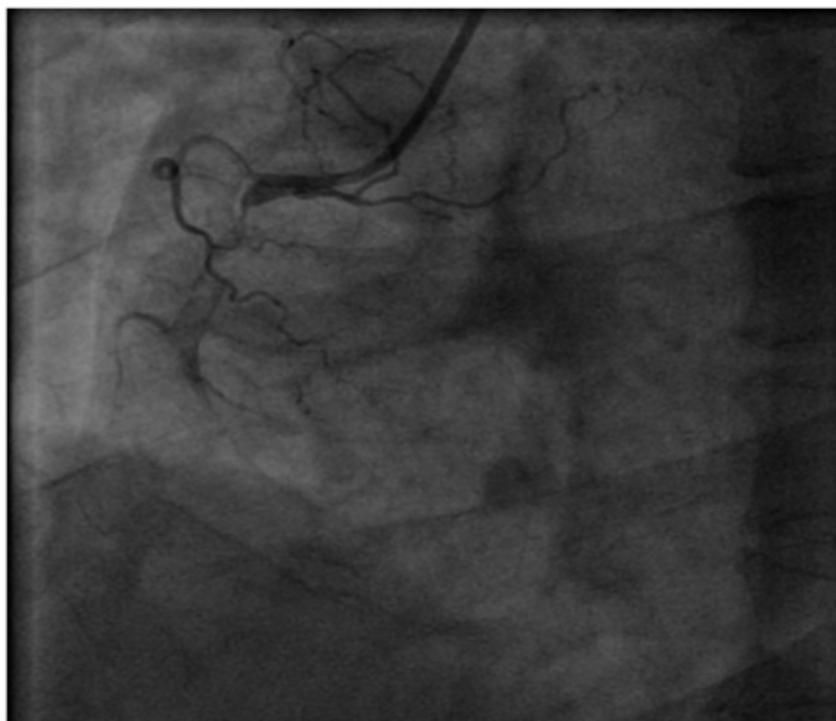
### Case report

A 54 year old diabetic male presented complaint of epigastric discomfort, retrosternal burning sensation associated with nausea and retching of 12 hours duration. At admission, pulse rate was 116/minute and blood pressure was 150/100 mmHg. Abdomen was soft and non tender. 12 lead surface electrocardiography showed ST elevation in inferior leads (II, III, aVF) with reciprocal depression in leads I and aVL (figure-1). 2D transthoracic echocardiography showed regional wall motion in right coronary artery (RCA) territory with mild left ventricular

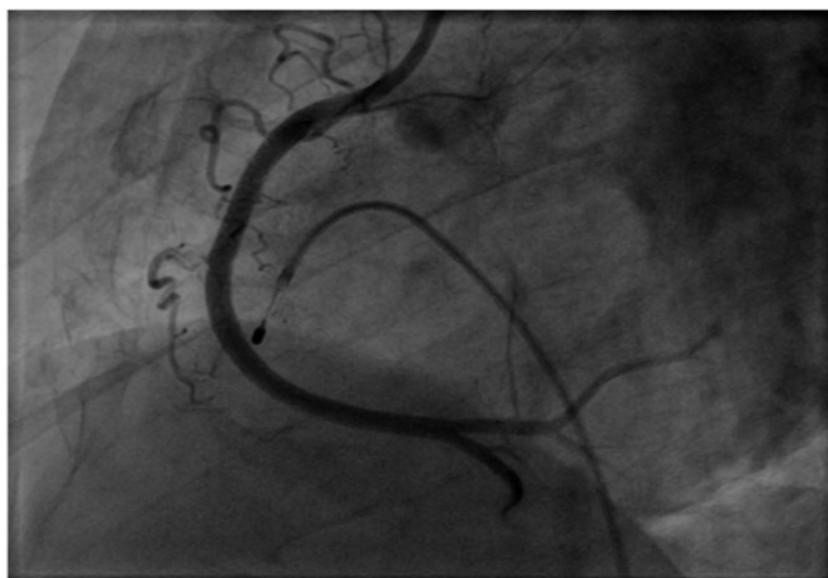
systolic dysfunction. Chest X ray was normal. Random blood sugar at admission was 260 mg/dl. Arterial blood gas analysis showed normal pH and ruled out diabetic ketoacidosis (DKA). Troponin T was elevated. Diagnosis of acute inferior wall ST segment elevation myocardial infarction (STEMI) was made and primary percutaneous intervention was planned. Coronary angiography showed 95% stenosis followed by complete thrombotic occlusion of proximal right coronary artery with normal left anterior descending (LAD) and left circumflex (LCX) arteries. Primary PTCA to RCA was done (figure-2). Proximal RCA had TIMI grade 4 thrombus. Thrombus aspiration was done with thrombosuction device was done after predilating the proximal lesion and 3.5x28 mm XIENCE Prime stent was deployed across the lesion. TIMI III flow was restored (figure-3). Intracoronary GP IIb/IIIa was given and intravenous infusion was started. Post procedure, chest and epigastric discomfort subsided but nausea and retching persisted. Liver function test was normal except for elevated alkaline phosphatase. Serum amylase and lipase were elevated 378 U/L and 490 U/L respectively. Ultrasound abdomen was normal. Patient was managed on the lines of acute pancreatitis. Serum lipase increased to 4230 U/L on day 3. Contrast enhanced CT abdomen showed mild inflammation of pancreas (figure-4). With treatment patient improved and his symptoms subsided on day 5. Patient was discharged from hospital on day 7.



