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Department of surgical diseases No. 2

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ACUTE PANCREATITIS

Textbook for students of 4 courses medical faculty of faculty surgery

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This teaching guide covers main issues about etiology, pathogenesis, clinical features, laboratory and instrumental diagnostics and complications of Acute pancreatitis.

Teaching guide “Acute pancreatitis” is made for “Faculty surgery” discipline in accordance with requirements of FSES HE, and is intended for students from medical universities and faculties, studying for specialty 31.05.01 General medicine.

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SURGICAL ANATOMY OF THE PANCREAS

The pancreas is both endocrine and exocrine organ situated retroperitoneally behind the stomach. It is a soft and fleshy gland(pancreas-all flesh), extending from the duodenum on the right side to the spleen on the left side, the entire length being 6 inches. It weighs approximately 80 g.

Parts

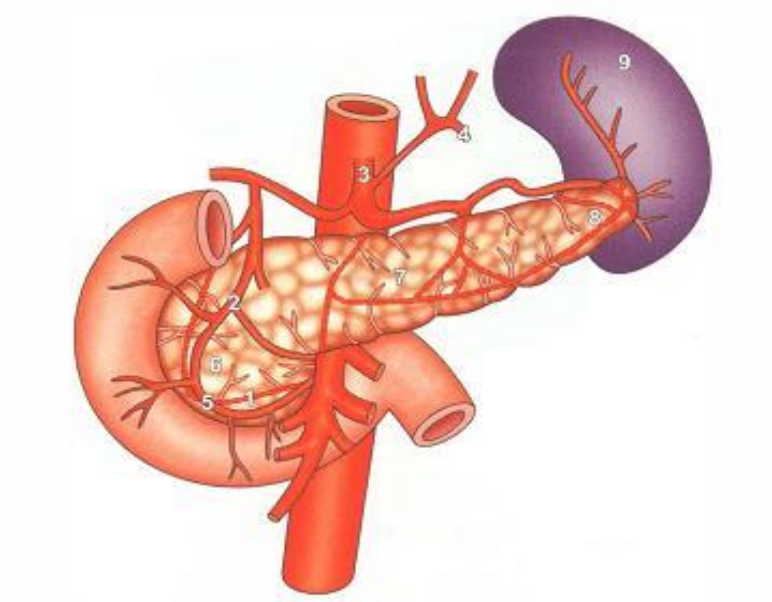
- The head lies within the C-loop of duodenum. The uncinate process projects from the left inferior portion of the head over which course the superior mesenteric vessels. There are 5-6 small thin veins connecting this portion of the head with superior mesenteric veins. These veins have to be carefully divided during pancreaticoduodenectomy. Superior mesenteric vein continues above as po1tal vein after joining the splenic vein. During pancreatico-duodenectomy for periampullary carcinoma, infiltration into the portal vein should be ruled out before any major structure is divided. This is done by inserting a finger between the portal vein and head of pancreas, both from above and below.
- The neck is about 2 cm and is related posteriorly to superior mesenteric vessels.
- Body and tail: The head and neck continue as body which is placed transversely. It slopes upwards across the aorta and ends as tail of the pancreas, which is enclosed within lienorenal ligament along with splenic vessels. A large cystadenoma arising from the tail of the pancreas can move with respiration because of its contact with the spleen.

Blood supply of the pancreas Arterial supply

- Splenic artery is the chief artery supplying the neck, body and the tail. Arteria pancreatica magna refers to one large branch of splenic artery.
- Superior and inferior pancreaticoduodenal arteries supply not only head of pancreas but also the adjacent duodenum. Thus, during any surgery which involves excision of the head, the C-loop of the duodenum is also removed. Thus, pancreaticoduodenectomy becomes a major surgery.

Venous drainage

- Body, neck and tail drain into splenic vein by means of multiple small veins.
- The head is drained by superior pancreaticoduodenal vein which drains into portal vein and inferior pancreatico- duodenal vein drains into superior mesenteric vein.



Blood supply and parts of pancreas: (1) Uncinate process, (2) Superior pancreaticoduodenal, (3) Coeliac artery, (4) Splenic artery, (5) Inferior pancreaticoduodenal, (6) Head of pancreas, (7) Body of pancreas, (8) Tail of pancreas and (9) Spleen

Islets of Langerhans (endocrine)

