Case Series of Pancreatitis with Uncommon Presentations

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Abstract

Acute pancreatitis is an inflammatory disease characterized by local tissue injury which can trigger a systemic inflammatory response. Vascular complications of pancreatitis are a major cause of morbidity and mortality. Pulmonary embolism in acute pancreatitis has been reported to be very rare. Cardiovascular complications include shock, hypovolemia, pericardial effusion, and even nonspecific ST-T changes in the electrocardiogram (ECG) mimicking acute myocardial infarction. Acute pancreatitis complicated with acute myocardial infarction has rarely been reported and the precise mechanisms of myocardial injury remain unclear. Here we report two cases of acute pancreatitis - one with acute pulmonary thromboembolism and other with acute myocardial injury.

Introduction

Acute pancreatitis is a sudden inflammation of the pancreas. It can have severe complications and high mortality despite treatment. Systemic complications include Acute Respiratory Distress Syndrome, multiple organ dysfunction, Disseminated Intravascular Coagulation, hypocalcaemia etc. Loco regional complications include pancreatic pseudocyst, splenic artery pseudo aneurysms, hemorrhage from erosions into splenic artery and vein, thrombosis of the splenic vein, progression to chronic pancreatitis etc.

Vascular complications of pancreatitis are a major cause of morbidity and mortality and are related to haemorrhage resulting from arterial erosion or pseudo-aneurysm, ischemic complications (either local or related to remote vascular events), and venous or arterial complications specifically splanchnic thrombosis and associated varices.¹ The frequency of pulmonary embolism in acute pancreatitis has been reported to be very rare. The diagnosis and management of ST elevation myocardial infarction (STEMI) in the setting of acute pancreatitis can be challenging. Electrocardiographic abnormalities mimicking myocardial ischemia have been reported in intra-abdominal conditions including acute pancreatitis.

Here we report 2 cases of acute pancreatitis with rare complications.

Case 1

28 year old unemployed male, chronic alcoholic came with complaints of abdominal distension of 10 days duration, which was sudden in onset, and epigastric pain which was diffuse and non colicky in nature. He also complained of dyspnoea since 3 days, class III, not associated with orthopnea or PND. He did not give history of yellowish discoloration of urine or sclera. No history of hematemesis, melena or altered sensorium. There was no history of similar episodes or any other major illness in the past.

On examination- Patient was conscious, oriented, Pulse-110/min, regular, blood pressure 100/70mm of Hg. He was febrile, tachypnoeic with respiratory rate of 30/min. JVP was not raised. There was no pallor, icterus, cyanosis, clubbing, pedal oedema or lymphadenopathy. On Per Abdominal examination - there was diffuse tenderness but no guarding or rigidity was present. Shifting dullness was present.

His routine investigations were: Hb-11.3gm%, TLC-10,200/mm³ (P-79% L-21%), Platelets-3.2lakh/mm³, BUN-5mg%, Serum Creatinine-0.8mg%, Serum Bilirubin-
1.1mg%, SGOT-36units/litre, SGPT-5units/litre, Alkaline phosphate-117units/litre, Sodium-130meq/litre, Potassium-3.7 meq/litre, Chloride-98 meq/litre, PT/INR-1.1, Serum Protein-5.7gm/dl, Serum Albumin-3.2gm/dl. Serum lipase was >4000U/L(high). Ascitic fluid was hemorrhagic with protein of 3.1gm% and sugar 110mg%.RBC; 35200, TLC; 1350 (Neutrophil-24%, Lymphocyte-76%). Fluid Amylase was18985 (N-25-115u/l, Adenosine deaminase level of 9.4U/L (Normal). X-ray chest was showing bilateral middle and lower zone haziness.

Patient was started on intravenous antibiotics and fluids, but he remained persistently tachypnoeic and hypoxic. CECT thorax was done, It showed evidence of pulmonary thromboembolism with pulmonary infarct (Figure 1). Diagnosis of Acute pancreatitis secondary to alcoholism with acute pulmonary thromboembolism was made. Patient was started on heparin. Dyspnoea of patient settled with anti-coagulation. He recovered well and was subsequently discharged.

**Case 2**

A 38 year male, businessmen by occupation presented with complaints of severe epigastric pain and dyspnoea of 2 days duration. He did not give history of abdominal distension, pedal oedema, jaundice, hematemesis, melena or altered sensorium. He was chronic alcoholic and ganja addict.He did not have past history of similar episodes or major illness like diabetes, hypertension or ischemic heart disease.

