Acute Pancreatitis

Speaker:
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*Pancreatitis is primarily due to the intracellular activation of trypsinogen to trypsin by numerous stimuli which, as yet have not been fully elucidated.*

*Acute:* acute condition presenting with abdominal pain and raised pancreatic enzyme levels in the blood or urine

Acute pancreatitis may recur

*Chronic:* a continuing inflammatory disease of the pancreas characterized by irreversible morphological change typically causing pain and/or permanent loss of function
Acute pancreatitis

-The disease may occur at any age, with a peak in the young male and the older female.

*Mortality rate* = 4-6%, Which increase to (15-40%) in severe cases.

-80% of patients will have a mild attack of pancreatitis.
Etiology

* 50-70% biliary calculus pancreatitis
* 25% Alcohol pancreatitis
* 30% idiopathic

Edematous/ hemorrhagic/ necrosis
Factors associated with acute pancreatitis. ERCP = endoscopic retrograde cholangiopancreatography.
Pathophysiology

1- Gallstone migration into the common bile duct is the main cause of acute pancreatitis

2- Pancreatic hyperstimulation and bile-pancreatic duct obstruction which increase duct pressure and active trypsin reflux

3- Microlithiasis for small concretions in gallbladder or biliary tree that less than 3 mm

4- Biliary sludge and microlithiasis

5- Hyperlipidemia causing 7% of all pancreatitis
6-Ischemic theory:
Hyperchylomicronemia cause sluggish flow in capillaries in the pancreatic bed causing ischemia

*Microcirculatory disturbance play a pivotal role in pathogenesis

*Pancreatic ischemia due to hypo perfusion may also be the critical factor which causes the progression from oedema to necrosis

*Microcirculation is lower in obese patients

*Fat tissue are likely to contribute to increase inflammation in obese rodents.
Clinical Presentation

*No path gnomonic symptoms and signs.
*Pain-develops quickly, severe, agonising, refractory to analgesia, constant in nature, squeezing, boaring and constricted in nature, started epigastric, localized at RUQ-LUQ or felt diffusely in the abdomen

*50% have radiation to back

*Relieving by leaning forward or sitting
* Associated with nausea, frequent vomiting and retching, hiccoughs
Acute pancreatitis should be suspected in the patient who:

1-Develops marked abdominal pain, fever or unexplained shock following abdominal surgery

2-Presents with diabetic coma and shock

3-has clinical features suggesting myocardial infarction with abdominal distension
On examination

-Tachypnea, tachycardia and hypotension
-SIRS (Systemic inflammatory response syndrome)

* Fever of more than 38°C or less than 36°C
* Heart rate of more than 90 beats per minute
* Respiratory rate of more than 20 breaths per minute or a PaCO2 level of less than 32 mm Hg
* Abnormal white blood cell count (>12,000/µL or < 4,000/µL or >10% bands)
Grey turner sign
Cullen’s sign
- Bleeding into fascial planes can produce bluish discoloration of flanks (Grey Turner sign)

- Umbilicus (Cullen’s sign)

- Abdominal examination: distension, ascites with shifting dullness

- A mass in the epigastrium due to inflammation.
- Muscle guarding in the upper abdomen

- 10-20% develop pleural effusion
Investigations

- Serum amylase (x4)
- Plain abdomen x-ray: (not diagnostic)
  Generalized or localized ileus (sentinel loop)
  a colon ‘cut-off’ sign
  a renal ‘halo sign’
  calcified gallstones and pancreatic calcifications
- CXR:
  Pleural effusion
  Alveolar intersitial odema (ARDS)
*U/S:
The pancreas is poorly visualized
-Can detect free peritoneal fluid, gallstones, dilatation of the common bile duct

*C-T Scan with contrast*
RADIOLOGICAL SCORING OF ACUTE PANCREATITIS

CT CALCULATED SEVERITY INDEX

I: CT Acute Pancreatitis Grading

A.....Normal pancreas

.................................................................0

B.....Enlarged pancreas......................................................1

C.....Inflamed pancreas with or peri-pancreatic fat inflammation ......2

D.....Single peri-pancreatic fluid collection...............................3

E.....Two or more peri-pancreatic collections.........................4
Grading Of Acute Pancreatitis Necrosis

Necrosis percentage Additional points
0%.............................................................0

30-50%......................................................4

>50%...........................................................6

*CT severity index is calculated by adding CT grade points to points assigned for percentage of necrosis (from 0 to 10.)

\[
CT \ SEVERITY \ INDEX = \text{____ + ____ = ____ (from 0-10)}
\]
**Ranson’s Score**

