Acute Pancreatitis; Causes, Symptoms and Treatment

The principal function of the pancreas is to make food-digesting enzymes (exocrine) and Insulin (endocrine). It comprises only 0.1% of total body weight and has 13 times the protein-producing capacity of the liver and reticuloendothelial system combined, which make up 4% of total body weight.

Several mechanisms enable the pancreas to avoid digesting itself.

First, proteins are translated into an inactive form called proenzymes. The proenzymes are packaged in a paracrystalline arrangement with protease inhibitors.

Zymogen granules have an acidic pH and a low calcium concentration, which are factors that guard against premature activation

Under various conditions, these protective mechanisms are disrupted, resulting in intracellular enzyme activation and pancreatic autodigestion, a condition called **acute pancreatitis**.

This condition typically causes abdominal pain, usually associated with elevated pancreatic enzyme levels in the blood and inflammation of the pancreas.

The overall mortality rate of patients with acute pancreatitis is 10-15%.
In patients with severe disease, the mortality rate is approximately 30%.
In the first week of illness, most deaths result from multiorgan system failure.
In subsequent weeks, infection plays a more significant role, but organ failure still constitutes a major cause of mortality.

In general, acute pancreatitis affects males more often than females.

The etiology in males is more often related to alcohol; in females, to biliary tract disease.

Signs and symptoms of acute pancreatitis

History

The cardinal symptom of acute pancreatitis is abdominal pain, which is characteristically dull, boring, and steady. Most often, it is located in the upper abdomen, usually in the epigastric region, but it may be perceived more on the left or right side, depending on which portion of the pancreas is involved.

The pain radiates to the back in approximately half of the cases. The duration of the pain varies but typically lasts more than a day.

The pain may be aggravated by eating or lying supine and it may be alleviated by fasting or lying on the left side with the knees and hips flexed.

Associated symptoms (eg, anorexia, nausea, vomiting) are common, and some patients experience diarrhea due to indigestion. Avulsion to fatty foods may be reported.

Physical examination

The following physical examination findings vary with the severity of the disease.

1. Fever (76%) and tachycardia (65%) are common abnormal vital signs.

2. Abdominal tenderness, muscular guarding (68%), and distension (65%) are observed in most patients.
Bowel sounds are often hypoactive.

Pancreatitis has been associated with AIDS; however, this may be the result of opportunistic infections, neoplasms, or drug therapies

A minority of patients exhibit jaundice (28%).

Some patients experience dyspnea (10%), which may be caused by irritation of the diaphragm (resulting from inflammation) or by a more serious condition, such as respiratory distress syndrome.

In severe cases, hemodynamic instability is evident (10%) and hematemesis or melena sometimes develops (5%).

A few uncommon physical findings are associated with severe **necrotizing pancreatitis.**

The **Cullen sign** is a bluish discoloration around the umbilicus resulting from hemoperitoneum.

The Grey-Turner sign is a reddish-brown discoloration along the flanks resulting from retroperitoneal blood dissecting along tissue planes.

Erythematous skin nodules may result from focal subcutaneous fat necrosis.

Rarely, abnormalities on funduscopic examination may be seen in severe pancreatitis. Purtscher retinopathy, this ischemic injury to the retina appears to be caused by activation of complement and agglutination of blood cells within retinal vessels. It may cause temporary or permanent blindness.

Causes of acute pancreatitis

Although pancreatitis has numerous etiologies, alcohol dependence and biliary tract disease cause most cases.

In 10-30% of cases, the cause is unknown, and careful evaluation may identify a rare etiology in 10% of cases.

1. Biliary tract disease (approximately 38%)
The most common cause of acute pancreatitis is **gallstones** passing into the bile duct and

temporarily lodging at the sphincter of Oddi. The risk of a stone causing pancreatitis is inversely proportional to its size.
Abnormal anatomy (eg, choledochal cysts, juxtapapillary diverticula) is also associated with acute pancreatitis.

2. Alcohol (approximately 35%)
Alcohol abuse is a major cause of pancreatitis, evidence shows that alcohol may cause acute pancreatitis in the absence of chronic disease.

