Ischemic Pancreatitis In A Patient With Cardiogenic Shock
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Citation

Abstract
A case of acute ischemic pancreatitis in a patient with pericardial tamponade resulting from an acute ascending aortic submural haematoma (acute aortic syndrome) is described. As the patient had gallstones and no history of alcohol, gallstone pancreatitis was suspected on admission. Review of literature suggests that ischemic pancreatitis is often unrecognised.

CASE REPORT
A 71 year old gentleman with a past medical history of mild hypertension was admitted to hospital with a history of collapse. He had complained of sharp pain between his shoulder blades prior to collapse. Clinical examination within 4 hours of onset of symptoms showed the patient to be in shock with profound hypotension. Blood investigations showed acute renal failure and abnormal liver function tests with raised ALT (732 U; normal < 40 U) and amylase (234 U; normal < 100 U). A CT scan of the abdomen (figure 1) showed fluid around the pancreas suggestive of acute pancreatitis, and multiple gallbladder stones with non dilated biliary system. A large pericardial effusion was also noted on the scan.

Subsequent urgent transthoracic echocardiogram confirmed a large pericardial effusion with signs of severe pericardial tamponade accounting for the development of cardiogenic shock in the patient. An emergency surgical pericardial drainage of a large amount of blood clot through xiphisternal approach resulted in immediate improvement in his haemodynamic stability.

On the following day, ALT (2781 U) was persistently rising but bilirubin and alkaline phosphatase were within normal limits. The patient scored 3 on Ranson criteria which was indicative of severe pancreatitis. In view of the provisional diagnosis of severe pancreatitis secondary to gallstones an ERCP (figure 2) was undertaken after a detailed discussion with pancreatic surgeon, gastroenterologist and ITU consultant. ERCP showed normal common bile duct with no evidence of ductal stones or sludge.

Patient general condition gradually improved and ALT eventually returned back to normal. Subsequent transoesophageal echocardiogram confirmed a large submural haematoma in the ascending aorta and this was suggestive of aortic dissection but CT scan of the thorax excluded aortic dissection but confirmed moderate size ascending aortic aneurysm, hence the term acute aortic syndrome was coined. The patient was discharged home after 6 weeks of hospital admission and on subsequent follow up 6 weeks later patient remained well with no recurrence of pancreatic problems.

Figure 1
Figure 1: CT scan of the abdomen showing

1. a) fluid around the pancreas suggestive of acute inflammation
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Figure 2
Figure 2: ERCP showing

1. a) common bile duct with normal width and calibre and inflated balloon in low CBD
2. b) Normal pancreatic duct

DISCUSSION
The causes of pancreatitis are well known, although enzymatic injury to the pancreas is the main factor in acute pancreatitis, pancreatic ischemia due to hypoperfusion is common in a state of cardiovascular shock patients and an important etiological factor for pancreatitis. Acute ischemic pancreatitis (1) may express itself as prolonged hyperamylasemia with only minimal symptoms and signs of inflammation and most cases resolve spontaneously. However, in some cases it can be complicated with necrotising inflammation and abscess formation.

During cardiogenic shock, mesenteric organs are susceptible to ischemia due to hypoperfusion. The pancreas like the kidney is highly vulnerable to ischemic injury. In a study by Warshaw and O’Hara (2) autopsy examination of patients dying after oligemic shock showed a 9% incidence of major pancreatic injury if there was no concomitant acute tubular necrosis, but a 50% incidence with concomitant acute tubular necrosis (2). This suggest that circulatory shock is an important cause of pancreatitis but is often unrecognised. This study also revealed that no pancreatic lesions were found in patients dying within 24 hours of cardiac surgery compared to 19% incidence of acute pancreatitis in patients dying more than 24 hours after cardiac surgery. Authors in this study suggest that ischemic event takes more than 24 hours to cause meaningful insult. In our case patient had radiological and biochemical evidence of pancreatitis within 4 hours of onset of symptoms raising the possibility of gallstone induced pancreatitis.

In experimental studies involving pigs (3), simulating cardiogenic shock by inducing pericardial tamponade, it was shown that the rennin - angiotension axis plays an important role in selective pancreatic vasoconstriction leading to pancreatic ischemia. This may explain the fundamental haemodynamic etiologic of ischemic pancreatitis and may open further opportunities for research with respect to ischemic pancreatitis in humans.

In summary, we present a case of acute pancreatitis caused by ischemia due to hypoperfusion of the pancreas. This patient was found to have gallstones, which were initially thought to be the cause of pancreatitis. However the acute pancreatitis was in fact the consequence of ischemia secondary to pericardial tamponade. We emphasise that shock is an important cause of acute pancreatitis and must be considered in its differential diagnosis.

References
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