- 75% : β Cells
- 20% : a Cells
- 4% : γ Cells

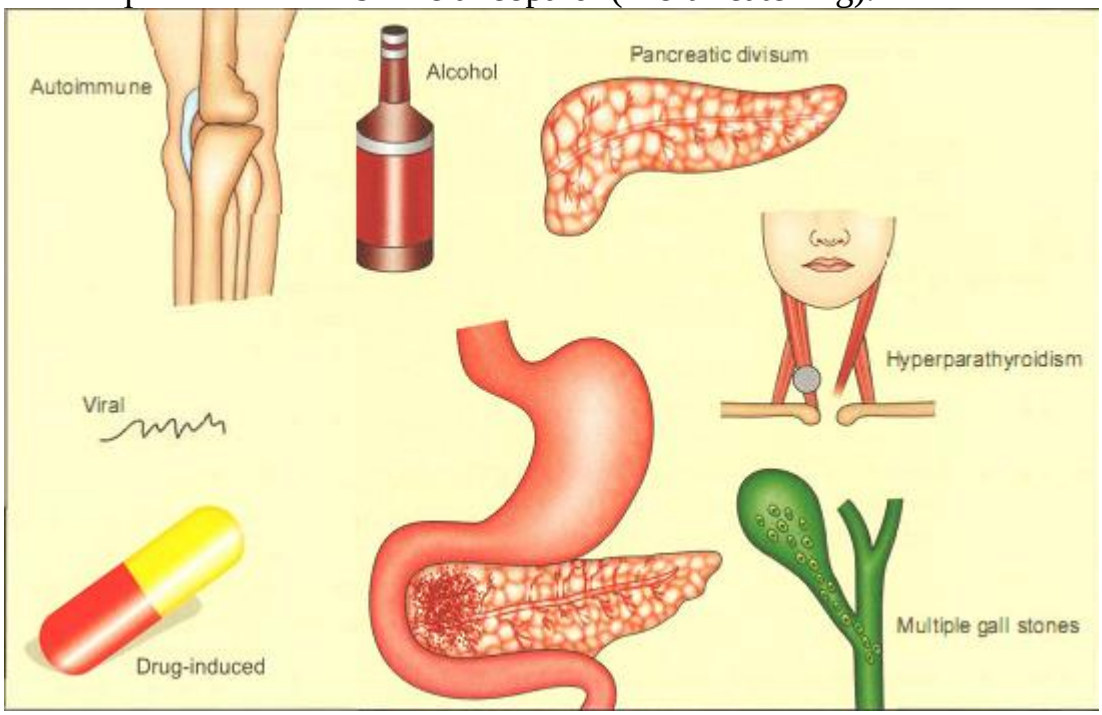
Pancreatic duct

- The main pancreatic duct (duct of Wirsung), a tubular structure drains entire pancreas from tail to the head. It joins the common bile duct and formns ampulla of Vater. This ampulla opens on the duodenal papilla (a nipple-like elevation) in the 2nd part of the duodenum. Normal diameter of pancreatic duct is 2-3 mm. When it is dilated more than 6-8 mm, as in chronic pancreatitis, longitudinal pancreatico-jejunostomy can be done.
- Accessory pancreatic duct of Santorini drains the uncinate process and lower portion of the head and opens into the duodenum 2 cm above the opening of the main duct. The two ducts communicate with each other at many sites.
- The main pancreatic duct is lined by columnar epithelium which becomes cuboidal in the ductules.

ACUTE PANCREATITIS

Definition: It is defined as acute nonbacterial inflammatory condition caused by activation, interstitial liberation and auto-digestion of pancreas presenting as acute abdominal pain.

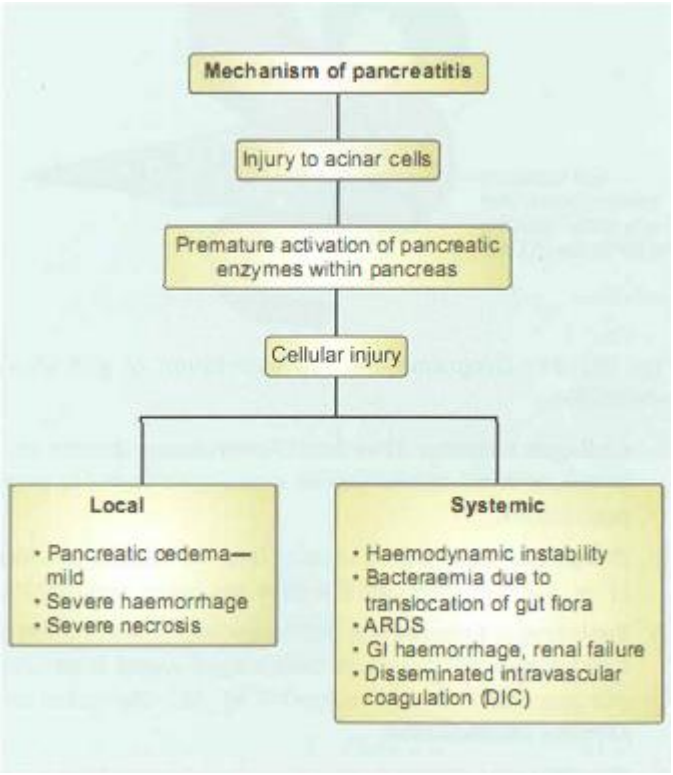
• Acute pancreatitis stings like a scorpion (produces severe pain).
• Acute pancreatitis drinks like a fish (produces dehydration).
• Acute pancreatitis eats like a wolf (pancreatic necrosis).
• Acute pancreatitis burrows like a rodent (produces fistula).
• Acute pancreatitis kills like a leopard (life-threatening).