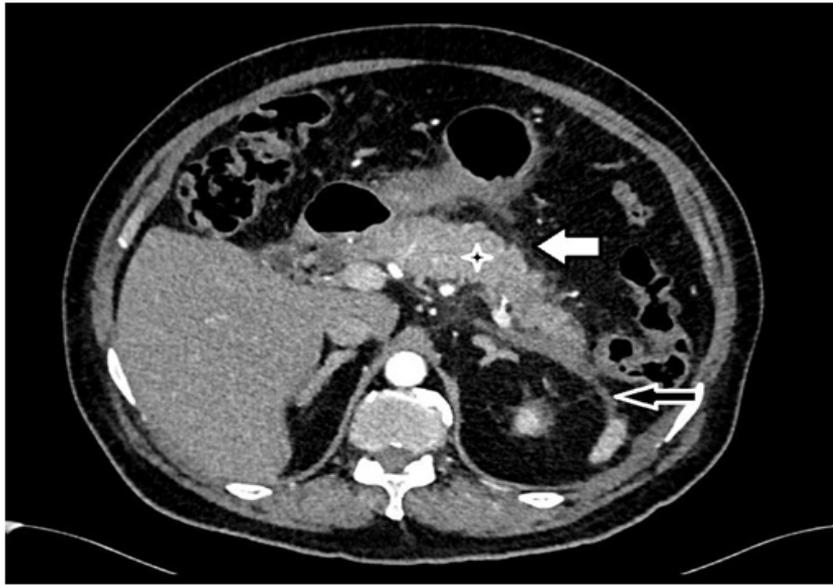
**Figure 1:** 12 lead surface electrocardiography showing sinus tachycardia and ST segment elevation in inferior leads (II, III, aVF) along with reciprocal depression in leads I and aVL



**Figure 2:** Left anterior oblique 60° angiographic view showing 95% stenosis of proximal right coronary artery followed by complete thrombotic occlusion.



**Figure 3:** Left anterior oblique 60° angiographic view showing good distal flow in right coronary artery after percutaneous intervention.



**Figure 4:** Axial section of contrast enhanced CT scan of abdomen showing bulky pancreas (star) with peripancreatic fat stranding (white solid arrow) and thickening of left Gerota's fascia (black arrow).

## Discussion

Acute myocardial infarction (AMI) triggers an acute inflammatory response, which serves to repair heart. In addition to local inflammation in the myocardium, amplified systemic inflammation response has been documented in patients with AMI. Markers for systemic inflammatory response are predictors of adverse clinical outcomes, such as death, recurrent myocardial infarction (MI), and heart failure in patients with AMI. [1] But acute pancreatitis in AMI is rare.

Acute pancreatitis is an acute inflammatory condition of pancreas. Hemostatic abnormalities are known in acute pancreatitis, and the development of a transient hypercoagulable state may be responsible for thrombotic complications. The overlap of some of the symptoms of the acute pancreatitis and AMI may cause diagnostic difficulty. Electrocardiographic abnormalities mimicking myocardial ischemia have been reported in intra-abdominal conditions, including acute pancreatitis. ST elevation in acute pancreatitis is a rare phenomenon, but other electrocardiographic changes are relatively common, including arrhythmia, conduction abnormalities and changes in the T wave and QT interval. [2] Several hypotheses proposed to explain these ST changes are vagal reflexes associated with acute pancreatitis, electrolytic abnormalities such as hyperkalemia, hypocalcaemia and hyponatremia, and severe hemodynamic disturbances such as profound hypotension. [3-4] Pancreatic proteolytic enzymes including trypsin may directly damage the membrane of the myocyte with subsequent changes of cell permeability and possible cellular necrosis, as well as secondary electrical disturbance. These enzymes may change platelet adhesiveness and influence the coagulation system, thus leading to coronary thrombosis. [5-6] Some studies revealed higher frequency of cardiovascular lesions in individuals with acute or chronic pancreatitis without relation to the common cardiovascular risk factors. [7] Although

angiographic or morphologic analysis of patients with ST elevation in pancreatitis revealed normal coronary arteries in a high percentage of cases, a cardiovascular investigation is recommended in such cases. [8] Acute pancreatitis complicated with true myocardial infarction is very rare. [9] The initial differential diagnosis of pseudo or true myocardial infarction is important because their treatment strategies differ markedly. Erroneously administered thrombolytic agents in pseudo MI cases may result in disastrous outcome. [10] Management issues include the choice of revascularization therapy, the safety of antiplatelet and anticoagulant therapy, intravenous fluid administration, and the use of cardiac medications that potentially can cause hypotension.

This patient presented with acute myocardial infarction and was subsequently diagnosed to have acute pancreatitis which recovered with conservative management. Though the association of these two entities is uncommon but they both have inflammatory cascade giving rise to SIRS which may be the probable explanation of this association. The present patient only had persistent retching and nausea as the predominant symptoms post PCI and diagnosis of pancreatitis could have been easily overlooked. This case highlights the fact that though the possibility of pancreatitis in acute MI is remote but still a simple blood test, serum lipase may guide to the diagnosis if patient has gastrointestinal (GI) symptoms.

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