Pancreatitis: causes and symptoms

Acute Pancreatitis

Introduction

Pancreatitis is inflammation of the pancreas. It is widely accepted that it is caused by pancreatic enzymes digesting their own gland. This leads to inflammation of the pancreas.

There are two main forms of pancreatitis, acute and chronic. In acute pancreatitis the pancreas can usually heal itself without any lasting changes to function or structure of the gland.

If the pancreas heals but then inflammation reoccurs intermittently and causes irreversible changes to structure and function then it is known as chronic pancreatitis (1).

Pathophysiology

The exact mechanism is not fully understood however it is believed that the initial events take place within the acinar cells of the pancreas. Injury of the acinar cells leads to an inflammatory reaction localized within the cells. If this inflammation is excessive it can lead to a systemic inflammatory response.

The inflammatory process can cause systemic effects because of the presence of cytokines, such as bradykinins and phospholipase A. These cytokines may cause vasodilation, increase in vascular permeability, pain, and leukocyte accumulation in the vessel walls all leading to inflammation. Fat necrosis may also occur causing hypocalcaemia; and pancreatic B-cell injury leading to hyperglycemia (2).

A marked systemic inflammatory reaction can lead to ‘ distant organ damage and multiple organ dysfunction syndrome (MODS)’ (1). This is the primary cause of morbidity and mortality in acute pancreatitis.

The disease progression can be seen in the following three steps:

1) Local inflammation of the pancreas,

2) A generalized inflammatory response,

3) Multi-organ dysfunction (1)

When there is injury or disruption of the pancreatic acini pancreatic enzymes namely trypsin, chymotrypsin and elastase leak into the pancreatic tissue. These enzymes become activated and initiate autodigestion and lead to acute pancreatitis.

The activated enzymes break down the pancreatic tissue and cell membranes which leads to oedema, and vascular damage which leads to hemorrhage and necrosis.

Some patients who have had a severe attack of pancreatitis who survive through the initial event die following a rather minor insult that would not be life threatening normally. It is said that the two hit hypothesis comes in to play here. The initial excessive systemic inflammatory response primes the immune system so that if another event takes place (a small insult in comparison) for example a chest infection, the immune system is overwhelmed leading to an exaggerated inflammatory response which can lead to death (1).

History and examination

The main presentation of acute pancreatitis is epigastric pain or right upper quadrant pain radiating through to the back. In many patients sitting forward can relieve the pain a little. The patient would usually also complain of nausea, vomiting and fever. It is important to note a history of previous biliary colic and binge alcohol consumption. The patient may also be tachycardic, tachypneic, hypotensive and mildly jaundiced (2).

Abdominal tenderness, distension, guarding, and rigidity are quite common as are diminished or absent bowel sounds. If the inflammation should spread to the lungs then basilar rales may be noted on auscultation of the lung. In severe cases Grey Turner or Cullens sign may also be noted (2).

Aetiology

There are many causes of pancreatitis. The most common causes being habitual chronic alcohol consumption and biliary stones. In western countries including the UK alcohol abuse is the most common cause of acute pancreatitis. A recent study showed that 44% of patients have alcohol as the primary risk factor for acute or chronic pancreatitis (3).

Gall stones can cause pancreatitis as they may become wedged in the pancreatic duct or ampulla of Vater and obstruct the pancreatic duct, leading to release of enzymes into the parenchyma.

Other less common causes include: injury (e. g. post ERCP), drugs (such as NSAIDS, azathioprine), viruses (e. g. mumps), autoimmune conditions (e. g. SLE), hyperlipidaemia, malignancy and Scorpion and snake bites (4).

Investigations to be done if pancreatitis is suspected

1) Serum enzyme levels: Serum amylase in pancreatitis is more than four times the normal value and lipase is twice the normal and this is diagnostic as there is no other source other than the pancreas, but this test is not always available (5).

2) Full blood count, U+E, glucose, CRP: the CRP value is significantly lower in drug-induced acute pancreatitis and a raised bilirubin and serum aminotransferase is suggestive of gall stones. Low serum calcium levels are quite common in acute pancreatitis and hypocalcaemia is also relatively common.

3) Plain erect abdominal x-ray: this is done to exclude other causes of the symptoms such as intestinal obstruction or perforation.

4) Chest x-ray: this can show if there is a rise in one hemidiaphragm, acute respiratory distress syndrome or pleural effusions which can occur in severe cases of acute pancreatitis.

5) CT with contrast enhancement: this can be diagnostic if clinical results were inconclusive. CT may show swelling, fluid collection and change in the density of the gland.

6) Ultrasound: this is useful to see if the pancreas is swollen and if the common bile duct is dilated. It can also detect gallstones (5).

