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FACULTY OF NURSING

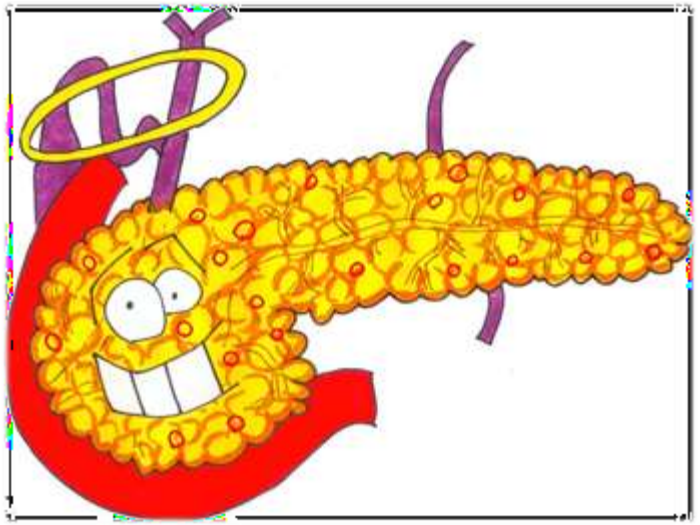
Acute Pancreatitis

BY:-
Kalpana Devi
Nursing Tutor
MSN Department
Rama College Of
Nursing

Pancreatitis

- Inflammation of the pancreatic parenchyma.
- Types:

1. Acute: Emergency condition.
2. Chronic: Prolonged & frequently lifelong disorder resulting from the development of fibrosis within the pancreas.

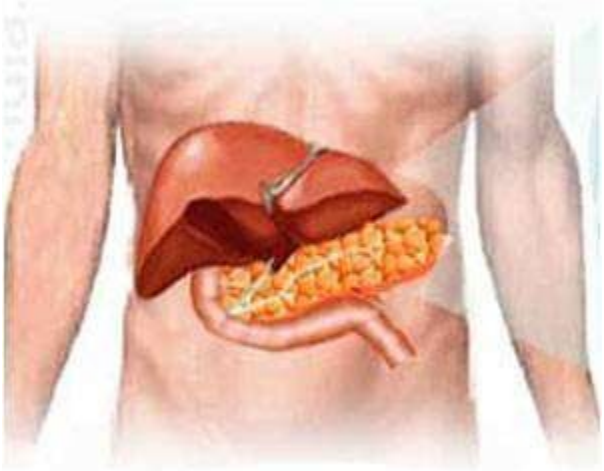


Acute Pancreatitis

- **Definition:**

  Acute condition of diffuse pancreatic inflammation & autodigestion, presents with abdominal pain, and is usually associated with raised pancreatic enzyme levels in the blood & urine.

•Reversible inflammation of the pancreas



- Ranges from mild to severe.

Epidemiology

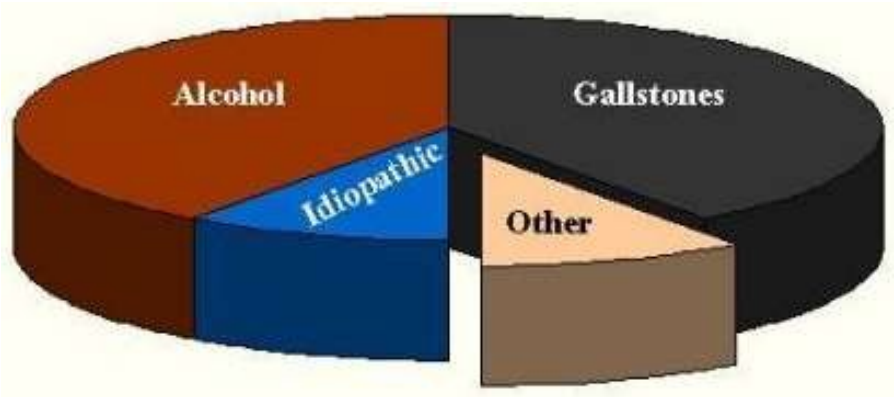
- Acute pancreatitis accounts for 3% of all cases of abdominal pain among patients admitted to hospital in the UK.
- Affect 2 – 28 per 100 000 of population.
- It may occur at any age, peak incidence is between 50 and 60 years.
- Women are affected more the men, but men are more likely to suffer recurrent attacks.

