PROFESSIONAL GUIDE TO DISEASES
(Fourth Edition)
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PANCREATITIS

Pancreatitis, inflammation of the pancreas, occurs in acute and chronic forms and may be due to oedema, necrosis, or haemorrhage. In men, this disease is commonly associated with alcoholism, trauma, or peptic ulcer; in women, with biliary tract disease. Prognosis is good when pancreatitis follows biliary tract disease but poor when if follows alcoholism. Mortality rises as high as 60 percent when pancreatitis is associated with necrosis or haemorrhage.

CAUSES.

The most common causes of pancreatitis are biliary tract disease and alcoholism, but it can also result form pancreatic carcinoma, trauma, or certain drugs, such as glucocorticoids, sulphonamides, chlorothiazide, and azathioprine. This disease may also develop as a complication of peptic ulcer, mumps, or hypothermia. Rarer causes are stenosis or obstruction of the sphincter of Oddi, hyperlipemia, metabolic endocrine disorders (hyperparathyroidism, haemochromatosis), vasculitis or vascular disease, viral infections, mycoplasmal pneumonia, and pregnancy.

Afro-Asian syndrome (diabetes, pancreatic insufficiency and calcification) occurs in some persons, probably from malnutrition and alcoholism, and leads to pancreatic atrophy. Regardless of the cause, pancreatitis involves autodigestion: the enzymes digest pancreatic tissue.

SIGNS AND SYMPTOMS.

In many patients, the first and only symptom of mild pancreatitis is steady epigastric pain centred close to the umbilicus, radiating between the tenth thoracic and sixth lumbar vertebrae, and unrelieved by vomiting. However, a severe attack causes extreme pain, persistent vomiting, abdominal rigidity, diminished bowel activity (suggesting peritonitis), crackles at lung bases, and left pleural effusion. Severe pancreatitis may produce extreme malaise and restlessness, with mottled skin, tachycardia, low-grade fever (100 to 102F 37.7 - 38.8C), and cold, sweaty extremities. Proximity of the inflamed pancreas to the bowel may cause ileus.

If pancreatitis damages the islets of Langerhans, complications may include diabetes mellitus. Fulminant pancreatitis causes massive haemorrhage and total destruction of the pancreas, resulting in diabetic acidosis, shock, or coma.

DIAGNOSIS.

A careful patient history (especially for alcoholism) and physical examination are the first steps in diagnosis, but the retroperitoneal position of the pancreas makes physical assessment difficult. Dramatically elevated serum amylase levels - frequently over 500 units - confirm pancreatitis and rule out perforated peptic ulcer, acute cholecystitis, appendicitis, and bowel infarction or obstruction. Similarly, dramatic elevations of amylase also occur in urine, ascites, or pleural fluid. Characteristically, amylase return to normal 48 hours after onset of pancreatitis, despite continuing symptoms.

Supportive laboratory values include:

* increased serum lipase levels, which rise more slowly than serum amylase
* low serum calcium (hypocalcaemia) from fat necrosis and formation of calcium soaps
* white blood cell counts range from 8,000 to 20,000/mm3, with increased polymorphonuclear leukocytes
* elevated glucose levels - as high as 500 to 900 mg/dl, indicating hyperglycaemia
* haematocrit occasionally exceeding 50 percent concentrations

Results of other tests may include:

* EKG changes (prolonged Q-T segment but normal T wave), which help diagnose hypercalcemia
* abdominal x-rays that show dilation of the small or large bowel or calcification of the pancreas
* GI series, indicating extrinsic pressure on the duodenum or stomach due to oedema of the pancreas head
* chest x-rays showing left-side pleural effusion
* ultrasound or CT scan to help distinguish acute cholecystitis from acute pancreatitis
* analysis of abdominal fluid to detect amylase levels as high as 7,000 units. In the patient with a perforated bowel, it may also detect bacteria or bile

TREATMENT

Treatment must maintain circulation and fluid volume, relieve pain and decrease pancreatic secretions. Emergency treatment for shock (the most common cause of death in early-stage pancreatitis) consists of vigorous IV treatment replacement of electrolytes and proteins. Metabolic acidosis secondary to hypovolemia and impaired cellular perfusion requires vigorous fluid volume replacement.

