CLINICAL MANIFESTATIONS AND DIAGNOSIS OF
ACUTE PANCREATITIS

Raed Abu Sham’a, M.D
ACUTE PANCREATITIS

Acute inflammatory process of the pancreas that resolves both clinically and histologically. It is usually associated with severe acute upper abdominal pain and elevated blood levels of pancreatic enzymes.
ETIOLOGY

- Biliary tract disease
- Alcoholism
- Drugs
- Infection
- Hypertriglyceridemia
- ERCP
- Pancreatic duct abnormalities
- CBD abnormalities
- Scorpion sting
- Surgery
- Vascular disease
- Trauma
- Hyperparathyroidism
- Hypercalcemia
- Renal transplant.
- Hereditary pancreatitis
- Uncertain causes
In biliary tract disease

- Temporary impaction of a gallstone in the sphincter of Oddi before it passes into the duodenum.
- Obstruction of the pancreatic duct in the absence of biliary reflux can produce pancreatitis, suggesting that increased ductal pressure triggers pancreatitis.
Alcohol intake

- Alcohol intake > 100 g/day for several years may cause the protein of pancreatic enzymes to precipitate within small pancreatic ductules.
- In time, protein plugs accumulate, inducing additional histologic abnormalities.
- Because of premature activation of pancreatic enzymes
PATHOLOGY

**EDEMA - NECROSIS - HEMORRHAGE**

- **Tissue necrosis** is caused by activation of pancreatic enzymes, *including trypsin* and *phospholipase A2*.
- **Hemorrhage** is caused by activation of pancreatic enzymes, *including pancreatic elastase*, which dissolves elastic fibers of blood vessels.
Pancreatic exudate containing toxins and activated pancreatic enzymes permeates the retroperitoneum and at times the peritoneal cavity, inducing a chemical burn and increasing the permeability of blood vessels.

This causes extravasation of large amounts of protein-rich fluid from the systemic circulation into “third spaces,” producing hypovolemia and shock.
On entering the systemic circulation, these activated enzymes and toxins increase capillary permeability throughout the body and may reduce peripheral vascular tone, thereby intensifying hypotension.

Circulating activated enzymes may damage tissue directly.
CLINICAL FEATURES

- Abdominal pain
- Nausea
- Vomiting
- Restlessness
- Agitation
- Shock
- Coma
ABDOMINAL PAIN

- Acute in the epigastrium at the onset, and may be right upper quadrant, diffuse, or, infrequently, confined to the left side relieved on bending forward.

- The pain of pancreatitis can last for days. Its onset is rapid, but not as abrupt as that with a perforated viscus, is band-like radiation to the back.

- The abdominal pain is typically accompanied by nausea and vomiting, restlessness, and agitation.
PHYSICAL EXAMINATION
<table>
<thead>
<tr>
<th>Fever</th>
<th>Guarding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia</td>
<td>Abdominal</td>
</tr>
<tr>
<td>Hypotension</td>
<td>distention</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Hypoactive</td>
</tr>
<tr>
<td>Shallow</td>
<td>bowel</td>
</tr>
<tr>
<td>respirations</td>
<td>sounds</td>
</tr>
<tr>
<td>Epigastric</td>
<td>Shock</td>
</tr>
<tr>
<td>tenderness</td>
<td>Coma</td>
</tr>
<tr>
<td></td>
<td>Jaundice</td>
</tr>
</tbody>
</table>
OTHER SIGNS

- **Grey-Turner’s sign** Ecchymotic discoloration in the flank
- **Cullen's sign** Ecchymotic discoloration in the periumbilical region
- **Epigastric Mass** due to pseudocyst formation
- **Subcutaneous Nodular Fat Necrosis**, 0.5 to 2 cm tender red nodules, are usually located over the distal extremities but may occur elsewhere,
- **Thrombophlebitis** in the legs
- **Polyarthritis**
GREY-TURNER'S SIGN
Grey Turner's sign
CULLEN'S SIGN
FAT NECROSIS
FINDINGS INDICATIVE OF UNDERLYING DISORDERS

- Hepatomegaly in alcoholic pancreatitis
- Xanthomas in hyperlipidemic pancreatitis
- Parotid swelling associated with mumps
JAUNDICE
MUMPS
Mumps
DIFFERENTIAL DIAGNOSIS

- Perforated duodenal ulcer
- Mesenteric infarction
- Strangulating intestinal obstruction
- Ectopic pregnancy
- Dissecting aneurysm
- Biliary colic
- Appendicitis
- Diverticulitis
- Inferior wall MI
- Hematoma of abdominal muscles or spleen.
LABORATORY DIAGNOSIS

PANCREATIC ENZYMES

- Serum amylase
- Urine amylase
- Serum lipase
SERUM AMYLASE

- It rises within 6 to 12 hours of onset
- Its half-life is 10 hours
- In uncomplicated attacks, serum amylase is usually elevated for three to five days
- It is usually more than three times the upper limit of normal.
LIMITATIONS OF SERUM AMYLASE

1. May be normal or minimally elevated in patients with fatal pancreatitis or those who have a mild attack.

2. May remain normal if destruction of acinar tissue happened during previous episode.

3. May remain normal if there is coexisting hypertriglyceridemia.

4. Increased in other disorders.
Hyperamylasuria occurs in acute pancreatitis

Amylase-to-creatinine clearance ratio (ACCR), increases to approximately 10%

Moderate renal insufficiency interferes with accuracy and specificity of the ACCR.

