Acute and Chronic pancreatitis, Splenectomy

MO teaching slides
2nd August 2016
Acute Pancreatitis

- **Diagnosis**
  - At least 2 out of 3 criteria
    - Abdominal pain (cardinal symptom) – constant, epigastric, radiating to the back
    - Raised serum amylase/lipase levels 3x ULN
    - Radiological evidence of pancreatitis on CT scan with contrast/MRI pancreas

- **Etiology**
  - “I GET SMASHED”, Idiopathic – most common
  - Hx of biliary colic, alcohol intake/dependence, trauma, drugs- steroids, ERCP, high TG, Ca, PTH

To note:
- If gallstone pancreatitis, pain can be in RUQ. Others- poorly localised
- Painless pancreatitis presenting with hypotension e.g critically ill, post-operatively
- 90% have nausea and vomiting that can last for several hours
- Imaging is discouraged esp within initial 48hrs, unless there is clinical doubt of the diagnosis.
- US abdomen – often done TRO gallstones
- Serum amylase not as sensitive and specific as lipase. Rises early and normalises by 3-5 days
- MRI pancreas more sensitive than CT scan to characterise solid vs fluid
Examination

- Vitals: Fever / Tachycardia / Hypotension
- Dyspnoeic, respiratory distress, jaundice
- Abdominal pain, guarding
- Abdominal distension, diminished bowel sounds from ileus
- Cullen and Grey Turner sign - severe necrotizing pancreatitis

Investigations

- FBC, LFTs, RP, Calcium and Albumin, LDH, Fasting lipids, ABG
- ANA, IgG4 if suspect autoimmune pancreatitis
- Imaging: US abdo, EUS (if cause is unclear: pancreatic ductal abn, small tumors at/near ampulla, microlithiasis in GB/bile duct, early chronic pancreatitis)
- CT/MRCP – if suspect malignancy from hx ( >40y, LOW, new onset DM)
- CXR
Predictors of severity

Scoring systems - none proven to be accurate in predicting severity. However, they are superior to clinical judgement for triaging pts to ICU and aggressive therapy

- Ranson's criteria - done at 0h and 48h
  - Score 3- mortality 0-3%, score >3 11-15% mortality, score > 6 – 40% mortality

- APACHE II score - 12 measures. APACHE O (inc BMI improves predictability of severe AP)
  - Score < 8 – mortality < 4%, Score > 8 mortality 11-18%

- Glasgow score - less sensitive than Ranson’s (73% vs 91%), similar specificity (71% vs 74%)

- CT severity Index (Balthazar)
  - Based upon the degree of necrosis, inflammation, and the presence of fluid collections
  - Detection of pancreatic necrosis does not necessarily predict organ failure but it does alter treatment approach
  - Internal validation study showed any degree of pancreatic necrosis was a/w mortality of 23%. Strong association with morbidity/mortality if > 30% necrosis.
  - Score > 6 = severe disease
Predicting severity – American College of Gastroenterology guidelines

■ Patient characteristics
  - Age >55 years
  - Obesity (body mass index >30 kg/m²)
  - Altered mental status
  - Comorbid disease

■ SIRS syndrome (* early / persistent>72h)
  - Presence of >2 of the following criteria:
    - Pulse >90 beats/min
    - Respiration >20/min or PaCO₂ >32 mm Hg
    - Temperature >38 or <36°C
    - White blood cell count >12,000 or <4,000 cells/mm³ or >10 percent immature neutrophils (bands)

■ Laboratory findings
  - Blood urea nitrogen (BUN) >20 mg/dl
  - Rising BUN
  - Haematocrit (HCT) >44 %
  - Rising HCT
  - Elevated creatinine

■ Radiology findings
  - Pleural effusions
  - Pulmonary infiltrates
  - Multiple or extensive extrapancreatic collections