Treatment may also include meperidine for pain (although it may cause spasm of the sphincter of Oddi); diazepam for restlessness and agitation; and antibiotics such as gentamicin, clindamycin, or chloramphenicol, for bacterial infections. Hypocalcaemia requires infusion of 10 percent calcium gluconate; serum glucose levels greater than 300 to 350 mg/dl require insulin therapy.

After the emergency phase, continuing IV therapy should provide adequate electrolytes and protein solutions that don't stimulate the pancreas (glucose or free amino acids) for 5 to 7 days. If the patient is not ready to resume oral feedings by then, hyperalimentation may be necessary. Non stimulating elemental gavage feedings may be safer because of the decreased risk of infection and over infusion. In extreme cases, laparotomy to drain the pancreatic bed, 95 percent pancreatectomy, or a combination of cholecystostomy-gastrostomy, feeding jejunostomy, and drainage may be necessary.

SPECIAL CONSIDERATIONS.

Acute pancreatitis is a life-threatening emergency. Design your care-plan to provide meticulous supportive care and continuous monitoring of vital systems.

* Monitor vital signs and pulmonary artery pressure closely. If the patient has a central venous pressure line instead of a pulmonary artery catheter, monitor it closely for volume expansion (it shouldn't rise above 10 cmH20). Give plasma or albumin, if ordered, to maintain blood pressure. Record fluid intake and output; check urine output hourly, and monitor electrolyte levels. Assess

for crackles, rhonchi, or decreased breath sounds

* For bowel decompression, maintain constant nasogastric suctioning, and give nothing by mouth. Perform good mouth and nose care
* Watch for signs of calcium deficiency - tetany cramps, carpopedal spasm, and convulsions. If you suspect hypocalcaemia, keep airway and suction apparatus handy and pad side rails
* Administer analgesics, as needed, to relieve the patient's pain and anxiety. Remember that anticholinergics reduce salivary and sweat gland excretions. Warn the patient that he may experience dry mouth and facial flushing. Caution: Narrow-angle glaucoma contraindicates the use of atropine or its derivatives
* Watch for adverse reactions to antibiotics: nephrotoxicity with aminpglycosides; pseudomembranous enterocolitis with clindamycin; and blood dyscrasias with chloramphenicol
* Don't confuse thirst due to hyperglycaemia (indicated by serum glucose levels up to 350 mg/dl and sugar and acetone in urine) with dry mouth due to nasogastric intubation and anticholinergics.
* Watch for complications due to hyperalimentation, such as sepsis, overhydration and metabolic acidosis. Watch for fever, cardiac irregularities, changes in arterial blood gas measurements, and deep respirations. Use strict aseptic technique when caring for the catheter insertion site.

CHRONIC PANCREATITIS

Chronic pancreatitis is usually associated with alcoholism (in over half of all patients), but can also follow hyperparathyroidism, hyperlipemia, or infrequently, gallstones, trauma or peptic ulcer. Inflammation and fibrosis causes progressive pancreatic insufficiency and eventually destroy the pancreas. Symptoms of chronic pancreatitis include constant dull pain with occasional exacerbations, malabsorption, severe weight loss, and hyperglycaemia (leading to diabetic symptoms). Relevant diagnostic measures include patient history, x-rays showing pancreatic calcification, elevated ESR, and examination of stool for steatorrhea.

The severe pain of chronic pancreatitis often requires large doses of analgesics or narcotics, making addiction a serious problem. Treatment also includes a low-fat diet and oral administration of pancreatic enzymes such as pancreatin or pancrelipase to control steatorrhea, insulin or oral hypoglycaemics to curb hypoglycaemia, and occasionally, surgical repair of biliary or pancreatic ducts, or the sphincter of Oddi to reduce pressure and promote the flow of pancreatic juice. Prognosis is good if the patient can avoid alcohol; poor if he can't.