Marseille's classification of pancreatitis

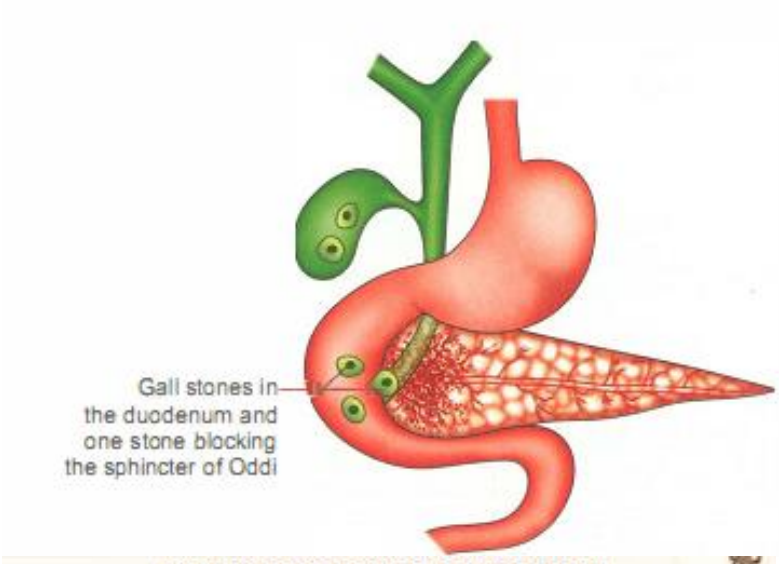
I. Acute pancreatitis
2. Acute relapsing pancreatitis
   - In both these conditions, pancreas returns back to normal.
3. Chronic pancreatitis
4. Chronic relapsing pancreatitis
   - In both these conditions there is always permanent damage to the pancreas.

AETIOLOGY



1. Alcohol abuse (40 to 50%): It is the major cause of acute pancreatitis in our country and is seen in about 50% of the cases. Alcohol stimulates pancreatic secretions rich in protein, forms protein plugs and results in obstruction to the pancreatic duct. Alcohol stimulates trypsinogen. It causes spasm of sphincter of Oddi. It also has direct toxic effect on the pancreas. Tobacco smoking contributes to its effects.
2. Biliary tract disease: Stone in the biliary tree (gallstone pancreatitis) is the major cause of acute pancreatitis in the Western world (40%). In our country, it may be responsible for pancreatitis in about 20-30% of patients.
3. Collagen vascular disorders: Autoimmune disease such as polyarteritis nodosa can be a causative factor in acute pancreatitis.
4. Drugs: Corticosteroids, tetracycline, oestrogens, azathio-prine, valproic acid and diuretics can cause pancreatitis.
5. Endoscopic procedures: Sphincterotomy, cannulation of CBD or pancreatic duct, or basketing of stones from CBD can precipitate acute pancreatitis by duct disruption and enzyme extravasation.
6. Familial or genetic factors have been blamed or acute pancreatitis. Hereditary pancreatitis may be due to mutation of cationic trypsinogen gene. Symptoms begir in early childhood.
7. Hyperparathyroidism causing hypercalcaemia ma) stimulate pancreatic juices and can cause pancreatitis. I also facilitates precipitation of calcium in the ducts.
8. Hyperlipidaemia (> 1000 mg/dl of triglycerides) can also cause pancreatitis. Dietary control can cure pancreatitis.

9. Hypothermia and hypotension can cause ischaemia to the pancreas resulting in acute pancreatitis.
10. Injury to the pancreas either postoperative or following penetrating injury can result in pancreatitis.
11. Infection: Virus such as Mumps and Coxsackie can cause pancreatic. Scorpion sting can also cause pancreatitis abnormality.
12. Ductal: Around 20%. Some of them are due to pancreatic divisum seen during ERCP. The opening of the minor papilla is inadequate for drainage of pancreatic juice.
13. Postoperative pancreatitis
• It follows operations on CBD-open or laparoscopic. More common after T-tube insertions (now it is less).
• Sphincteroplasty, ERCP, stone extractions
• Pancreatic biopsy
• Gastrectomy (distal/total)
• Cardiac surgery. Here risk actors are perioperative administration of calcium, postoperative hypotension, pre-operative renal failure.
14. Idiopathic: It is seen in about 15% of cases. Even though, classified as idiopathic (no cause is found), they are found to have sludge/gall stones undetected by ultrasound examination.



QUICK REFERENCE TO COMMON CAUSES OF PANCREATITIS

- Pancreatitis in teens—suspect **hereditary or APBDJ**[1]
- Pancreatitis in women—suspect **gall stones**
- Pancreatitis in males—alcoholic pancreatitis
- Pancreatitis with **fleeting joint pains—autoimmune pancreatitis.**
- Pancreatitis with bony lesions/cysts—**hyperparathyroidism**
- Pancreatitis with fever—viral

