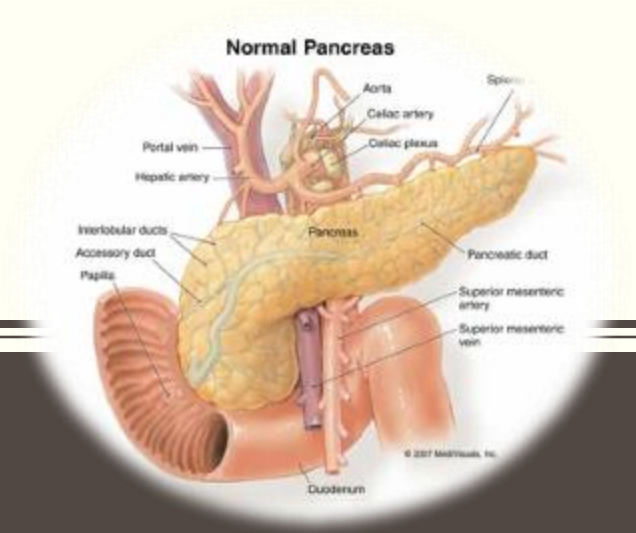
PANCREATITIS A22 SEMINAR 1



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Content Layout

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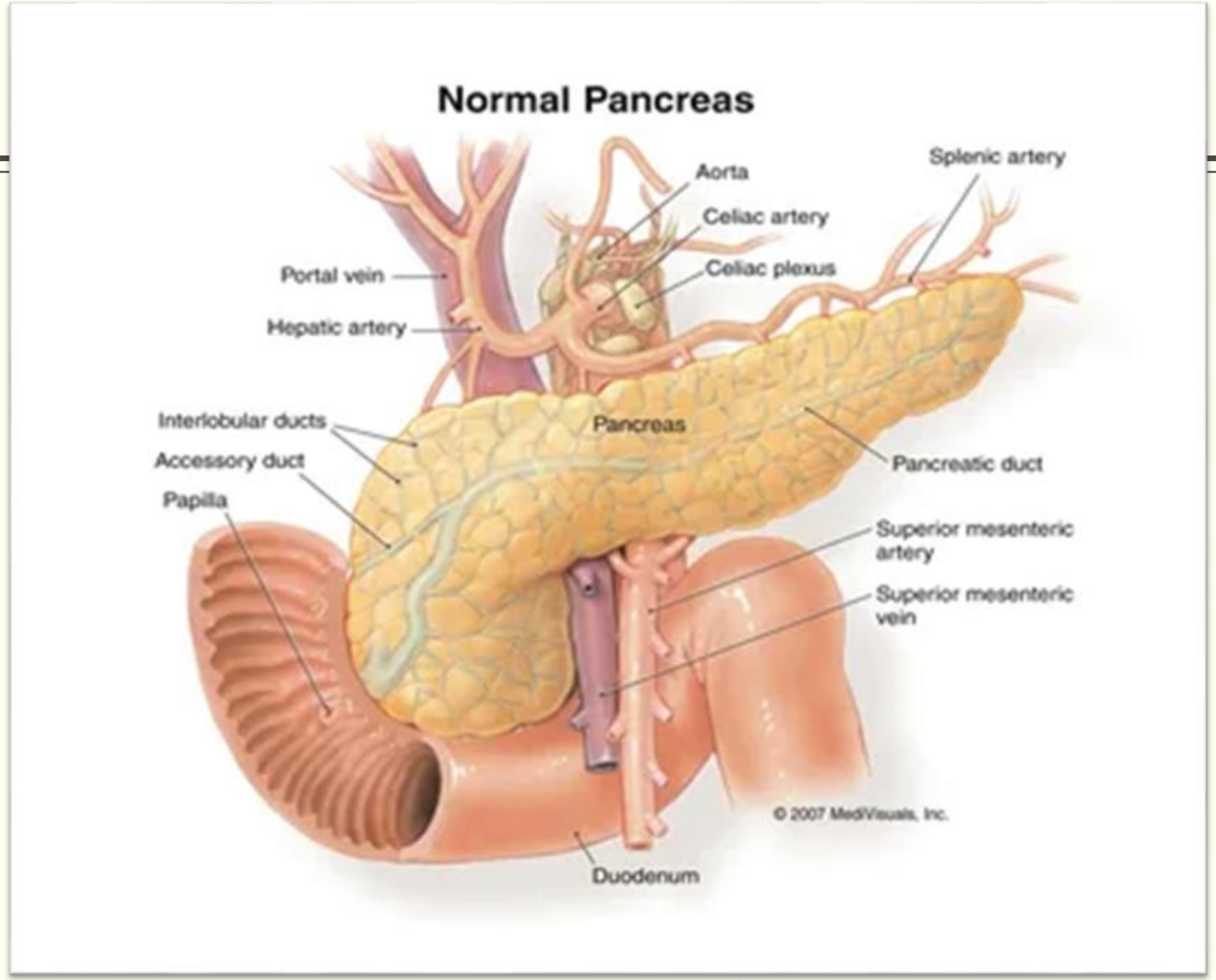
- Definition
- Epidemiology and etiology
- Pathogenesis
- Sign & symptoms
- Investigations
- Management
- Complications