Etiology

- 80% of the cases are due to gallstones & alcohol.

- The remaining 20 % of cases are due to:

1. Congenital: Pancreatic divisum
2. Metabolic: Hyperlipidemia, Hypercalcemia.
3. Toxic: Scorpion venom
4. Infective: Mumps, Coxsackie B, EBV, CMV.



5. Drugs: Azathioprine, Sulfonamides, Steroids, Thiazides, Estrogens.
6. Vascular: Ischemia, Vasculitis (SLE, PAN).
7. Autoimmune: Hereditary pancreatitis.
8. Traumatic.
9. Miscellaneous: CF, Hypothermia, Periampullary Tumors.
10. Idiopathic.

BILIARY PANCREATITIS

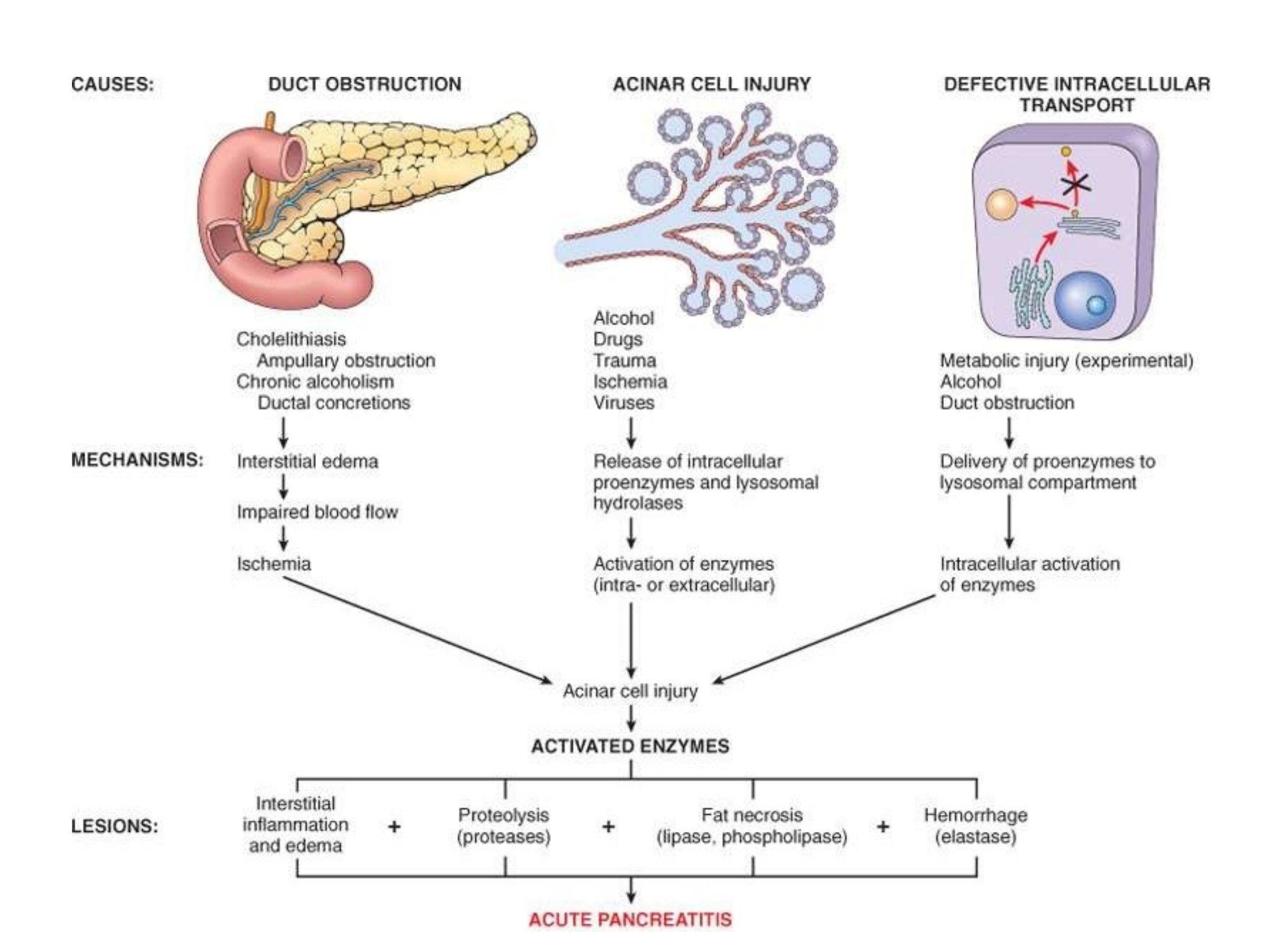
- **Biliary Pancreatitis:**

1. Common channel theory
2. Incompetent sphincter of Oddi
3. Obstruction of the pancreatic duct

Alcoholic Pancreatitis:

-Direct toxic effect on the pancreatic acinar cells

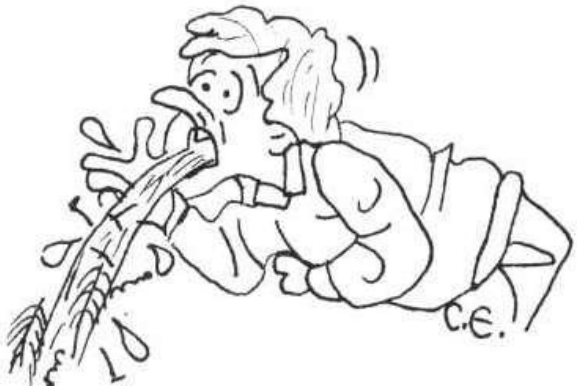
- Stimulation of the pancreatic secretion
- Constriction of the sphincter of Oddi


CAUSES:
DUCT OBSTRUCTION
ACINAR CELL INJURY
DEFECTIVE INTRACELLULAR TRANSPORT
Cholelithiasis
Ampullary obstruction
Chronic alcoholism
Ductal concretions
Alcohol
Drugs
Trauma
Ischemia
Viruses
Metabolic injury (experimental)
Alcohol
Duct obstruction
MECHANISMS:
Interstitial edema
Impaired blood flow
Ischemia
Release of intracellular proenzymes and lysosomal hydrolases
Activation of enzymes (intra- or extracellular)
Delivery of proenzymes to lysosomal compartment
Intracellular activation of enzymes
Acinar cell injury
ACTIVATED ENZYMES
LESIONS:
Interstitial inflammation and edema
+
Proteolysis (proteases)
+
Fat necrosis (lipase, phospholipase)
+
Hemorrhage (elastase)
ACUTE PANCREATITIS

Symptoms

- Upper Abdominal pain, sudden onset, sharp, severe, continuous, radiates to the back, reduced by leaning forward.

  Generalized abdominal pain, radiates to the shoulder tips. Patient lies very still.
- Nausea, non-projactile vomiting, retching
- Anorexia
- Fever, weakness



Signs

- Distressed, moving continuously, or sitting still
- Pale, diaphoretic. Confusion
- Low grade fever
- Tachycardia, Tachypnea
- Shallow breathing
- Hypotension
- Mild icterus

- Abdominal distension (Ileus, Ascites)
- Grey Turner’s sign, Cullen’s sign, Fox’s sign
- Rebound tenderness, Rigidity
- Shifting dullness, reduced bowel sounds

Cullen's Sign

Grey Turner's Sign

Fox's Sign

Panniculitis

- Subcutaneous nodular fat necrosis
- Tender red nodules
- Usually measures 0.5 – 2 cm
- Usually over the extremities

Differential Diagnosis

- ✓ Perforated viscus (DU)
- ✓ Acute cholecystits, Biliary colic
- ✓ Acute intestinal obstruction
- ✓ Esophageal rupture
- ✓ Mesenteric vascular obstruction
- ✓ Renal colic
- ✓ Dissecting aortic aneurysm
- ✓ Myocardial infarction
- ✓ Basal pneumonia
- ✓ Diabetic ketoacidosis

Investigations

Blood tests:

- Complete Blood Count
- Serum amylase & lipase
- C-reactive Protein
- Serum electrolytes
- Blood glucose
- Renal Function Tests
- Liver Function Tests
- LDH
- Coagulation profile
- Arterial Blood Gas Analysis

Serum Amylase:

- Sensitivity: 72% Specificity: 99%
- Released within **6-12 hours** of the onset, & Remains elevated for **3-5 days**.
- Elevation > 3X normal is significant.