[1]Abnormal pancreatico-biliary duct junction

PATHOGENESIS

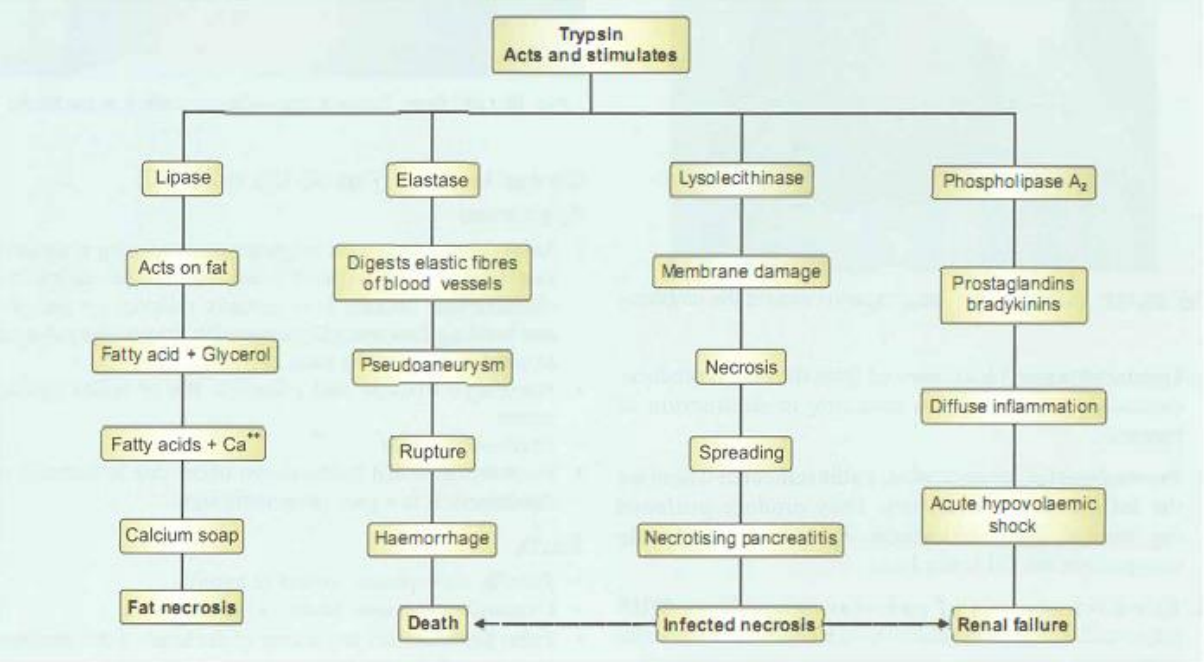
- Autodigestion is the final common pathway leading to pancreatitis, a few theories have been explained as possible actors for autogenesis.

1. Obstruction/secretion: Recurrent attacks of pancreatitis as in chronic cases can be due to multiple strictures, stasis, resulting in autodigestion.
2. Common channel theory: In about 90% of cases, bile duct and pancreatic duct converge into ampulla of Vater. However, only in 10% of cases they have common channel. Reflux of bile into pancreas has been blamed for 'gall stone pancreatitis'. However, strong evidence is lacking.
3. Duodenal reflux: Activation of enzymes takes place in the duodenum through action of enterokinase. Hence, it is postulated that duodenal reflux is one of the causes of acute pancreatitis.
4. Back diffusion: Back diffusion of pancreatic enzymes through the ductal epithelium is a possible actor, when damaged by alcohol/bile acids, etc.
5. Systemic sepsis: Organ failure

- ARDS, renal failure
- Endotoxins originate from bacteria.

- Acute pancreatitis is an autodigestion following activation of trypsinogen. This is brought about by various agents mentioned above.
- It may also be due to relux of bile into the pancreas.
- Trypsinogen is converted into trypsin. It acts and stimulates:

1. Lipase: Lipase splits the fat into fatty acids and glycerol. Fatty acids combine with calciun to form calcium soap. This is represented as at necrosis seen in the omentum, subsynovial pockets of knee joint, etc. This also explains hypocalcaemia and tetany seen in acute pancreatitis.
2. Elastase: It digests the elastic fibres of the blood vessels resulting in rupture and haemorrhage into the peritoneal cavity.
3. Lysolecithinase: This is derived from the bile. It produces extensive tissue necrosis resulting in destruction of pancreas.
4. Prostaglandins, bradykinins, kallikrein, etc.: These are the inflammatory mediators. They produce profound hypotension, shock and collapse, due to loss of fluid in the retroperitoneum (III space loss).
5. Extensive necrosis of pancreas: It produces MDF (myocardial depressant actor) which depresses ventricular contraction resulting in cardiac failure. Ultimate result is the development of multi-organ failure.



CLINICAL FEATURES

Symptoms

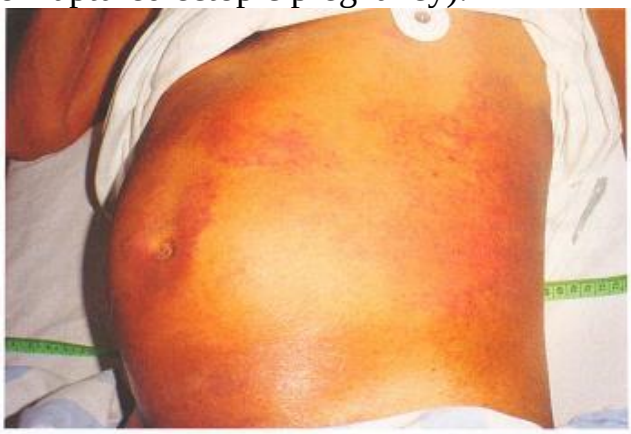
- Severe upper abdominal epigastric pain radiating to the back increases over a period of hours-illimitable agony is a characteristic feature. It is partially relieved on stooping and bending forwards (Mohammedan Prayer sign). A meal or alcohol triggers the pain.
- Vomiting-frequent and effortless due to reflex pyloro-spasm.
- Fever-low grade
- Haematemesis and melaena can occur due to necrosis of duodenum. It is a poor prognostic sign.