3. Post-ERCP (approximately 4%)

4. Trauma (~1.5%)
Abdominal trauma causes an elevation of amylase and lipase levels in 17% of cases and clinical pancreatitis in 5% of cases.

5. Drugs (~1.4%) of drug-induced pancreatitis is usually mild.
Drugs definitely associated with acute pancreatitis include azathioprine, sulfonamides, sulindac, tetracycline, valproic acid, didanosine, methyldopa, estrogens, furosemide, 6-mercaptopurine, pentamidine, 5-aminosalicylic acid compounds, corticosteroids, and octreotide.

6. *Infection* (<1%))Viral causes include mumps, Epstein-Barr, coxsackievirus, echovirus, varicella-zoster, and measles.
Bacterial causes include Mycoplasma pneumoniae, Salmonella, Campylobacter, and Mycobacterium tuberculosis.
Worldwide, ascariasis is a recognized cause of pancreatitis resulting from the migration of worms in and out of the duodenal papillae.

7.Hereditary pancreatitis (<1%)
This type of pancreatitis is an autosomal dominant disorder related to mutations of the cationic trypsinogen gene

8. Hypercalcemia (<1%)
Hypercalcemia from any cause can lead to acute pancreatitis

9. Developmental abnormalities of the pancreas (<1%)

10. Hypertriglyceridemia (<1%)

11.A tumor (<1%)
Obstruction of the pancreatic ductal system by a pancreatic ductal carcinoma, ampullary carcinoma,

Pathophysiology

Acute pancreatitis occurs as a result of premature intracellular trypsinogen activation, releasing proteases which then digest the pancreas and surrounding tissue.

There are a number of triggers for this including alcohol, gallstones and pancreatic duct obstruction. There is simultaneous activation of nuclear factor kappa B (NF?B), leading to

mitochondrial dysfunction, autophagy, and a vigorous in?ammatory response.

The normal pancreas has only a poorly developed capsule, and adjacent structures, including the common bile duct, duodenum, splenic vein and transverse colon, are commonly involved in the in?ammatory process.

The severity of acute pancreatitis is dependent upon the balance between the activity of released proteolytic enzymes and antiproteolytic factors.

The latter comprises an intracellular pancreatic trypsin inhibitor protein and circulating ?2-macroglobulin, ?1-antitrypsin, and Cl-esterase inhibitors.

Acute pancreatitis is often self-limiting but in some patients with severe disease, local complications,
such as necrosis, pseudocyst or abscess, occur, as well as systemic complications that lead to multi-organ failure.

Diagnosis

The diagnosis is based upon raised serum amylase or lipase concentrations. A persistently elevated
serum amylase concentration suggests pseudocyst formation. Peritoneal amylase concentrations are massively elevated in pancreatic ascites

Carry out Imaging studies such as:

1.Abdominal radiography
These radiographs are primarily used to detect free air in the abdomen, indicating a perforated viscus.
The presence of calcifications within the pancreas may indicate chronic pancreatitis.

2.Abdominal ultrasonography
This is the most useful initial test in determining the etiology of pancreatitis and is the technique of choice for detecting gallstones.

3. Abdominal CT scanning
This is generally not indicated for patients with mild pancreatitis unless a pancreatic tumor is suspected (usually in elderly patients).

Treatment

Management comprises several related steps:

1. Establishing the diagnosis and disease severity
2. Early resuscitation, according to whether the disease is mild or severe
3. Detection and treatment of complications
4. Treating the underlying cause.

Treat pain with opioids and hypovolaemia should be corrected using normal saline or other

crystalloids. All severe cases should be managed in a high-dependency or intensive care unit.

A central venous line and urinary catheter should be established to monitor patients with shock.

Oxygen should be given to hypoxic patients, and those who develop systemic in?ammatory response syndrome (SIRS) may require ventilatory support.

Hyperglycaemia should be corrected using insulin, but it is not usually necessary to correct hypocalcemia by intravenous calcium injection, unless tetany occurs.

Nasogastric aspiration is only required if paralytic ileus is present.

Enteral feeding should be st