- Undergoes renal clearance. After its serum levels decline, its urinary level remains elevated.
- Its **level doesn't correlate** with the disease activity.

Serum Lipase:

- More pancreatic-specific than s. Amylase.
- Sensitivity: about 100% Specificity: 96%
- Remains **elevated longer** than amylase (up to week).
- Useful in patients presenting late to the physician.

- **S. Amylase** tends to be higher in **gallstone pancreatitis**
- **S. Lipase** tend to be higher in **alcoholic pancreatitis**

OTHER PANCREATITIC ENZYME

- These include phospholipase A, trypsin, carboxylester lipase, carboxypeptidase A, and co-lipase.

- None of these, alone or in combination, has a significant clinical advantage over amylase and lipase.

Imaging Investigations:

- **Plain erect chest X-ray:** not diagnostic on pancreatitis, but to rule out other D/D
- Pleural effusion, diffuse alveolar infiltrate (ARDS)

RADIOLOGY

RADIOLOGY- Abdominal plain film

- helps to exclude other causes of abdominal pain such as obstruction and bowel perforation.
- range from unremarkable in mild disease to localized ileus of a segment of small intestine ("sentinel loop") or the "colon cutoff sign" in more severe disease.
- The latter reflects a paucity of air in the colon distal to the splenic flexure due to functional spasm of the descending colon secondary to spread of pancreatic inflammation to that area.
- Generalized ileus may occur in severe disease.
- A ground glass appearance may indicate ascites.

ABDOMINAL ULTRASOUND

Abdominal ultrasound

- A diffusely enlarged, hypoechoic pancreas is the classic ultrasonographic image of acute pancreatitis.
- It can also detect gallstones in the gallbladder and biliary tree.
- 25 to 35 percent of patients have bowel gas that may obscure the pancreas.
- It cannot clearly delineate extrapancreatic spread of pancreatic inflammation or identify necrosis within the pancreas; these important findings are best seen by CECT

- **CT Scan:** not indicated in every patient, **only in:**

1. Diagnostic uncertainty.
2. Severe acute pancreatitis.
3. Clinical deterioration, with multi-organ failure, sepsis, progressive deterioration.
4. Local complications occurs (fluid collection, pseuodocyst, pseudo-aneurysm).