Signs

- Febrile, tachypnoeic patient in agony.
- Cyanosis-improper perfusion of lungs.
- Faint jaundice due to oedema of the head of the pancreas.
- Features of shock-feeble pulse, tachycardia, hypotension, cold extremities.
- Abdominal findings

- Tenderness in epigastrium
- Upper abdominal guarding and rigidity
- Distension of the abdomen
- Mass in epigastrium
- Muscle guarding
- Abdominal distension due to either accumulation of blood or fluid in the peritoneal cavity or due to paralytic ileus.

- Cullen's sign
- Bluish ecchymotic discolouration seen around umbilicus (first described or ruptured ectopic pregnancy).



Grey Turner's sign—discolouration in the flanks

(Fig. 1)

- Grey Turner's sign (Fig. 1)
- Bluish discolouration in the flanks
- Both these signs are due to peri-pancreatic and retroperitoneal haemorrhage and seepage of blood along fascial planes, into the anterior abdominal wall and spread through falciform ligament.
- Evidence of respiratory signs: Tachypnoea, dullness, effusion, crepitations, rhonchi (pulmonary oedema, ARDS).

INVESTIGATIONS

1. Haemogram (CBP): Hb% may be low due to haemorrhagic pancreatitis.
- Total count is raised above 15,000 cells/mm3 due to inflammation.
2. Blood or urea, creatinine to rule out renal failure
3. Serum amylase (widely used test) (Key Box 25.32).
- Normal levels are 40-80 Somogyi units
- Values around 400 are suggestive and values more than 1000 Somogyi units are diagnostic of acute pancreatitis.
- It is increased in the first 24-48 hours and retunrs to normal within 3-4 days

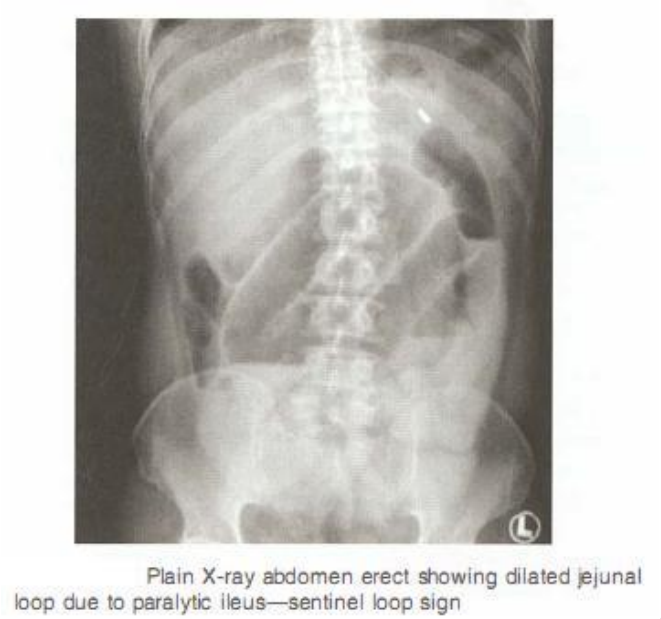
INCREASED AMYLASE LEVELS ARE SEEN IN

- Acute pancreatitis and its complications
- Parotitis
- Afferent loop obstruction
- Spasm of sphincter of Oddi
- Biliary peritonitis—duodenal injuries
- Mesenteric infarction
- Ruptured ectopic gestation

- Persistent high level of amylase in acute pancreatitis indicates:

- Unresolving inflammation
- Recurrent attacks of pancreatitis
- Complications-pseudocyst, pancreatic abscess
- Serum lipase levels-more specific but difficult to measure. Lipase is only secreted by pancreas.

4. Blood and urine sugar estimation: Glycosuria is present in almost 100% of patients.
5. Serum calcium levels: Hypocalcaemia is seen, due to hypoalbuminaemia or fat necrosis.
6. Total proteins are usually low, especially albumin.
7. Plain X-ray abdomen (erect position) (Fig. 2)