Urinary amylase excretion is not increased in macroamylasemia.
The sensitivity for the diagnosis of acute pancreatitis is between 85 and 100%.

Lipase elevations occur earlier and last longer than amylase elevations.

The combination of enzymes does not improve diagnostic accuracy.
LABORATORY DIAGNOSIS

- The WBC count increases to 12,000 to 20,000/mL
- Hct may increase to as high as 50 to 55
- Hyperglycemia may occur
- Serum Ca concentration falls as early as the first day because of the formation of Ca “soaps” secondary to excess generation of free fatty acids
- Serum bilirubin increases in 15 to 25% of patients
Abdominal plain film

- Normal
- Sentinel Loop: Localized ileus of a segment of small intestine
- Colon cutoff sign: a paucity of air in the colon distal to the splenic flexure due to functional spasm of the descending colon
- Generalized ileus may occur in severe disease
Approximately one-third of patients will have abnormalities:

- Elevation of a hemidiaphragm
- Pleural effusions
- Basal atelectasis
- Pulmonary infiltrates
- ARDS
ABDOMINAL ULTRASOUND

- The classic ultrasonographic image of acute pancreatitis is a diffusely enlarged, hypoechoic pancreas.
- It can detect gallstones in the gallbladder.
- A less frequent pattern is the presence of focal hypoechoic areas.
- 25 to 35 percent of patients have bowel gas that may obscure the pancreas.
The most important imaging test for the diagnosis of acute pancreatitis and its intraabdominal complications.

Patients who do not improve with initial conservative therapy or who are suspected of having complications should undergo CT scan to identify any areas of pancreatic necrosis.

CT or US guided needle aspiration can differentiate between sterile and infected pancreatic necrosis or a pseudocyst.
**Ranson's Prognostic Signs**

At Admission

1. Age > 55 yr
2. Serum glucose > 200 mg/dL
3. Serum LDH > 350 IU/L
4. AST > 250 U
5. WBC count > 16,000/mL.

48 H After Admission

1. Hct decrease > 10%
2. BUN rise > 5 mg/dL
3. Serum Ca < 8 mg/dL
4. PaO2 < 60 mm Hg
5. Base deficit > 4 mEq/L
6. Estimated fluid sequestration > 6 L.
RANSON'S PROGNOSTIC SIGNS

Mortality increases with the number of positive signs

- If fewer than three signs are positive, the mortality rate is < 5%
- If three or four are positive, it is 15 to 20%
- If 5 positive criteria the mortality rate is 100%
Pancreatitis associated with necrosis and hemorrhage has a mortality rate > 10 to 50%.

If CT shows only mild pancreatic edema, the prognosis is excellent.

A markedly swollen pancreas denotes a more severe prognosis, especially when extravasation of fluid.
COMPLICATIONS

- Death
- Shock
- Sepsis
- Renal failure
- ARDS
- Pancreatic abscess
- Pancreatic necrosis
- Pancreatic pseudocysts
PANCREATIC ABSCESS
PSEUDOCYSTS

Collections of fluid escaped from the pancreatic ductal tree disrupted by acute inflammation and/or obstructed by stricture or stone, lacking an epithelial lining.
Large pseudocyst and pancreatic calcifications.
More common in alcoholic than biliary pancreatitis (15 vs 3%)
Occur in upto 20% of acute pancreatitis
>50% resolve spontaneously within 4-6 wks

Complications
1. Infection 11%
2. Haemorrhage into cyst (30-60% mortality)
3. Spontaneous rupture
4. Obstruction of duodenum
5. Obstruction of CBD
MANAGEMENT

- Fluid replacement
- Oxygenation
- Minimising pancreatic secretion
- Nutritional support
- Antibiotics
- Metabolic complications
TREATMENT

Mild Edematous Pancreatitis

- Pain treated with meperidine
- NPO until manifestations of acute inflammation subside
- IV fluids
- Insertion of a nasogastric tube
INDICATIONS FOR ICU ADMISSION

- Hypotension
- Oliguria
- Hypoxemia
- Hemoconcentration
Severe Acute Pancreatitis

- **Pain** should be treated with meperidene
- **Fasting** is maintained for 2 wk and possibly 3 to 4 wk.
- **Nasogastric tube** usually counteracts vomiting and intestinal ileus
- **H2 blockers**
- **Fluid** resuscitation 6 to 8 L/day
- **Blood Transfusion**
- **CVP** line or Swan-Ganz catheter
Severe Acute Pancreatitis

- **Hyperglycemia** treated with Insuline
- **Hypocalcemia** generally is not treated
- **Hypoxia** treated with Oxygen
- **Renal failure** should be treated by IV fluids if there is prerenal azotemia
- **Antibiotic** use is controversial
- **Peritoneal lavage** remains controversial
- **TPN** initiated within the first few days
SURGICAL INTERVENTION

during the first several days

1. Severe blunt or penetrating trauma
2. Uncontrolled biliary sepsis
3. Poor response to supportive treatment
4. Clinical deterioration
5. Progression of organ failure
6. Peritonitis
7. Inability to distinguish acute pancreatitis from a surgical emergency
THANK YOU

IMET 2000 Pal
International Medical Education Trust 2000 - Palestine

Raed Abu Sham'a, M.D