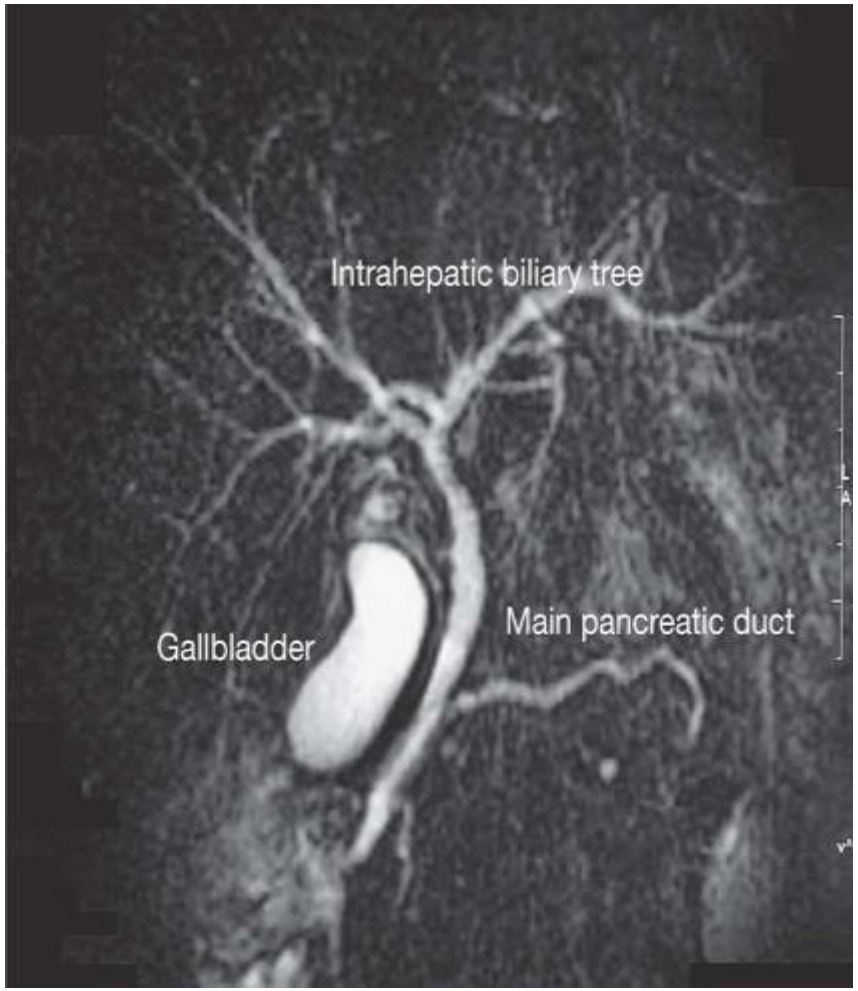
MAGNETIC RESONANCE IMAGING

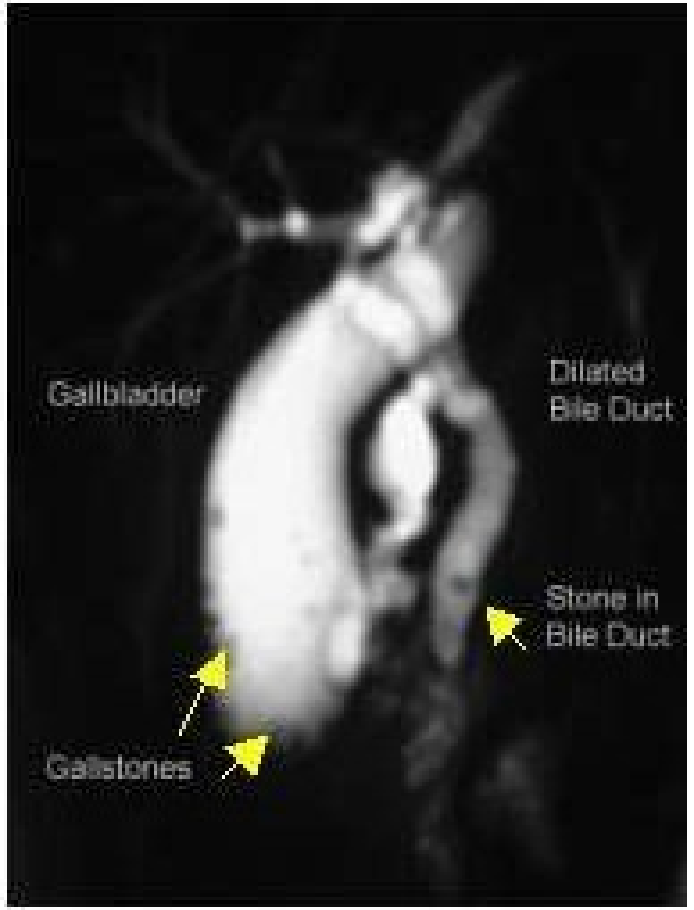
- MRCP, both enhanced and non-enhanced, has a strong correlation with contrast-enhanced CT in acute pancreatitis.

- The advantages of MRI over CT include: lack of nephrotoxicity of gadolinium, ability of MRI to better categorize fluid collection as acute fluid collections, necrosis, abscess, hemorrhage, and pseudocyst , and the greater sensitivity of MRI to detect mild acute pancreatitis compared to CT.

- MRCP delineates the pancreatic and bile ducts better and is comparable to ERCP for the detection of choledocholithiasis.