Normal Pancreas
Aorta
Splenic artery
Celiac artery
Celiac plexus
Portal vein
Hepatic artery
Interlobular ducts
Pancreas
Accessory duct
Pancreatic duct
Papilla
Superior mesenteric artery
Superior mesenteric vein
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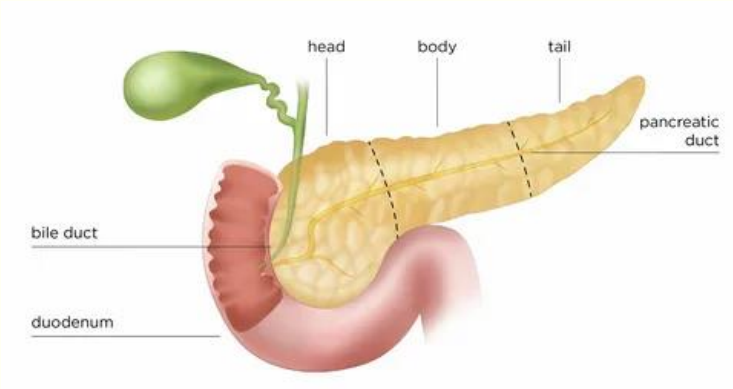
Duodenum

Intro ; Pancreas revision

- The Pancreas is an accessory organ and it weighs about 90grams with12-15 cm long.
- It lies behind the stomach and lesser sac and wraps around superior mesenteric artery and vein.
- It is supplied by the pancreatic branches of the splenic artery.
- The pancreas has endo & exo-crine function



