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A Case Of Acute Pancreatitis Complicated With Non St Elevation Myocardial Infarction

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Abstract

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Introduction

BACKGROUND

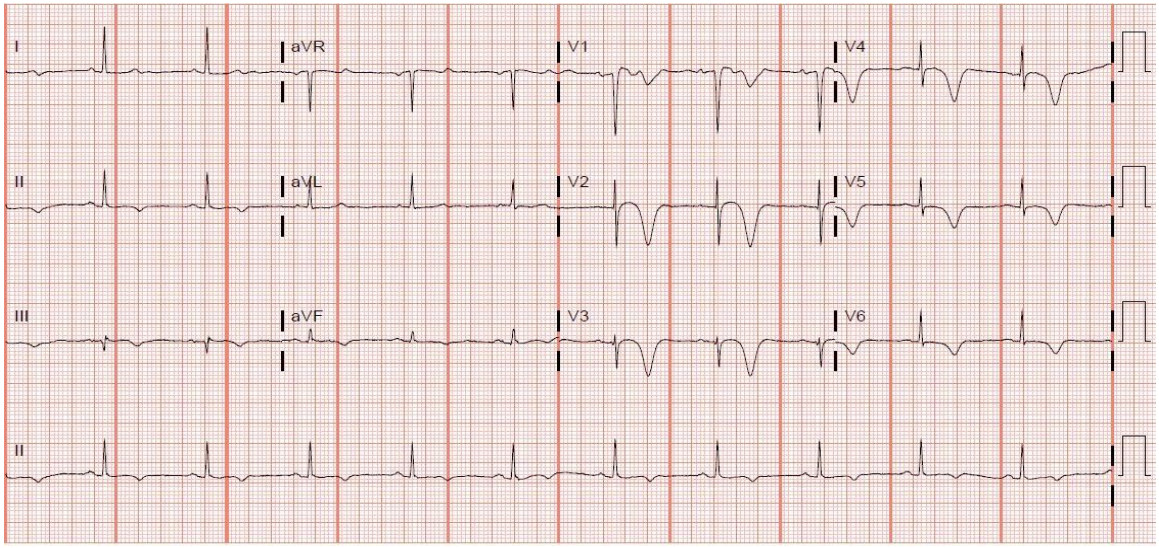
Acute pancreatitis is an inflammatory condition of pancreas which can range in severity from mild to severe disease. Most patients develop self-limiting disease but a minority of patients progress to a severe form with both systemic and local complications. Cardiovascular complications of acute pancreatitis include shock with systemic inflammatory response syndrome, hypovolemia, pericardial effusions and non-specific ST segment changes.Acute pancreatitis can be associated with electrical changes mimicking acute coronary syndrome with normal coronary arteries. The association of acute pancreatitis with ST-segment elevation and elevated cardiac enzymes has been reported in few observations. The pathophysiological mechanisms of this association remain poorly understood.

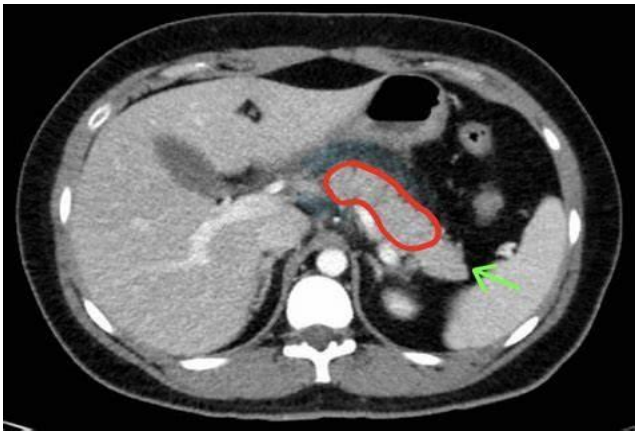
Here we report a case of 48year old lady who developed a non ST elevation myocardial infarction as a complication of acute pancreatitis.