* CRP > 150 at 48hrs is predictive of severe pancreatitis
Clinical outcomes

- 85% of patients with acute pancreatitis have mild disease
- 15% have necrotising pancreatitis with necrosis of pancreatic parenchyma or peripancreatic tissue
- Most patients have mild disease and recover over 3-5 days
- 20% will develop moderately severe to severe pancreatitis with either local or systemic complications or organ failure
- Overall mortality is 5%. With interstitial pancreatitis (3%) having a lower mortality than necrotising pancreatitis (17%)
Revised Atlanta Classification (2012)

Acute Pancreatitis

**Two phases**
- Early: 1st week
- Late: After 1st week

**Severity**
- Mild: No organ failure
- Moderate: Organ failure less than 48 h
- Severe: Organ failure longer than 48 h

**Two types**
- Oedematous: Complications
  - < 4 wk: acute peripancreatic collection
  - > 4 wk: pseudocyst
- Necrotizing: Complications
  - < 4 wk: acute necrotic collection
  - > 4 wk: walled-off necrosis
Revised Atlanta Classification (2012)

Acute Pancreatitis - Fluid Collections

- Interstitial Pancreatitis
  - < 4 weeks
    - Acute Peripancreatic Collection
      - > 4 weeks
        - Pseudocyst
  - > 4 weeks
    - Walled off Necrosis
- Necrotizing Pancreatitis
  - < 4 weeks
    - Acute Necrotic Collection

Acute Peripancreatic Collection
- < 4 weeks
- In interstitial pancreatitis
- Homogeneous - fluid density
- No fully definable wall
- Adjacent to pancreas
- Confined by normal fascial planes

Acute Necrotic Collection
- < 4 weeks
- In necrotizing pancreatitis
- Heterogeneous collection
- No fully definable wall
- Intra- or extrapancreatic

Pseudocyst
- > 4 weeks
- In interstitial pancreatitis
- Homogeneous - fluid density
- Well defined wall
- Adjacent to pancreas
- No non-liquid component

Walled-off Necrosis
- > 4 weeks
- In necrotizing pancreatitis
- Heterogeneous collection
- Well defined wall
- Intra- or extrapancreatic
Management

- **Supportive treatment**
  - **Fluid resuscitation**
    - Critical for aggressive fluid replacement in initial 48hrs
    - Adjust according to Clinical assessment, BUN, Creatinine
    - Lactated Ringer’s solution reduces SIRS / CRP cpd to Normal saline

- **Pain control**
  - Opioids
  - Fluid resuscitation (ischaemia)

- **Active monitoring** – vitals, urine output, electrolytes, hypocount – if raised, increases risk of infection

- **Nutrition**
  - Mild pancreatitis – IV hydration, resume oral diet (low fat) within 1 week if pain improving, no ileus, N/V
  - Moderate-severe – initiate within 48hrs
    - Enteral feeding preferred to parenteral: maintain the intestinal barrier and prevents bacterial translocation from the gut
    - High protein, low fat, semi-elemental feeding formulas (eg, Peptamen AF) because of a reduction in pancreatic digestive enzymes

- Prophylactic antibiotics are not recommended regardless of type or disease severity

- If clinical deterioration noted after 72hrs – CT with IV contrast indicated to assess for local complications and pancreatic necrosis
Management of complications

- **Acute pancreatic fluid collection** – early phase. Usually asymptomatic, resolves spontaneously w/i 7-10 days without need for drainage.

- **Walled-off pancreatic necrosis/ Pancreatic pseudocyst** (after 4 wks)
  - Mnx depends on sx, characteristics and location of fluid collection
  - Expectant management for those w/o cystic neoplasm, pseudoaneurysm or minimal symptoms
    - Repeat CT scan every 3-6 mths until cyst resolves/stabilizes at a small size
    - If pseudoaneurysm present but asymptomatic – embolization of aneurysm followed by expectant management

- IF drainage required, 3 main options:
  - **Endoscopic drainage**
    - Contraindicated if pseudoaneurysm present
    - Good for relatively small pseudocysts in communication with the main pancreatic duct for transpapillary stent placement
    - For larger symptomatic cysts abutting the stomach, duodenum, transmural puncture to relieve symptoms possible
    - 10 to 15% morbidity, 70 to 80% fluid collection resolution, 10-15% recurrence rate.