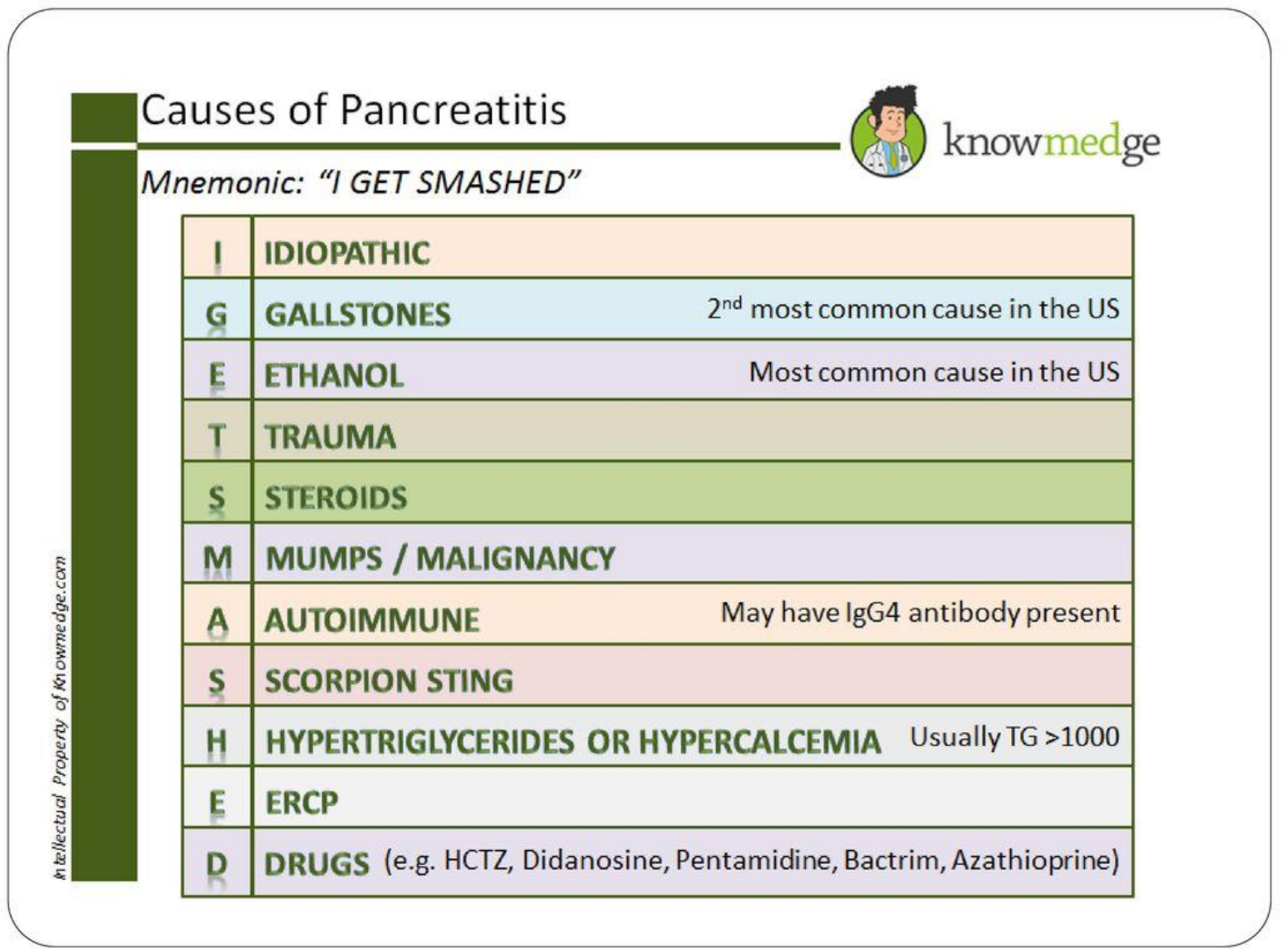
Endocrine	Exocrine
Alpha cells – Glucagon Beta cells – insulin Gama cells – polypeptides Delta cells – somatostatin	Alpha amylase – starch digestion Lipase – fat digestion Proteases – protein digestion

Acute pancreatitis

- Acute inflammation of pancreas due to insult that lead to premature activation of pancreatic enzymes and autodigestion of peripancreatic tissues.
- An acute condition presenting with abdominal pain, a three fold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/ or characteristic findings of pancreatic inflammation on contrast- enhanced CT.