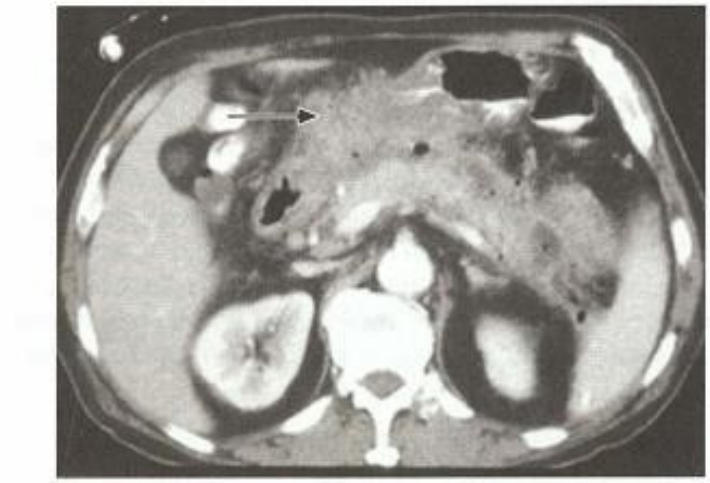


Plain X-ray abdomen erect showing dilated jejunal loop due to paralytic ileus—sentinel loop sign

(Fig. 2)

- Calcification suggests chronic pancreatitis.
- To rule out perforation of peptic ulcer.
- Sentinel loop sign-one dilated jejuna! loop of intestine which is seen in the region of pancreas.
- Colon 'cut-off' sign refers to mild distension of transverse colon with collapsed descending colon.

8. Abdominal ultrasound-can demonstrate oedematous pancreas, fluid in the abdomen or biliary tract disease.
9. Contrast enhanced CT scan of abdomen is done after 3-5 days in patients who fail to respond to conservative treatment. IfCT scan demonstrates infected necrosis, an urgent CT-guided FNAC is done and Gram stain is sent. If Gram stain is positive, it has to be treated urgently (Fig. 3).



Contrast enhanced CT (CECT) scan is the gold standard investigation in cases of acute pancreatitis. Useful after 7 days of acute pancreatitis. This photograph shows extensive necrosis. CT scan also helps in detecting pseudocysts, abscesses, presence of gall stones or any other unsuspected pathology in the abdomen. Modern thinking is to wait for 4–6 weeks for separation of necrosis before surgical necrosectomy

(Fig. 3)

Indications for CT scan in acute pancreatitis

- Patients with severe pancreatitis
- When the diagnosis is in doubt
- Patients with organ failure
- Patients with sepsis
- Localised complications such as pseudocyst, pseudo- aneurysms.
- Clinical deterioration

MANAGEMENT

- Almost always conservative. However, a few scoring systems have been followed (Table 1).

Prognostic indicators/severity predictors

Ranson score	Glasgow Scale
ON ADMISSION	
Age > 55 years	Age > 55 years
WBC count > 16 × 10^9/L	WBC count > 15 × 10^9/L
Blood glucose > 10 mmol/L	Blood glucose > 10 mmol/L
LDH > 700 units/L	Serum urea > 16 mmol/L
AST > 250 Sigma Frankel unit/unit	PaO_2 < 60 mm Hg
WITHIN 48 HOURS	
Increase in blood urea nitrogen levels > 5 mg%	Serum calcium < 2 mmol/L
Drop in haematocrit > 10%	Serum albumin < 32 g/L
Arterial oxygen saturation (PaO_2 < 60 mmHg)	LDH > 600 units/L
Serum calcium < 2 mmol/L	AST/ALT > 600 units/L
Base deficit > 4 mmol/L	
Fluid sequestration > 6 L	

(Table 1)

- Even though management principles of any acute abdomen includes 'nil per oral' so as to give rest to the part, in casei of pancreatitis it has been divided broadly into 3 categories. It is desirable to know the treatment followed fo1 these categories. It has been given in Table 2.

Management principles of acute pancreatitis

	Acute oedematous (Mild—80%)	Necrotising pancreatitis (Sterile necrosis—10%)	Infected necrosis (Very severe—5%)
Admission	Acute ward with monitoring of vitals	Intensive care unit	Intensive care unit
IV fluids/ hypotension	**Early correction of hypotension,** hypovolaemia—crystalloids	IV fluids and inotropic* support may be required	May require inotropes and vasopressors for a long period
Blood transfusion	Rarely required	May be required	Definitely required
Antibiotics	No antibiotics	Early antibiotic prophylaxis is required	Broad spectrum antibiotics—imipenem, ciprofloxacin, metronidazole, etc.
Oral/nutrition	Oral fluids, soft diet by 3–4 days, once pain and ileus settle down	If pain is still present even after 4 days, nasojejunal feeding to be done	Enteral feeding/nasojejunal feeding. If calories are not sufficient, total parenteral nutrition is required
Hypomagnesaemia/ hypocalcaemia	Usually will not be a problem	Correction is required	Correction is required
Oxygen	By nasal cannula/face mask may be required	Early ARDS—ventilatory support	Ventilatory support may be required
Role of surgery/ natural course	Majority of patients will not require surgery. If gall stones are impacted at sphincter of Oddi, endoscopic basketing and laparoscopic cholecystectomy should be done	By 10–15 days, may resolve completely or may develop into pseudocyst or infected necrosis which require surgery	Ideal time to operate is after 4 weeks when necrosis is demarcated well

* Dopamine 2–5 µg/kg/min can help in renal perfusion. 5–10 µg/kg/min acts as an inotrope and 10–20 µg/kg/min as a vasopressor. Noradrenaline 50–200 ng/kg/min as a vasopressor.