MRCP


Intrahepatic biliary tree
Gallbladder
Main pancreatic duct

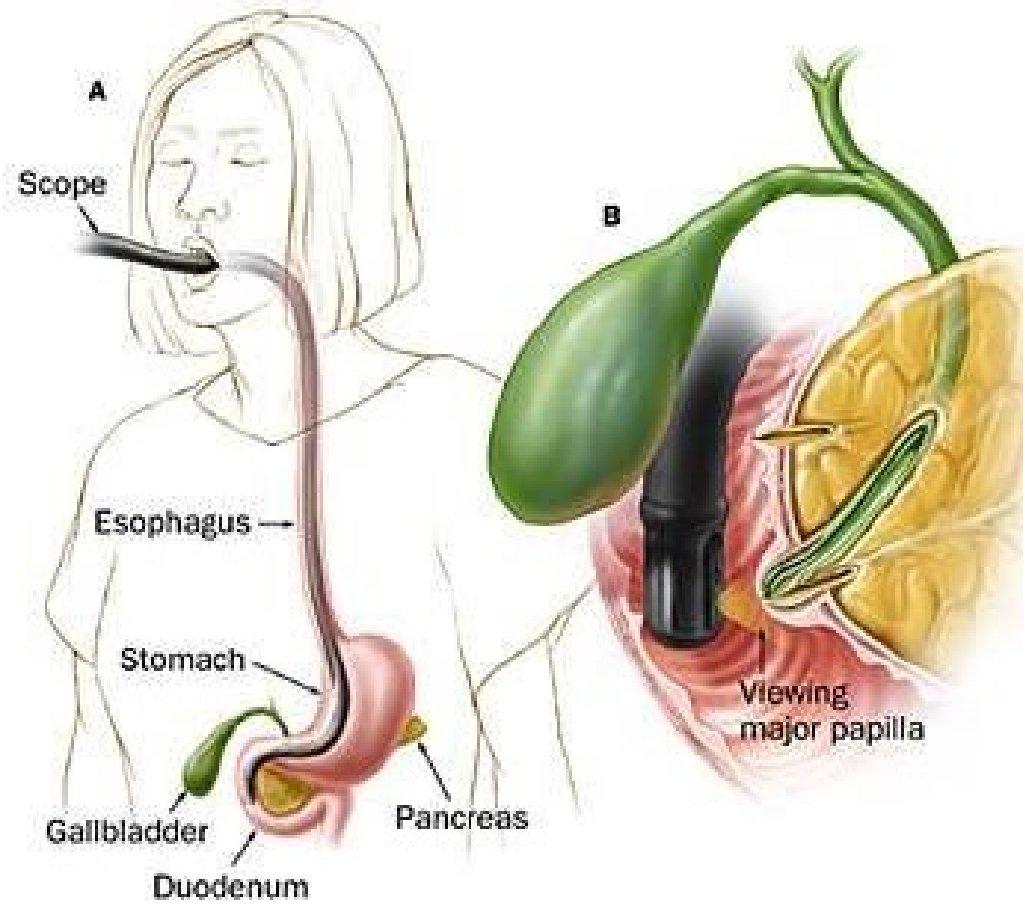

Gallbladder
Dilated
Bile Duct
Stone in
Bile Duct
Gallstones

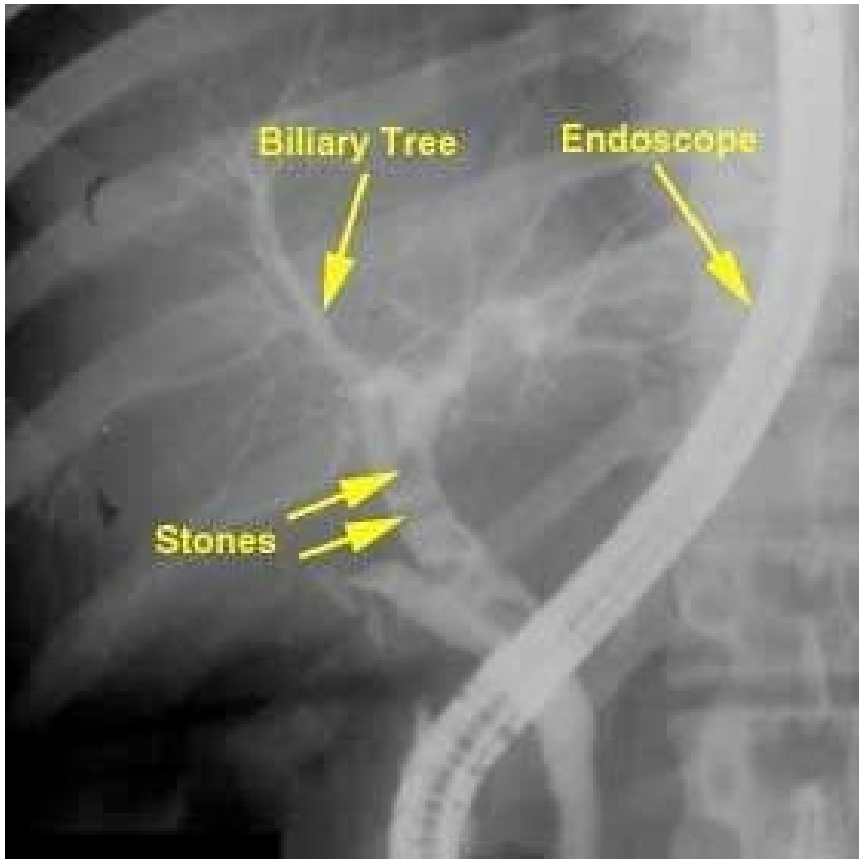
- **Endoscopic Ultrasound, MRCP:** CBD stones detection, assessment of pancreatic parenchyma. Not widely available.