  - **Surgical drainage**
    - Can be open or lap. Mainly used in cases of endoscopic failures, recurrence of collection
    - Cystogastrostomy or a cystojejunostomy depending on location
    - Open approach a/w substantial morbidity and mortality (25%, 5% respectively)

  - **Percutaneous drainage**
    - As effective as surgery in draining and resolving sterile and infected walled-off pancreatic fluid collections
    - Stent diameter may need to be increased with time for necrotic debris to drain. Frequent irrigation required to maintain patency
    - Risk of infection, pancreaticocutaneous fistula formation
Indications for intervention in absence of signs of infection

- Ongoing GOO, biliary obstruction due to mass effect 4-8 wks after onset
- Persistent symptoms of abdo pain, N/V/anorexia/LOW >8 wks after onset
- Disconnected duct syndrome with persistent sx of pain and obstruction >8 wks after onset

Signs of systemic toxicity: fever, leukocytosis, hypotension

- Delay necrosectomy by 4 wks to allow minimally invasive debridement

-Occurs in 1/3
-Onset >10d
-Commonly due to gut microbes: E.coli, Pseudomonas, Klebsiella, Enterococcus
Management of Gallstone pancreatitis

- Gallstone pancreatitis
  - **ERCP**
    - ERCP shld be performed early (w/i 24hrs) for patients with gallstone pancreatitis and cholangitis
    - ERCP also indicated if imaging shows CBD obstruction with visible stone, dilated CBD or increasing LFTs in absence of cholangitis.
    - Evidence for URGENT ERCP is controversial- *early ERCP reduced pancreatitis-related complications but not mortality in patients predicted to have severe pancreatitis*
    - ERCP NOT indicated even in severe gallstone pancreatitis WITHOUT cholangitis

- **Cholecystectomy**
  - Can be done within 7 days of index hospitalization in mild pancreatitis
  - In severe necrotizing pancreatitis, cholecystectomy shld be delayed
  - Risk of recurrence acute pancreatitis
  - Cholecystectomy shld also be offered to patients with acute pancreatitis and biliary sludge
Chronic Pancreatitis

■ **Definition:**
  - Syndrome involving progressive inflammatory changes in the pancreas that result in permanent structural damage which can lead to impairment of exocrine or endocrine function

■ **Distinguishing features:**
  - Asymptomatic over long period of time
  - Present with a fibrotic mass with symptoms of pancreatitic insufficiency without pain.
  - Normal levels of amylase and lipase

■ **Morphologically:**
  - Patchy focal disease characterised by mononuclear infiltrate and fibrosis vs diffusely large portion of pancreas involved with a neutrophilic inflammatory response in acute pancreatitis

■ **Pancreatic insufficiency** ( >90% of pancreatic function is lost)
  - Fat malabsorption – steatorrhoea prior to protein deficiency. Malabsorption of fat soluble vitamins
  - Glucose intolerance occurs in chronic pancreatitis, overt diabetes mellitus occurs late in disease

■ **Classic triad:** steatorrhoea, diabetes mellitus, pancreatic calcifications – late advance stage disease

■ Can develop acute pancreatitis with sudden worsening or change in pattern of symptoms

■ Watch for possibility of a pancreatic carcinoma. Increased risk in pts with chronic pancreatitis