<table>
<thead>
<tr>
<th>Criterion†</th>
<th>All Causes</th>
<th>Biliary Origin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>On Hospital Admission</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>&gt;55</td>
<td>&gt;70</td>
</tr>
<tr>
<td>White blood cell count, × 10⁹/L</td>
<td>&gt;16</td>
<td>&gt;18</td>
</tr>
<tr>
<td>Serum glucose level, mmol/L (mg/dL)</td>
<td>&gt;11.1 (&gt;200)</td>
<td>&gt;11.1 (&gt;220)</td>
</tr>
<tr>
<td>Serum LDH level, U/L</td>
<td>&gt;350</td>
<td>&gt;400</td>
</tr>
<tr>
<td>Serum AST level, U/L</td>
<td>&gt;250</td>
<td>&gt;250</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Within 48 Hours of Hospital Admission</strong></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Hematocrit decrease</td>
<td>&gt;0.1</td>
<td>&gt;0.1</td>
</tr>
<tr>
<td>BUN level increase, mmol/L (mg/dL)</td>
<td>&gt;1.8 (&gt;5)</td>
<td>&gt;0.7 (&gt;2)</td>
</tr>
<tr>
<td>Serum calcium level, mmol/L (mg/dL)</td>
<td>&lt;2 (&lt;8)</td>
<td>&lt;2 (&lt;8)</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>&lt;60</td>
<td></td>
</tr>
<tr>
<td>Base deficit, mmol/L</td>
<td>&gt;4</td>
<td>&gt;5</td>
</tr>
<tr>
<td>Estimated fluid sequestration, L</td>
<td>&gt;6</td>
<td>&gt;4</td>
</tr>
</tbody>
</table>

*Zero to 2 criteria met indicates mild pancreatitis; 3 or more criteria, severe pancreatitis.

† LDH indicates lactate dehydrogenase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; and ellipsis, not applicable.
Management

* Ranson’s score
* Glasgow score
* Imri’e score
* APACHE 2: a score of 9 or more indicates a severe attack
* CRP
Conservative treatment

* Based on the severity
* Nil per mouth
* I.V fluids
* Strong analgesia-opioids
* Monitoring (ABGs)
* Ensure homeostasis of respiratory-C.V.S and Renal system
* Antibiotics are not indicated unless there is evidence of infection (Imipenem)
In case of cholangitis----→ E.R.C.P
1 Superior sphincter
2 Inferior (submucosal) sphincter
3 Sphincter ampullae
4 Pancreatic sphincter
Complications

1-General:

It needs a multidisciplinary team.
Once the presence of infected necrosis, appropriate debridement (Necrectomy) may be performed.

C.V.S
RESPIRATORY
RENAL
HAEMATOLOGICAL
METABOLIC
GIT
NEUROLOGY
2-Local:

- **Acute fluid collection**: located in or near the pancreas, the wall encompassing the collection is ill defined.
- **Acute pseudocyst**: It needs 4-6 weeks from onset of a cute pancreatitis.
- **Pancreatic necrosis**: A diffuse or focal area of dead parenchyma which is typically associated with peri-pancreatic fat necrosis.
- **Pancreatic abscess**:
- **Pancreatic effusion**
- **Pancreatic ascites**: Chronic generalized peritoneal enzyme-rich effusion usually associated with pancreatic duct disruption.
- **Psuedoaneurysm**:
- **Mesenteric venous thrombosis**
- **Portal venous thrombosis**
- **Hypocalcaemia and electrolyte disturbances**
Abdominal compartment syndrome
Management of local complications

* Conservative Mx
* If fluid collections cause symptoms or impair function, then percutaneous or transgastric drainage is appropriate
* Sterile necrosis should not be drained
* The only indication for surgery is infected necrosis
* Repeated laparatomies performed until there is a clean granulation cavity.
* Nutrition support is mandatory
cystogastrostomy
Necrezeectomy
Prognosis
Chronic pancreatitis

* is a chronic inflammatory disease in which there is irreversible progressive destruction of pancreatic tissue (fibrosis).

* The ducts become distorted and dilated with areas both stricture formation.

* The ducts may become occluded with a gelatinous proteinaceous fluid and debris-deformed cysts

* Repeated attacks lead to ductular metaplasia and atrophy of acini hyperplasia of duct epithelium and interlobular fibrosis
Clinical features

- Pain (dull and gnawing) is the outstanding symptom (epigastric and RUQ) - back pain
- Radiation to the left shoulder
- Nausea and vomiting.
- Weight loss (food fear)
- Abuse analgesia
- Increase the incidence of D.M with time
- Steatorrhoea
Investigations

*The prime investigation is either an MRCP or a C-T scan which will show the outline of the pancreas

*MRCP----→Biliary obstruction
*E.R.C.P
Treatment

*Diet* (low fat-alcohol free-stop smoking)
*Pancreatic enzyme supplementation*
*Nutrition support.*

* Surgery: 
  - Pancreticoduodenectomy (frey- pasteuw).
  - Longitudinal pancreatico-jejunostomy (Frey)
Lateral pancreatrico-jejunostomy
Pancreatico-dudenectomy
Prognosis

* Development of pancreatic cancer is a risk in those with chronic pancreatitis for long time

* Exclude pancreatic cancer
Thank you