



Acute Pancreatitis Complicated by true non ST Elevation Myocardial Infarction- A Rare Case

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Abstract

Acute pancreatitis is an inflammatory condition with different spectrum of local and systemic complications. Acute myocardial infarction is rarely reported complication of acute pancreatitis. We report a case of 56 year old female presenting with acute pancreatitis complicated by true non ST elevation acute myocardial infarction (AMI). On investigation there was raised serum amylase, lipase and cardiac troponin. Electrocardiogram (ECG) was suggestive anterolateral wall ischemia. Contrast computerized tomography abdomen revealed acute severe pancreatitis. In acute pancreatitis, Nonspecific ECG changes can occur. The diagnosis of true myocardial infarction in setting of acute pancreatitis can be challenging even using ECGs, 2D echocardiography, cardiac biomarkers and coronary angiography. Choice of revascularization therapy and use of antiplatelet agents and anticoagulant therapy is also challenging. Decision making regarding the management of such a patient is also critical.

Keywords: *Acute pancreatitis, Acute coronary syndrome, Acute myocardial infarction, Non STEMI and ECG abnormalities.*

Introduction

Acute pancreatitis is an inflammation of pancreas and peripancreatic tissue from activation of potent pancreatic enzymes particularly trypsin. It has variable severity and a range. Cardiovascular system is complicated with shock, hypovolemia, pericardial effusion and non specific ST segment changes. We report a case of acute pancreatitis who developed acute myocardial infarction in anterolateral wall. She managed successfully as

per guideline for pancreatitis and non ST elevation MI.

Case Presentation

A 56 years old lady was admitted in medicine department in Dr. RPGMC Kangra at Tanda H.P, with chief complaints of acute onset epigastric pain and vomiting for 3 days. She was admitted in CCU. She was known case of type 2 diabetes mellitus for 6 years and hypertension for 3 years.

Her diabetes was well controlled with HbA1c of 6.5% and without significant micro or macrovascular complications. She was nonalcoholic and nonsmoker. After two days of admission she developed retrosternal heaviness, ghabrahat and shortness of breath suggestive of acute coronary syndrome. Chest discomfort was sudden onset, lasting for 20 mins and responded to the medications given in hospital.

On physical examination there was epigastric tenderness no rigidity and no guarding. There was no rebound tenderness. Cardiovascular and respiratory examinations were within normal limit. Her blood pressure was 140/96 mmHg, pulse rate of 106 bpm, spo2 90% on room air, 12 lead electrocardiogram (ECG) revealed T wave inversions in inferior, anterior and lateral leads including I, aVL, Leads 2 and 3 and V3 to V6 (fig.1). Her serum amylase level on admission was 1036 U/L (20-115 U/L). Serum lipase level 189 U/L, TLC 22103/mm³, neutrophilic predominant. Serum calcium, creatinine phosphokinase (CPK) and triglyceride levels were normal. Therefore she belongs to the moderately severe acute pancreatitis according to revised Atalanta criteria (2013). Contrast Computerized tomography (CT) abdomen revealed acute severe pancreatitis without pseudo cyst formation her serum troponin I titer was 24 ng/ml (normal less than 0.8 ng/ml). 2D echo revealed RWWA lateral wall hypokinesia with preserved left ventricular function. She got free of chest pain and haemodynamically stable following starting treatment with aspirin, clopidogrel, atorvastatin and nitroglycerin. Low molecular weight heparin was given to her for five days. She was given intensive care and pancreatitis was managed with intravenous fluids and empirical antibiotics. The pancreatic pain subsided within 72 hours. Three days later her repeat serum troponin level decreased 10.7 ng/ml and amylase level was 600 U/L. Due to constrain of money coronary angiogram could not be done during hospital stay. Patient was discharged after a week. She got her coronary angiography done after 2 weeks of

discharge. It showed 30-40% stenosis at proximal left anterior descending artery.

Discussion

Systemic complications of acute pancreatitis involve cardiovascular, renal, nervous system and pulmonary system. In acute pancreatitis different electrocardiographic changes are common like tachy and brady arrhythmic, conduction abnormalities including bundle branch blocks and changes in ST -T wave and QT interval.¹ Many hypothesis have been proposed to explain these ST and T wave changes. In acute pancreatitis there is vagal reflex stimulation which can cause cardiac damage by acting directly on the myocardium and indirectly altering coronary flow by increased secretion of pancreatic enzymes. These abnormalities are seen in approximately 50% of patients². In acute pancreatitis these are myocardial ultra-structural disturbances including cardiomyocyte hypoxia, myofibril over activity, intercellular edema between cardiomyocytes and cardiomyocyte hypertrophy with collage nation of myocardial supporting cells.^{3,4} There may be dyselectrolytemia such as hypokalemia, hypocalcemia and hyponatremia in acute pancreatitis and can alter the phase of repolarization on the ECG.