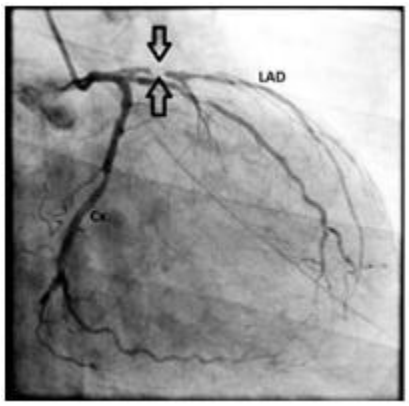
CASE PRESENTATION

A 48 year old lady was admitted to our unit with acute onset epigastric pain and vomiting for 4 h duration. She is a diagnosed patient with type 2 diabetes mellitus for 9 years and hypertension for 5 years. Her diabetes was well controlled. She is a alcoholic. Following admission she complained of retrosternal tightening type of a chest pain suggestive of acute coronary syndrome. Pain was sudden onset, lasted for 20 min in moderate intensity and responded to the medications given in hospital. Physical examination revealed severe epigastric tenderness. There was no rebound tenderness. She was haemodynamically stable with a blood pressure of 130/90 mmHg and pulse rate of 72 bpm. 12 lead electrocardiogram (ECG) revealed T wave inversions in anterior leads V1 to V4. Her serum troponin I titer was 14 ng/mL (normal less than 0.8 ng/mL). 2D echo revealed lateral wall hypokinesia with preserved left ventricular function. Her serum lipase level was seven times more than normal limit. serum amylase level was 1884U/L. Abdominal tomography demonstrated Balthazar grade B pancreatitis, without vesicular lithiasis. Serum calcium, creatinine phosphokinase (CPK) and triglyceride levels were normal. She was free of chest pain and haemodynamically stable following starting treatment with aspirin, clopidogrel, atorvastatin and nitroglycerin. Therefore she was given unfractionated heparin for 72 h and a coronary angiogram planned to be done later. pateint was started on antbioties inj carbamezepine twice was given. Her serial ECGs showed minor dynamic T wave changes. She was given intensive care and improved during next few days with standard management protocol of acute

pancreatitis. Three days later her repeat serum troponin level remained at 9.2 ng/mL and amylase level was 504 U/L. A coronary angiogram was done after 2 weeks. It revealed minor coronary artery disease with only 20% stenosis at proximal left anterior descending artery.







DISCUSSION

Electrocardiographic changes are relatively common in acute pancreatitis. They include tachy and brady arrhythmias, conduction abnormalities including bundle branch blocks and changes in T wave and QT period. These abnormalities are seen in approximately 50% of patients. Experimental studies have reported that in acute pancreatitis there are myocardial ultra-structural disturbances including interstitial edema and cardiomyocyte hypoxia, myofibril over activity, intercellular oedema between cardiomyocytes and cardiomyocyte hypertrophy with collagenation of myocardial stroma. Elevated levels of cardiac troponins can be seen among patients with acute pancreatitis without true myocardial infarctions . This can be attributable to rhabdomyolysis which is associated with acute pancreatitis. In a study on the determination of myoglobin in acute pancreatitis patients has shown that 20% of acute pancreatitis patients had serum myoglobin concentrations above the upper normal limit .

The association of stress cardiomyopathy and acute pancreatitis has been rarely described. Nine cases were previously reported, seven out of nine patients were women and age over 55 years. They all presented, often with chest pain and dyspnea a week after having a pancreatitis, troponin level was high in all patients. Regarding ECG changes, there was T-wave flattening in four patients, especially in the anterior leads and ST segment elevation was noted in three patients. In most cases echocardiography objectified apical LV akinesis with hyperkinesis of the basal area and they were treated mainly with heart failure drugs with normalization of ventricular function after six weeks .

Complete recovery of ventricular function and normalization of segmental kinetic disorders is generally seen within eight weeks. More recent studies using two-dimensional echocardiography suggest that ventricular recovery may not be complete.

Although the precise pathophysiological mechanisms of Takotsubo syndrome are not fully understood, several studies have shown the role of sympathetic nervous system which, on the occasion of an emotional, physical or combined trigger, releases an excess of catecholamines that are thought to be the cause of myocardial kinetic disorders.

The increase in the level of catecholamines leads to modification of the permeability of the sarcolemma, which in turn to accumulation of calcium in the intracellular environment followed by necrosis of the myocytes. The evolution is generally favorable, since myocardial lesions are reversible due to the short duration of exposure to catecholamines.

CONCLUSION

Above described patient's main presentation was acute pancreatitis. Also she is a known patient with type 2 diabetes mellitus and hypertension. In that context she has developed a true myocardial infarction as evidenced by high troponin I level and echocardiographic evidence of lateral wall hypokinesia. Although high troponin levels can occur with rhabdomyolysis among acute pancreatitis patients, this patient's CPK level was normal. We had no facilities to carry out an urgent coronary angiogram at out Centre. Considering her haemodynamic stability and absence of ongoing chest pain we did coronary angiogram later and it revealed only 30% stenosis at proximal left anterior descending artery. Although she had risk factors for coronary artery disease, this episode of non ST elevation myocardial infarction cannot be attributable to atherosclerotic disease. That is because of her angiogram findings.

Diagnosis of acute myocardial infarction in a context of acute pancreatitis is a challenging task. ECG changes and elevated cardiac troponins due to rhabdomyolysis can mislead towards the diagnosis of acute myocardial infarction. Subsequent treatment particularly with thrombolytic therapy can have disastrous out come in a patient with acute pancreatitis. In such a context an urgent coronary angiogram would have a pivotal role to decide on exact diagnosis and management. Management of such a patient is also challenging with the choice of revascularization therapy and safety of antiplatelet agents and anticoagulant therapy. Therefore these issues need further evaluation based on research and evidence.

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