(Table 2)

INDICATIONS FOR SURGERY IN ACUTE PANCREATITIS

- Infected necrosis
- Pancreatic abscess
- Diagnosis is in doubt—perforated viscus cannot be ruled out.
- Complications such as massive bleeding not responding to conservative treatment.
- Cholangitis not responding to treatment.

- Those of you who find it difficult can remember at least few fundamentals of managing acute pancreatitis. They are given in the fonn of simple headlines: ABCDEF

A. Aspiration with Ryle's tube, to give rest to the pancreas (may be for one or two days).
B. Blood transfusion if Hb% is low, or albumin and amino acids if proteins are low.
C. Charts-increasing pulse, increasing temperature indicates pancreatic abscess which needs laparotomy and drainage.
D. Drugs: Prophylactic antibiotics-used in cases of severe pancreatitis for prevention of local or general complication. IV cefuroxime for imipenem or ciprofloxacin with metronidazole are given. Low molecular weight dextran (lomodex) 500 ml can be used to increase renal perfusion. Alternately, dopamine 2 µg/kg/min can be given IV which helps in renal perfusion (in case of oliguria).
E. Exploratory laparotomy, only when diagnosis is in doubt, when patient is not improving or when there is a complication of pancreatitis such as pancreatic abscess, fistula or necrosis. With the advent of wonderful imaging techniques, surgical intervention is rarely being done in the initial phase of acute pancreatitis. In early cases, pancreas should not be handled. Peritoneal lavage is done followed by insertion of tube drain. Lavage has shown some benefit. In cases of infected necrosis, necrosectomy is done.

- The wound can be left open as laparotomy or with mesh or with zip.

F. Fluid should be given early. Rapid infusion of 3-4 litres of Ringer lactate is used to treat hypovolaemic shock. Plasma or albumin may also be given.

- Factors predicting severe pancreatitis.

IDENTIFICATION OF SEVERE PANCREATITIS

• Age	: > 70 years
• Obesity	: BMI > 30 kg/m^2
• Pleural effusion	: Present
• CT necrosis	: > 50
• CT severity index	: CTSI
• Persistent	: Organ failure

PANCREATIC NECROSECTOMY

- Ideally done 4-6 weeks later when sepsis is still present.
- CT-guided FNAC/bacterial culture is a must.
- A midline laparotomy is done
- Lesser sac is entered
- Thorough debridement of the dead tissue is done.
- All luid and tissue should be sent or aerobic and anaerobic culture.
- Blunt dissection rather than sharp dissection is done to minimise bleeding.
- Antibiotics of choice should be carbapenems and quino-lones.
- Ileostomy should be done for retrocolic necrosis
- Cholecystectomy should be done for gall stone pancreatitis cases.
- Perfect haemostasis should be achieved using manual compression, sutures and ligatures.
- In cases of early necrosectomy, lesser sac is packed reoperation done after 48 hours-zipper closure of the abdomen is used.
- Continuous lavage of the lesser sac and retroperitoneum i: done.
- Other alternate methods are closed packing.
- Today all these cases are done through minimally invasive methods.
- Endoscopic necrosectomy, retroperitoneoscopic necrosec-tomy is also done.

COMPLICATIONS OF ACUTE PANCREATITIS

Complications of acute pancreatitis

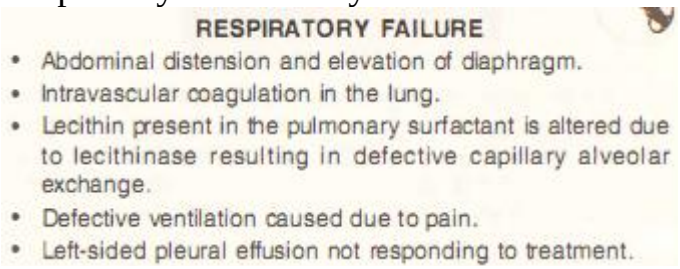
Local	General
• Pancreatic necrosis (sterile)	• Pulmonary—ARDS
• Pancreatic necrosis (infected)	• Cardiac—shock, arrhythmias
• Pancreatic abscess	• Renal—renal failure
• Pancreatic ascites	• Gastro—ileus, intestinal colonic necrosis
• Pancreatic pseudocyst	• Metabolic—hypercalcaemia, hypoglycaemia, hyperlipidaemia
• Peritoneal fluid collection	• Haematological
• Pleural effusion	
• Pseudoaneurysm	
• Partial/splenic vein thrombosis	

Systemic complications

1. Shock

- Hypovolaemia and hypoperusion are the major actors responsible for renal failure. Due to collection of large amount of fluid in the third space-peritoneal cavity, pleural cavity and extravascular space, shock occurs. Fluid replacement with blood or albumin should be done at appropriate time to treat the shock.
- Electrolyte abnormalities should be corrected.

