Acute Pancreatitis

Pancreatitis is a common nonbacterial inflammatory disease caused by activation, interstitial liberation, and autodigestion of the pancreas by its own enzymes.

Acute pancreatitis:

- There is sudden upper abdominal pain, nausea and vomiting, and elevated serum amylase

Chronis pancreatitis:

- Characterized by chronic pain, pancreatic calcification on x-ray, and exocrine (steatorrhea) or endocrine (diabetes mellitus) insufficiency
- Attacks of acute pancreatitis often occur in patients with chronic pancreatitis

Acute relapsing pancreatitis:

- Multiple attacks of pancreatitis without permanent pancreatic scarring, a picture most often associated with biliary pancreatitis

Chronic relapsing pancreatitis:

- Recurrent attacks superimposed on chronic pancreatitis. Alcoholic pancreatitis often behaves in this way

Etiology

- Gallstones and alcohol account for 80% cases of acute pancreatitis
- Gallstones less than 5mm diameter are more likely to cause pancreatitis than larger ones
- Less than 5% patients with gallstones develop pancreatitis
- 20% cases are idiopathic

Etiological factors can be summarised as follows:

- Idiopathic (15% of patients)
- Obstruction
- Choledocolithiasis

1. About 40% of cases of pancreatitis are associated with gallstone disease, which if untreated usually gives rise to additional acute attacks
2. Eradication of the biliary disease nearly always prevents recurrent pancreatitis

Mechanism:

- Transient obstruction of the ampulla of Vater and the pancreatic duct by the gallstones. However, only 25% of the time the stones are found.
- Thus, it is assumed that most attacks are caused by a gallstone or sludge traversing the common duct and ampulla of Vater (because 90% of patients excrete a gallstone in feces within 10 days after an acute attack)
- Ampullary or pancreatic tumours

- Pancreatic structural anomalies

Pancreas divisum – combine with further narrowing of the opening of the minor papilla occurs obstructive pancreatitis

- Toxins
- Alcohol
- Characteristically the patients have been heavy users of hard liquor or wine. Alcoholic pancreatitis is often considered to be synonymous with chronic pancreatitis no matter what the clinical findings are; because most commonly at least 6 years of alcoholic excess precede the initial attack.
- Acetaldehyde has been implicated as a mediator, since it can generate toxic oxygen metabolites under the influence of xanthine oxidase.
- Drugs - salicylates, azathioprine, cimetidine
- Trauma
- Accidental

- Iatrogenic

E.g. after common bile duct exploration, especially if sphincterectomy was performed

- Metabolic abnormalities

1. Hypercalcemia
2. Increased calcium concentrations in pancreatic juice that result from hypercalcemia may prematurely activate proteases
3. They may also facilitate precipitation of calculi in the ducts
4. Hyperlipidemia
5. In some patients – especially alcoholics – hyperlipidemia appears transiently during an acute attack
6. In those with primary hyperlipidemia – pancreatitis seems to be a direct consequence of the metabolic abnormality
7. Hyperlipidemia during an acute attack is usually associated with normal serum amylase levels, because the lipid interferes with the chemical determination for amylase; urinary output of amylase may still be high
8. One should inspect the serum of every patient with acute abdominal pain, because if it is lactescent, pancreatitis will almost

always be the correct diagnosis

- Infection
- Vascular anomalies

Epidemiology

- The incidence of acute pancreatitis is about 50 per 100,000 population per year
- 80% have mild disease
- 40% of those with severe disease develop infected pancreatic necrosis
- The mortality associated with infected necrosis is about 40%
- 50% of deaths occur within first week due to multi-organ failure
- This usually occurs in the absence of local complications

Clinical features

- Sudden onset epigastric pain
- Constant in nature and radiates through to back
- Patients are often pyrexial and dehydrated
- Tenderness may be localised to epigastrium or generalised

Differential diagnosis includes

- Perforated peptic ulcer
- Acute cholecystitis
- Mesenteric ischaemia

Eponymous signs of retroperitoneal haemorrhage are rare and appear late include

- Cullen's sign – bluish discoloration in periumbilical area
- Grey Turner's sign – bluish discoloration in the flank

Diagnosis

- Serum amylase has low sensitivity and specificity – detect pancreatic amylase, salivary, and macroamylase
- A serum amylase of 3x upper limit of normal has sensitivity = 60% and specificity = 95%
- 20% cases of pancreatitis have normal serum amylase (particularly alcoholic aetiology)
- Serum lipase more sensitive and may remain elevated longer
- A serum lipase of 4x upper limit of normal has higher sensitivity and good specificity
- Features suggestive of a gallstone aetiology include:

1. Female sex
2. Age more than 50 years
3. Amylases > 4000 IU/L
4. Bilirubin > 25 umol/L
5. AST > 100 IU/L
6. ALP > 300 IU/L

Other causes of hyperamylasaemia

- Perforated peptic ulcer
- Cholecystitis
- Generalised peritonitis
- Intestinal obstruction
- Mesenteric infarction
- Ruptured AAA
- Ruptured ectopic pregnancy
- Urine amylase is also increased and is of diagnostic value
- Increased owing to a decrease in tubular reabsorption of amylase