Patient was conscious, oriented, with pulse of 130/min, regular and BP of 100/70, and was febrile. Respiratory rate was 28/min. There was no pallor, icterus, cyanosis, clubbing, pedal oedema or lymphadenopathy. On per abdominal examination, there was diffuse tenderness with no guarding or rigidity.

Routine blood investigations were: Hb-20.7gm%, TLC-15,200/mm³ (P-90% L-10%), Platelets-90,000/mm³, BUN-33mg%, Serum Creatinine-1.2mg%, Serum Bilirubin-1.8mg%, SGOT-86units/litre, SGPT-26units/litre, Alkaline phosphate-49units/litre, Sodium-149meq/litre, Potassium-3.7meq/litre, Chloride-105meq/litre, PT/INR-1.1, Serum Protein-6.8gm/dl, Serum Albumin-3.8gm/dl. Serum lipase was 11045 U/L. CECT abdomen was suggestive of pancreatitis. His ECG showed qRBBB pattern (Figure 2). Troponin I was 5.42ng/ml (normal upto 0.04ng/ml). 2D ECHO was done suggestive of Regional wall motion abnormality, dilated left ventricle and ejection fraction of 20-25%. Diagnosis of acute pancreatitis with acute myocardial infarction was made.

Patient was started on low molecular weight heparin. He was not thrombolysed as pancreatitis is a relative contra-indication of thrombolytic therapy in myocardial infarction as there are more chances of haemorrhagic pancreatitis. Repeat ECG after the treatment showed persistent qRBBB pattern. He improved with the treatment and was discharged.

**Discussion**

Acute pancreatitis is an inflammatory disease characterized by local tissue injury which can trigger a systemic inflammatory response.¹ There is increasing evidence that endothelial dysfunction is one of the critical pathophysiologic manifestations in patients with severe form of acute pancreatitis.² 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.

The frequency of pulmonary embolism in acute pancreatitis has been reported to be very rare and there have been very few descriptions of it. Vascular thrombosis and hypercoagulable states complicating pancreatitis are thought to due to release of proteolytic enzymes from the pancreas and direct vasculitis. So some researchers consider the mechanism of formation of the pulmonary thrombus to be as follows: (1) a cyst communicating with the pancreatic duct penetrates into the vascular bed; (2) pancreatic juice enters the vascular bed and triggers the formation of a thrombus secondary to vasculitis; (3) hypercoagulability complicates pancreatitis and is thought to be due to a combination of hepatic dysfunction and hypertrypsinaemia (resulting in raised fibrinogen and Factor VIII concentrations) and cachexia; (4) vascular changes, due to proteolytic damage or inflammation, may also play a significant part; and (5) the acute pancreatitis provokes deleterious effects in endothelium-dependent relaxing response for Acetyl Choline in isolated mesenteric rings that were strongly associated with high plasma NOX-levels as consequence of intense inflammatory responses.

Thus pulmonary embolism can be a dreadful complication of acute pancreatitis. Early treatment with intravenous heparin is effective. A vascular filter is sometimes used in the management of vascular thrombosis in acute pancreatitis to prevent pulmonary embolism. Acute pancreatitis complicated with acute myocardial infarction has been reported, and 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 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.

In terms of cardiovascular events, ST segment elevation is relatively rare whereas other ECG findings such as arrhythmia, conduction abnormalities, and duration change in the T wave and QT period are relatively common. 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. Nevertheless, cases of acute pancreatitis complicated with true acute myocardial infarction are very rare. However, it shows that the multiple visceral damage associated with acute pancreatitis can include myocardial infarction which complications aggravate prognosis.

In our case, coronary angiography was not done as patient was not willing, but Troponin I levels were highly suggestive of myocardial injury and he had persistent ECG changes even after stabilization that could be suggestive of STEMI.

**Conclusion**

Major vascular complications in acute pancreatitis are there and have to be treated as an emergency due to their potentially fatal consequences. Haemostatic abnormalities are known in acute pancreatitis and development of transient hypercoagulable state may be responsible for thrombotic complications. Pulmonary thromboembolism is a rare but potentially lethal complication of pancreatitis. Familiarity with this complication will aid in its early diagnosis, therapy and can reduce mortality. Overlap of some of the symptoms of pancreatitis and myocardial infarction may cause diagnostic difficulties. Management issues include the choice of revascularization therapy and safety of antiplatelet agents and anticoagulant therapy. The diagnosis and management of acute MI in the setting of acute pancreatitis may be challenging.

**References**