key points

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Acute pancreatitis (AP) has an overall incidence of 30.0 per 100,000 population in the UK with an annual increase of 2.7% per year. Alcohol and gallstones are the aetiology in more than 90% of cases. AP has a mortality rate of 1-7% which can increase to about 20% in the presence of pancreatic necrosis. Development of other organ complications increases the mortality significantly which can be as high as 44% for patients requiring mechanical ventilation. While alcoholic pancreatitis is more common in men, gallstone pancreatitis is the other common cause of acute pancreatitis seen more commonly in women over the age of 60.

The pathogenesis of AP broadly involves three different mechanisms – ductal obstruction, acinar cell injury and defective intracellular transport. Increased intraductal pressure, such as in gallstone pancreatitis, leads to retrograde flow of pancreatic juice leading to intrapancreatic enzyme activation. Raised calcium, triglycerides, hereditary and autoimmune aetiologies cause defective intracellular transport precipitating AP.

Any patient presenting with acute abdominal pain should be assessed for a possible diagnosis of AP. A wide range of differential diagnoses should be considered which include intestinal obstruction, perforated peptic ulcer, mesenteric ischaemia, biliary colic, cardiovascular causes such as inferior wall myocardial infarction, dissecting aortic aneurysm and possible gynaecological causes such as ruptured ectopic pregnancy.

A thorough history of the presenting illness is needed to determine the onset, duration, progress and nature of the pain. AP pain typically presents as severe epigastric pain radiating to the back and is worsened by movement, and classically leaning forwards alleviates the pain. The pain is often associated with anorexia, nausea, vomiting and decreased oral intake. Risk factors for pancreatitis should also be assessed at the time of history taking. Although alcohol and gallstones are the two most common causes other aetiologies should also be explored, in particular family history can indicate possible hereditary pancreatitis or familial cancer syndromes. On examination patients with AP are often hypovolaemic and may also have tachypnoea, diaphoresis and tachycardia.

Any patient presenting with acute severe abdominal pain outside of A&E, such as in primary care, should be urged to attend A&E promptly to facilitate a rapid diagnosis and to commence treatment (most importantly intravenous fluids), which will improve outcomes in AP and in other causes of acute severe abdominal pain. Embarking on tests in primary care such as serum amylase if AP is being considered is unlikely to be worthwhile. The diagnosis of acute pancreatitis requires two of the following criteria to be present as per the revised Atlanta classification: abdominal pain in keeping with pancreatitis; serum amylase and/or lipase more than 3 times the upper limit of normal; CT or MRI findings consistent with acute pancreatitis.