■ **Investigations**
  - 72hr faecal fat test ( >7g/day fat diagnostic of malabsorption)
  - Plain XR – showing calcium deposition – common in alcoholic pancreatitis
  - US low sensitivity (70%), specificity (90%) vs CT 80% spec 85%
  - MRCP becoming diagnostic test of choice in view of demonstrating calcifications and pancreatic duct obstruction consistent with chronic pancreatitis
  - ERCP – now limited for therapeutic. Characteristic beading of main pancreatic duct and ectatic side branches is diagnostic of chronic pancreatitis
Chronic Pancreatitis

- After establishing a secure diagnosis of pancreatitis (i.e. ruling out other causes of symptoms e.g. PUD, biliary obstruction, pseudocysts, pancreatic carcinoma)

- Management
  - Treat underlying cause e.g. cessation of alcohol intake
  - Small portion, low fat meals (anecdotal evidence)
  - Smoking cessation to reduce risk of Pancreatic CA
  - Pain management – some may need chronic Opioids
  - Pancreatic enzyme (Creons, Vitamin ABDEK)

- Surgical options:
  - A meta-analysis of four trials concluded that duodenum-preserving pancreatic head resection was as effective as pancreatoduodenectomy for relief of pain, overall morbidity and postoperative endocrine insufficiency and was superior in some postoperative outcomes and quality of life
  - Total pancreatectomy is a treatment option in carefully selected patients with chronic pancreatitis in whom substance abuse is not a confounding factor
Complications of acute pancreatitis

- **Splanchnic venous thrombosis**
  - Involves splenic, portal, SMV
  - Effective treatment of underlying pancreatitis usually leads to spontaneous resolution
  - Anti-coagulation shld be initiated if there is extension of clot into portal or SMV causing hepatic decompensation or reduced bowel perfusion

- **Pseudo aneurysms**
  - Rare but serious complication of acute pancreatitis
  - High index of suspicion for those with acute pancreatitis and unexplained GI bleeding

- **Abdominal compartment syndrome**
  - Increased risk due to aggressive fluid resuscitation, peripancreatic inflammation, ascites and ileus
Splenectomy – Indications

- Traumatic Splenic injury

- Non-traumatic / Medical
  - Indications for splenectomy:
    - ITP (most common)
    - Hereditary spherocytosis
    - Thalassaemia
    - Sickle Cell anaemia
    - Hodgkin disease
    - Felty syndrome (RA, splenomegaly, neutropaenia)
Splenic injury

- Often occurs in blunt trauma due to MVA, falls
- Patient presents with trauma and is assessed with ATLS protocols
- FAST scan – hypoechoic rim of subcapsular fluid or intraperitoneal fluid around the spleen or in Morrison’s pouch (hepatorenal space)
- CT scan with IV contrast – arterial phase will show a blush/ extravasation, hypodensity can represent hematoma, hemoperitoneum by comparing Hounsfield units to differentiate from ascites
The shortcomings of this grading scale are:

• Underestimation of injury extent
• Significant inter-observer variability.
• Does not include:
  ◦ Active bleeding
  ◦ Contusion
  ◦ Post-traumatic infarcts
• Most importantly: no predictive value for non-operative management (NOM).
Management of traumatic splenic injuries

- **Non-operative management**
  - Hemodynamically stable
  - Low grade splenic injury (I-III)
  - Without evidence of intra-abdominal injury

- No evidence of active contrast extravasation/ blush on CT
  - Even those who require extra-abdominal injuries e.g fracture stabilization
  - Contraindications: hemodynamic instability, generalized peritonitis, other intra-abdominal injuries requiring surgical exploration. Portal hypertension – relative contraindication

- **Angio-embolisation**
  - Indications: Active contrast extravasation on CT, intra-parenchymal pseudoaneurysm formation
  - Variable success rates – institution dependent
  - Relative contraindication: Grade IV/V injuries due to vascular disruption, age >55yrs due to splenic capsule thinning out and associated with higher failure rates
  - If contrast extravasation is from splenic parenchyma supplied by short gastric vessels – then operative intervention prompted as they are less amenable to embolization