- **ERCP:** CBD stones identification & removal.

  Urgent ERCP in severe acute gallstone pancreatitis & signs of ongoing biliary obstruction & cholangitis.

ERCP


A
Scope
Esophagus
Stomach
Gallbladder
Duodenum
Pancreas
B
Viewing
major papilla


Biliary Tree
Endoscope
Stones

Goals of Treatment

- Aggressive supportive care
- Decrease inflammation
- Limit superinfection
- Identify and treat complications

(of pancreatitis & its treatment)

- Treat cause if possible

Conservative Management

- Gain IV access, obtain blood sample, rapid fluid resuscitation & electrolytes replacement.
- Give analgesics (IM pethidine).
- Give Anti-emetics.
- Keep the patient NPO (until pain free/2-3 days).
- NGT insertion to relieve vomiting.

- Urinary catheterization is done.
- Monitor the vital signs.

- Injection Ranitidine 50 mg IV 8 hourly, or Omeprazole 40 mg IV BD.
- Somatostatin or octreotide (pancreatic secretions inhibitors).
- Respiratory support: oxygen supplementation, or Venti mask
- ICU admission if severe acute pancreatitis.

Table 64.4 Early management of severe acute pancreatitis

Admission to HDU/ICU
Analgesia
Aggressive fluid rehydration
Oxygenation
Invasive monitoring of vital signs, central venous pressure, urine output, blood gases
Frequent monitoring of haematological and biochemical parameters (including liver and renal function, clotting, serum calcium, blood glucose)
Nasogastric drainage
Antibiotic prophylaxis can be considered (imipenem, cefuroxime)
CT scan essential if organ failure, clinical deterioration or signs of sepsis develop
ERCP within 72 hours for severe gallstone pancreatitis or signs of cholangitis
Supportive therapy for organ failure if it develops (inotropes, ventilatory support, haemofiltration, etc.)
If nutritional support is required, consider enteral (nasogastric) feeding