2. Respiratory insuficiency:

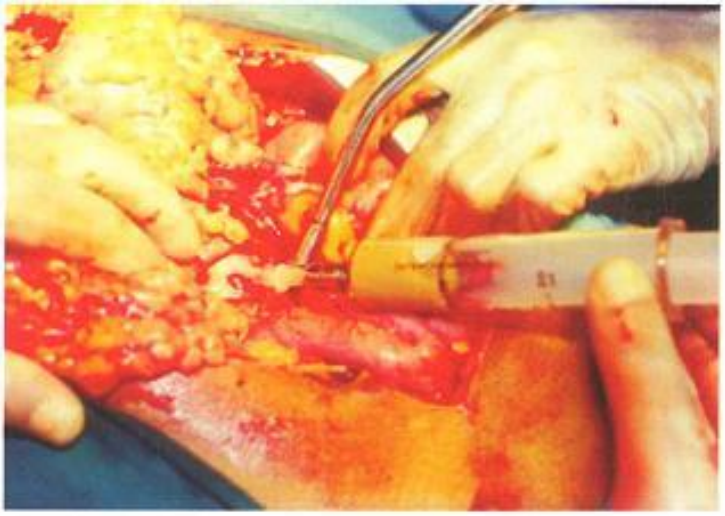


Measurement of arterial blood gas values and administration of oxygen is enough in the initial stages. In late stages, pulmonary insufficiency needs to be treated with ventilatory support.

3. Hypocalcaemia needs to be treated with calcium IV. It is due to hypoalbuminaemia and due to calcium soap.
4. Pleural effusion is treated by pleural tap (ultrasound- guided), if it is symptomatic.
5. ARDS, MODS: Some mediators such as phosphatase damage alveolar membrane of lungs causing 'ARDS'. It manifests as respiratory failure.

Local complications

1. Pancreatic abscess (Fig. 4)



Aspiration of pus from pancreatic abscess (Fig. 4)

- It develops after 3-4 weeks of pancreatitis. Secondary infection in a pseudocyst results in pancreatic abscess. It usually points out on the left flank.
- It has to be drained by CT-guided aspiration.
- Laparoscopy may be necessary not only or the diagnosi but also as a therapeutic means or removal of necrotic, pancreas.
- Otherwise, open drainage of the abscess is required.

2. Pseudocyst of pancreas (vide infra)

- This complication is encountered after 2nd week following an attack of acute pancreatitis.

- It is seen in about 20% of the patients.

3. Perforation of colon or stomach
4. Pseudoaneurysm resulting in massive upper gastro-intestinal or lower gastrointestinal bleeding. Bleeding into the pancreatic duct is called hemosuccus pancreaticus.
   - This condition occurs due to enzymatic digestion of the blood vessels in the vicinity of pancreas. Thus splenic artery, gastroduodenal artery, etc. are commonly involved.
   - It has very high mortality. Prompt angiography followed by embolisation is the treatment.
   - Otherwise, laparotomy, ligation of pseudoaneurysm with or without intracystic ligation of the bleeders is the treatment.

1. Following are true about anatomy of the pancreas except:
   A. 30% is by the head of pancreas
   B. 75% of islet cells are beta-cells producing insulin
   C. 80-90% pancreatic tissue is exocrine pancreatic tissue
   D. 5% of islet cells are A cells producing glucagon
2. Following are true or annular pancreas except:

A. It is more prevalent in children with Down syndrome
B. It surrounds 2nd part of duodenum
C. Duodenal obstruction causes vomiting in neonate
D. Gastrojejunostomy is the treatment of choice

3. The use of ultrasound in the diagnosis of acute pancreatitis is mainly to:
A. Rule out perforation
B. To look for oedematous pancreatitis
C. To rule out gall stones
D. To look for pancreatic stones

4. Role of CT scan in acute pancreatitis include following except:
A. To detect gall stones
B. Diagnostic uncertainty
C. To detect necrotizing pancreatitis
D. To find out the localized complication

5. Following are features of acute pancreatitis except:
A. Shock
B. Cullen's sign
C. Grey Tuner's sign
D. Kehr sign

6. Which one of the following is not routinely recom-mended in severe pancreatitis?
A. Aggressive fluid resuscitation
B. Nasogastric feeding
C. ICU monitoring with oxygenation
D. Antibiotic prophylaxis

7. Sudden rise in platelet counts in acute pancreatitis suggests:
A. Pancreatic fistula
B. Bleeding
C. Portal vein thrombosis
D. Pancreatic necrosis

8. Following are true about pseudocyst fluid except:
A. Ultrasound can easily detect pseudocyst
B. CEA levels are usually above 400 ng/ml
C. High amylase levels
D. Inflammatory cells in the aspirate

9. Following are true or tropical pancreatitis except:
A. Starts in young age
B. High incidence of stone formation
C. High incidence of diabetes mellitus
D. Does not predispose to pancreatic cancer

10. The best surgical treatment for chronic pancreatitis with dilated duct is:
A. Frey's operation
B. Whipple's operation
C. Longitudinal pancreaticojejunostomy
D. Distal pancreatectomy

ANSWERS
1-D. 2-D. 3-C. 4-A. 5-.D 6-B. 7-C. 8-B. 9-D. 10-C.

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