- **Surgical intervention**
  - Indication: hemodynamically unstable, unable to tolerate significant hypotension, failure of non-surgical management
  - Splenic salvage:
    - Splenorrhaphy refers to the suture repair of the spleen with or without splenic wrapping, and is generally supplemented by electrocautery techniques for control of parenchymal haemorrhage
    - Partial splenectomy is a form of splenic salvage and refers to the removal of a portion of the spleen based upon its segmental blood supply.
  - Total splenectomy is the safest option and is often done in emergency setting where the risk of blood loss outweighs the risk of OPSI
  - Replantation of splenic tissue — If splenectomy for injury is deemed necessary, heterotopic autotransplantation of splenic tissue into omental pockets may provide some splenic function, although this has not been proven conclusively
Post-splenectomy related issues

■ Activities
  - Restricted activities and avoidance of high risk activities for up to 3 months
  - No clinical studies to support duration

■ Vaccinations
  - Should be given 14 days before elective surgery
  - Given on POD 14 for emergency surgeries
  - Inactivated Influenza vaccines - NOT live attenuated vaccines

■ Antibiotics
  - In Children, daily penicillin (amoxicillin) up to 5yrs and 1 yr after splenectomy
  - In Adults – no indication for daily antibiotics unless in hypogammaglobulinemia, HIV infection, solid organ transplant recipients, and patients with advanced liver disease
  - However, if febrile, early administration of antibiotics reduces risk of severe infection
Thank you!
# Ranson criteria to predict severity of acute pancreatitis

<table>
<thead>
<tr>
<th>0 hours</th>
<th>48 hours</th>
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<tbody>
<tr>
<td><strong>Age</strong></td>
<td><strong>Hematocrit</strong></td>
</tr>
<tr>
<td>&gt;55</td>
<td>Fall by ≥10 percent</td>
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<tr>
<td><strong>White blood cell count</strong></td>
<td><strong>Blood urea nitrogen</strong></td>
</tr>
<tr>
<td>&gt;16,000/mm³</td>
<td>Increase by ≥5 mg/dL (1.8 mmol/L)</td>
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<tr>
<td><strong>Blood glucose</strong></td>
<td>despite fluids</td>
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<tr>
<td>&gt;200 mg/dL (11.1 mmol/L)</td>
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<tr>
<td><strong>Lactate dehydrogenase</strong></td>
<td><strong>Serum calcium</strong></td>
</tr>
<tr>
<td>&gt;350 U/L</td>
<td>&lt;8 mg/dL (2 mmol/L)</td>
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<tr>
<td><strong>Aspartate aminotransferase (AST)</strong></td>
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<tr>
<td>&gt;250 U/L</td>
<td><strong>pO₂</strong></td>
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<tr>
<td></td>
<td>&lt;60 mmHg</td>
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<tr>
<td></td>
<td><strong>Base deficit</strong></td>
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<td></td>
<td>&gt;4 MEq/L</td>
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<td></td>
<td><strong>Fluid sequestration</strong></td>
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<td>&gt;6000 mL</td>
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The presence of 1 to 3 criteria represents mild pancreatitis; the mortality rate rises significantly with four or more criteria.

## Assessing the severity of acute pancreatitis

Glasgow prognostic score: (NOTE PANCREAS ACRONYM)
- PaO₂ < 8kPa (60mmhg)
- Age > 55 years
- Neutrophils: (WBC >15 x 10⁹/l)
- Calcium < 2mmol/l
- Renal function: (Urea > 16mmol/l)
- Enzymes: (AST/ALT > 200 iu/L or LDH > 600 iu/L)
- Albumin < 32g/l
- Sugar: (Glucose >10mmol/L)

Any 3 factors means acute severe pancreatitis.

A score >= 3 indicates Acute Severe Pancreatitis
A score = 2 indicates Acute Moderate Pancreatitis
A score < 2 indicates Acute Mild Pancreatitis