- Hypocalcemia (in severe pancreatitis) – because calcium being complexed with fatty acids (liberated from retroperitoneal fat by lipase) and impaired reabsorption from bone owing to the action of calcitonin (liberated by high levels of glucagon)

Imaging studies:

- In two-thirds of cases, a plain abdominal film is abnormal

1. Sentinel loop
2. Isolated dilation of a segment of gut consisting of jejunum, transverse colon, or duodenum adjacent to the pancreas
3. Colon cutoff sign
4. Gas distending the right colon that abruptly stops in the mid or left transverse colon; due to colonic spasm adjacent to the pancreatic inflammation
5. Glandular calcification – chronic pancreatitis

- Chest films – pleural effusion on the left side
- CT – pancreatic phlegmon, necrosis, pseudocyst, abscess
- Ultrasound – may demonstrate gallstones
- ERCP – after the pancreatitis has subsided

Contrast-enhanced CT scoring system

Grade Criteria

1. A = Normal
2. B =

- Focal or diffuse glandular enlargement
- Small intra-pancreatic fluid collection

1. C =

- Any of the above
- Peripancreatic inflammatory changes
- Less than 25% gland necrosis

1. D =

- Any of the above
- Single extrapancreatic fluid collection
- 25-50% gland necrosis

1. E =

- Any of the above
- Extensive extrapancreatic fluid collection
- Pancreatic abscess
- More than 50% gland necrosis

Prognostic factors

- 80% of patients have mild pancreatitis with good recovery
- Mild disease accounts for less than 5% of the mortality form pancreatitis
- Mortality from pancreatitis due to:

1. Early multiple organ failure
2. Late infected pancreatic necrosis
3. Haemorrhage
4. Associated co-morbidity

- Aim of prognostic scores is to identify patients with severe pancreatitis
- Need to have high sensitivity and specificity
- Ideally should be applicable on admission
- There are some scoring system/criteria (e.g. Ranson’s criteria, APACHE II score)

Ranson's criteria

- On admission

1. Age > 55 yrs
2. WCC > 16,000
3. LDH > 600 U/l
4. AST >120 U/l
5. Glucose > 10 mmol/l

- Within 48 hours

1. Haematocrit fall >10%
2. Urea rise >0.9 mmol/l
3. Calcium < 2 mmol
4. pO2 < 60 mmHg
5. Base deficit > 4
6. Fluid sequestration > 6L

- Can not be applied fully for 48 hours
- Also poor predictor later in the disease
- 'Single snapshot in a whole feature length film'

Complications of acute pancreatitis

Local

- Necrosis +/- infection
- Pancreatic fluid collections, pseudocyst, abscess
- Colonic necrosis
- Gastrointestinal haemorrhage
- Splenic rupture

Systemic

- Hypovolaemia and shock
- Coagulopathy
- Respiratory failure
- Renal Failure
- Hyperglycaemia
- Hypocalcaemia

Medical treatment

Aims:

- To halt progression of local disease
- Prevent remote organ failure

1. Requires full supportive therapy
2. Urinary catheter, CVP line and possibly arterial line
3. Regular assessment of U+Es, Ca, blood sugar, LFTs

- Patients require:

1. Fluid resuscitation with both colloid and crystalloid
2. Correction of hypoxia with an increased inspired oxygen or ventilation
3. Adequate analgesia - opiate or epidural

- Increasing evidence that antibiotic prophylaxis useful in severe pancreatitis
- ERCP maybe of benefit within the first 48 hours in patients with predicted severe disease
- Peritoneal lavage – infusing and withdrawing 1-2 L of lactated Ringer's solution through a peritoneal dialysis catheter every hour for 1-3 days

Nutritional support

- Pancreatitis is associated with a catabolic state
- The benefit of pancreatic 'rest' by limiting oral intake is unproven
- Evidence that early enteral nutrition is safe
- Nasojejunal feeding limits pancreatic secretion
- Preferable to oral or nasogastric feeding

Endoscopic Spincterectomy

- Biliary pancreatitis is caused by a gallstone becoming lodged in the ampulla of Vater.
- In severe cases, endoscopic sphincterectomy performed within 72 hours of the onset of the disease has been shown to decrease the incident of concomitant biliary sepsis and lower the mortality rate from the pancreatitis

Surgical Treatment

- Is generally contraindicated in uncomplicated acute pancreatitis
- When laparotomy has been performed for diagnosis and mild to moderate pancreatitis is found, cholecystectomy and operative cholangiography should be performed if gallstones are present, but the pancreas should be left undisturbed
- Debridement of dead peripancreatic tissue, which is often colonized by bacteria, reduces the mortality rate
- At surgery, all peripancreatic spaces are opened and any necrotic tissue is removed by gentle blunt dissection
- A T-tube is inserted if there is bile duct obstruction, and cholecystectomy is performed for gallstone disease
- Two large drains are placed within the debrided spaces and are used postoperatively for sterile lavage
- About 8L of fluid are infused through this system daily for an average of 2 weeks