Management

In mild cases management is on a general medical ward. Analgesia is given to relieve the pain, usually with pethidine. Morphine is not usually used as it

can have a spastic effect on the sphincter of Oddi (4). The patient is given intravenous fluids and not allowed to take anything my mouth. If the patient is vomiting severely then a nasogastic tube is considered. Oral fluids and solids can be taken once symptoms have cleared and blood tests are normal. The cause must then be treated, for example if gallstones were the cause then they must be removed.

The severity of pancreatitis is determined by the Glasgow score or Ranson criteria which looks at patient demographics, electrolytes and enzyme levels on admission and 48 hours later (see 1)

Glasgow prognostic score

Ranson's criteria

* Age > 55 years

* WBC > 15 x109/l

* Urea > 16mmol/l

* Glucose > 10mmol/l

* pO2 <8kPa (60mmhg)

* Albumin <32g/l

* Calcium <2mmol/l

* LDH > 600 units/l

* AST/ALT > 200 units

Present on admission:

* Age > 55 years

* WBC > 15 x109/l

* Glucose > 10mmol/l

* LDH > 600 units/l

* SGOT > 250 units/l

Developing during first 48 hours:

* Haematocrit fall 10%

* Urea increase > 8mg/dl

* Serum Ca <8mg/dl

* Arterial O2 saturation <60mmHg

* Base deficit > 4meq/l

* Estimated fluid sequestration > 600ml

In Severe cases the patent is treated in ITU. There is a high chance of multiple organ failure and infected pancreatic necrosis in these patients so if there is evidence to suggest this then intravenous antibiotics should be administered straight away.

The patient should be fed via a nasogastric tube and where there are gallstones present and a high probability of a severe attack early ERCP should be done.

Local Complications

Pancreatic necrosis is likely if the CRP is rising and is confirmed by a CT scan. Infection occurs in 30-70% of cases of necrosis and this trebles the mortality risk. Fluid collections occurs in 30-50% of patients with acute pancreatitis but in most cases resolves spontaneously. Pancreatic abscess, acute pseudocysts and pancreatic ascites can also occur (6).

Systemic complications

These include pulmonary oedema, pleural effusions and ARDS with regards to the respiratory system and hypovolamenia and shock with regards to the cardiovascular system. Other complications include: disseminated intravascular coagulopathy, renal dysfunction, hypocalcaemia, hypomagnesaemia, hyperglycaemia and GI haemorrhage (6).

Summary

Acute pancreatitis is a disease in which there is inflammation of the pancreas. Acute abdominal pain and vomiting are the most common symptoms and increased serum concentrations of the enzymes amylase and lipase can confirm the diagnosis. Injury to the pancreas is mild in 80% of patients who recover well without complications. The rest have a more severe disease and present with local and systemic complications.

Alcohol abuse and gall stones are the two most common causes of acute pancreatitis in adults and treatment of mild pancreatitis is supportive and more serious disease needs intervention from quite a few members of the multidisciplinary team (5).

Improving the understanding of the pathophysiology and better investigation of the disease severity should improve the management and outcome of this compound disease (5).

References

1. Bhatia M, Wong FL, Cao Y, Lau HY, Huang J, Puneet P, Chevali L. Pathophysiology of acute pancreatitis. Pancreatology [online]. 2005; 5(2-3): 132-44. [cited 2009 Dec 4] Available from: URL: http://www. ncbi. nlm. nih. gov/pubmed/15849484
2. Ghattas K, Samer S Deeba. Pancreatitis. E medicine [online]. 2009; [cited 2009 Nov 21]. Avalable from: URL: http://emedicine. medscape. com/article/775867-overview
3. Whitcomb DC, Yadav D, Adam S, Hawes RH, Brand RE, Anderson MA, et al. Multicenter approach to recurrent acute and chronic pancreatitis in the United States: the North American Pancreatitis Study 2 (NAPS2). Pancreatology [online]. 2008; 8(4-5): 520-31. [cited 2009 Dec 1] Available from: URL: http://www. medscape. com/medline/abstract/18765957
4. Longmore M, Wilkinson I, Turmezei T, Cheung CK. Oxford Handbook of Clinical Medicine (Oxford Handbooks Series) 6th edition. Oxford university press; 2008
5. Frossard JL, Steer ML, Pastor CM. Acute pancreatitis. The Lancet [online]. 2008; 12; 371(9607): 143-52. [cited 2009 Nov 23] available from: URL: http://www. thelancet. com/journals/lancet/article/PIIS0140-6736(08)60107-5/abstract

6. Willacy H, Kavanagh S. Acute Pancreatitis. Patient Plus UK [online]. 2008. [cited2009 Nov 29] Available from: URL: http://www. patient. co. uk/doctor/Acute-Pancreatitis. htm