Coronary perfusion can be diminished by profound hypotension and cause ischemia leading to ECG changes, mainly in those patients who have coronary artery disease. Pancreatic proteolytic enzymes including trypsin may directly affect 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.^{4,5} In acute and chronic pancreatitis some studies revealed higher frequency of cardiovascular lesions in individuals who do not have cardiovascular risk factors. There is also the possibility of exacerbation of these lesions during the pancreatic event.⁶ In high percentage of cases

some time on angiographic and morphologic analysis of patients with ST/NST elevation in pancreatitis showed normal coronary arteries. Complete cardiovascular investigation is advised in such cases. 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 limit⁷ Acute pancreatitis associated with true myocardial infarction is a rare incident and Kumara et al.⁸ have reported such a true ST elevation myocardial infarction in a young patient with acute pancreatitis. In 2005, Krantzopoulos et al.⁹ also have reported another case of myocardial infarction with acute pancreatitis. In acute pancreatitis diagnosis of acute myocardial infarction can be difficult. Our patient represents a true case of Non STEMI based on changes in the cardiac enzyme levels, ECG, echocardiographic and coronary angiographic findings.

Differential diagnosis of false or true myocardial infarction is important because their treatment plans differ markedly. If thrombolytic agents administered in false MI cases it may result in disastrous outcome. Mann et al.¹⁰ reported a 47-year old male with alcohol related acute pancreatitis, who died of severe retroperitoneal hematoma apparently related to the thrombolytic therapy. Cafri et al.¹¹ reported a 54-year old male, who underwent thrombolytic therapy after being misdiagnosed with myocardial infarction. A report by Qazi et al.¹² showed that immediate thrombolysis in acute pancreatitis when followed by coronary angiography showed 90% stenosis of right coronary artery.

Conclusion

Diagnosis of acute myocardial infarction in a case of acute pancreatitis is a challenging task. ECG changes and elevated cardiac troponins due to rhabdomyolysis can mislead regarding the diagnosis of acute myocardial infarction. Subsequent treatment particularly with thrombolytic therapy can have disastrous outcome in a patient with acute pancreatitis. In such a

condition an urgent coronary angiogram would have an important role to decide on exact diagnosis and management. A standard management protocol has not been defined because of paucity of reports of cases of pancreatitis complicated with AMI. 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.

References

1. Faintuch JJ, Abrahao MM, Giacaglia LR, et al. Electrocardiographic changes in pancreatitis. *Arq Bras Cardiol.*1989; 52:259-60.
2. Pezzilli, R, Barakat B, Billi P, et al. Electrocardiographic abnormalities in acute pancreatitis. *Eur J Emerg Med.* 1999;6:27-9.
3. Morrison M.L, Swulim W.A Role of the gastrointestinal tract in production of cardiac symptoms. Experimental and clinical observation. *J Am Med Assoc.*1940; 114:217.
4. Lieberman JS, Taylor A, Wright IS. The effect of intravenous trypsin administration on the electrocardiogram of the rabbit. *Circulation.* 1954;10:338-342.(PubMed).
5. Kellner A, Robertson T. Selective necrosis of cardiac and skeletal muscle induced experimentally by means of proteolytic enzymes solution given intravenously. *J Exp Med.* 1954;99:387-404 (PubMed).
6. Gulio L, Stella A, Labo G. Cardiovascular lesions in chronic pancreatitis. *Dig Dis Sci.*1982;27:716-722.(PubMed).
7. Spritzer HW, Peterson CR, Jones CR, et al. Electrocardiographic abnormalities in acute pancreatitis: two patients studied by selective coronary arteriography. *Milit Med.* 1969;134:687-693. (Pub.Med).

8. Korantzo Poulos P, Pappa E, Dimitrova V, et al. ST segment elevation pattern and myocardial injury induced by acute pancreatitis. *Cardiology*. 2005;103:128-130. (PubMed).
9. Hsu Po Chao, Liw Tsung Hsien, Su Ho Ming, Liw ZU Yau, Lai Wew Ter, Sheu Sheng-Hsiung. Acute necrotizing pancreatitis complicated with ST elevation acute myocardial infarction; a case report and literature review. *Kaohsiung J Med Sci*. 2010;26:200-205(PubMed).
10. Mann G, Heath D, Candlish W, et al. Dangers of thrombolysis, *BMJ*. 1990;300:811. (PubMed).
11. Cafri C., Basok A., Katz A. Thrombolytic therapy in acute pancreatitis presenting as acute myocardial infarction. *Int J Cardiol*. 1995;49:279-281. (PubMed).
12. Qazi YA, Sekovski B, Qazi KJ. Is thrombolytic therapy an option in myocardial infarction with acute pancreatitis? *AM J Med*. 2000;108:178 (PubMed).