Role of Antibiotics

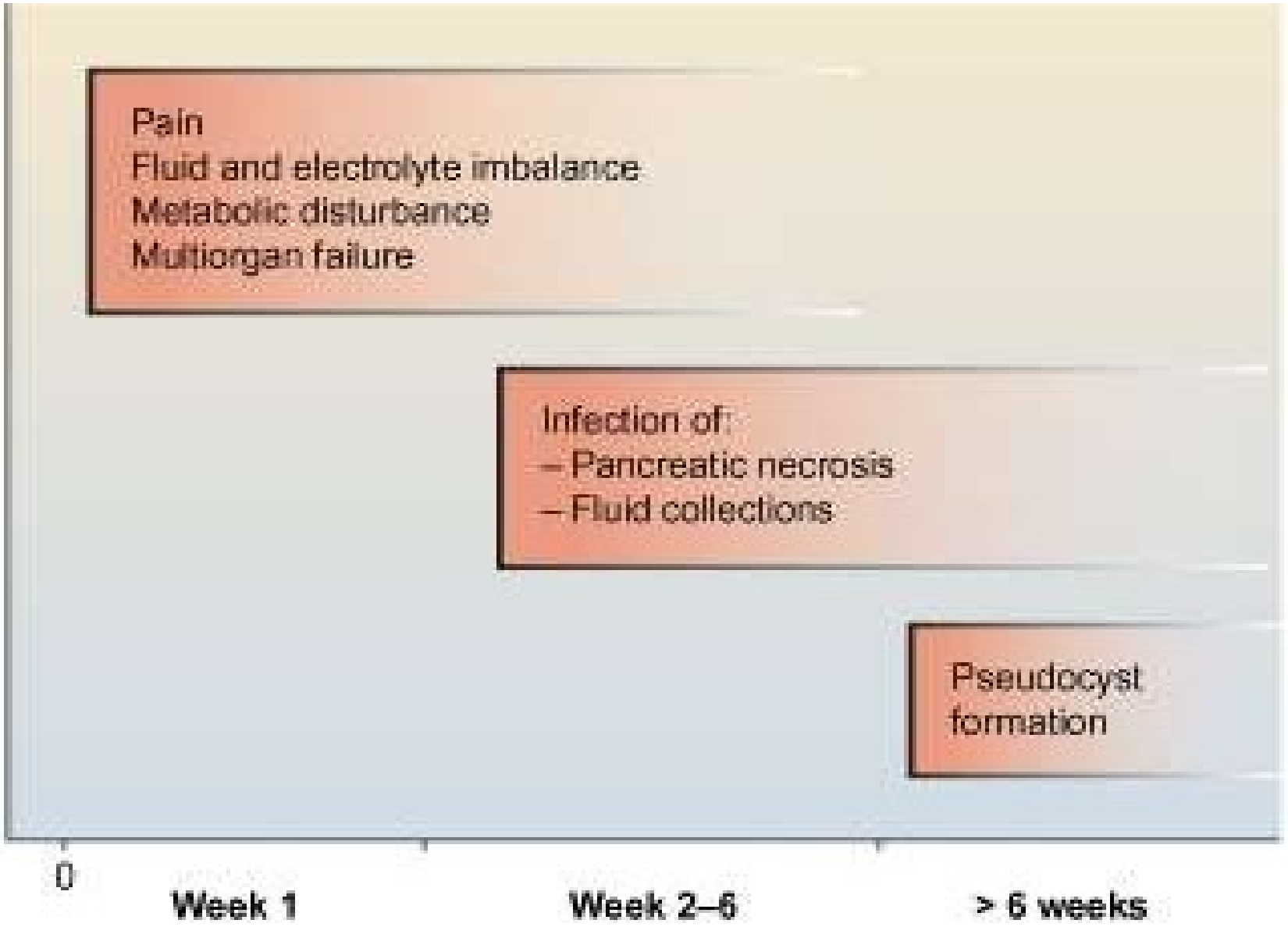
- Prophylactic antibiotics have shown **No decrease** in mortality in severe acute pancreatitis.
- **<u>Antibiotics are justified if:</u>**

1. Gas in retroperitoneal space
2. Needle aspiration of necrotic material confirms infection
3. Sepsis
4. CRP of > 120 mg/L
5. Peri-pancreatic fluid collection
6. Organ dysfunction
7. APACHE II Score of > 6

Operative Management

- Surgery has **no immediate role** in acute pancreatitis.
- Aggressive surgical pancreatic debridement (Necrosectomy) should be undertaken soon after confirmation of the presence of infected necrosis.
- Pseudocyst: Cystogastrostomy, Cystodudenostomy, Roux-en-Y cystojejunostomy.

Complications



COMPLICATIONS

Systemic Complications:

- **Cardiovascular:** Shock, Arrhythmias, Pericardial effusion
- **Pulmonary:** Basal atelactasis, pleural effusion, ARDS
- **Renal:** ATN, Renal failure
- **Haematological:** DIC
- **Metabolic:** Hypocalcemia, Hyperglycemia, Hyperlipidemia
- **GIT:** Ileus
- **Neurological:** Confusion, Irritability, Encephalopathy
- **Miscellaneous:** Subcutaneous fat necrosis, Arthralgia

Local complications (Occurs after the 1st week)

Acute fluid collection
Sterile pancreatic necrosis
Infected pancreatic necrosis
Pseudoaneurysm
Pancreatic abscess
Splenic vein thrombosis

Sterile and infected pancreatic necrosis:

- Diffuse or focal area of non-viable parenchyma, typically associated with peripancreatic fat necrosis. These areas can be identified by an absence of contrast enhancement on CT.
- They’re sterile to begin with, but can become subsequently infected, due to the gut bacterial translocation.
- Sterile necrotic material should not be drained or interfered with.
- If the patient shows signs of sepsis, then one should determine whether the necrosis is infected.

Mortality

- Mild acute pancreatitis: Mortality rate of 1%
- Severe pancreatitis: Mortality rate of 75-90%
- Overall mortality rate of 15-20%

- First week of illness -> MODS
- Subsequent weeks -> infection

THANK YOU