Epidemiology and etiology

- The two major causes of acute pancreatitis are biliary calculi, which occur in 50–70% of patients, and alcohol abuse, which accounts for 25% of cases



Etiology

I GET

- Idiopathic
- Gallstones
- Ethanol
- Trauma

SMA

- Scorpion bite
- Mumps \ Metabolic
- Autoimmune

SH

- Steroids
- Hyperlipidemia – hypercalcemia

ED

- ERCP
- Drugs
  - Estrogen
  - Corticosteroids
  - Azathioprine
  - Valproic
  - thiazide

Chronic pancreatitis

- a progressive inflammatory disease in which there is irreversible destruction of pancreatic tissue.
- Its clinical course is characterized by severe pain and, in the later stages, exocrine and endocrine pancreatic insufficiency.
- Causes :

1. High alcohol consumption is the most frequent cause of chronic pancreatitis, accounting for 60–70% of cases
2. pancreatic duct obstruction
3. Genetic
   - autoimmune

CHRONIC PANCREATITIS

Clinical features

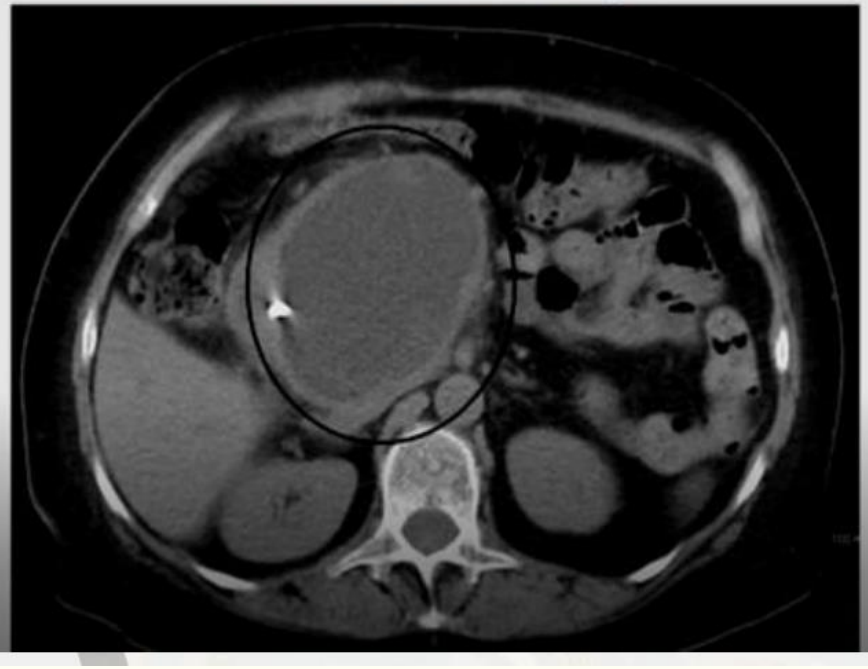
- Pain
  - Nausea and vomiting
  - Weight loss
- Impact on the social life
  - Steatorrhea
  - DM

Pseudocyst

- Usually occurs **more than 4 weeks** after acute episode
- Walled-off collection of edema/fluid
  - Contain minimal or no necrosis
- "Pseudo:" no epithelium
  - Granulation/fibrous tissue surrounds fluid
- Usually outside the pancreas
- Diagnosed by CT or MRI imaging
- Most common location is lesser sac
  - Posterior to stomach

•Usually resolves without intervention if not → drainage.

Pancreatic Pseudocyst



PATHOGENESIS

Acute pancreatitis

The two types of acute pancreatitis:

1. Mild pancreatitis: the main response to the injury is inflammation (edema)
2. Severe pancreatitis: the main response to the injury is necrosis (to the pancreas and nearby organs)

- THE MAIN CAUSE TO ACUTE PANCREATITIS IS OBSTRUCTION OF AMPULLA OF VATER :
- The obstruction leads to flow back of pancreatic enzymes to the pancreas parenchyma (proteolytic enzymes and lipase)

Acute pancreatitis

These enzymes when reach the pancreas leads to :

- Destruction of pancreatic parenchyma by proteolytic enzymes
- Destruction of blood vessels leading to interstitial hemorrhage
- Microvascular leakage causing edema
- Necrosis of fat by lipases

Chronic pancreatitis

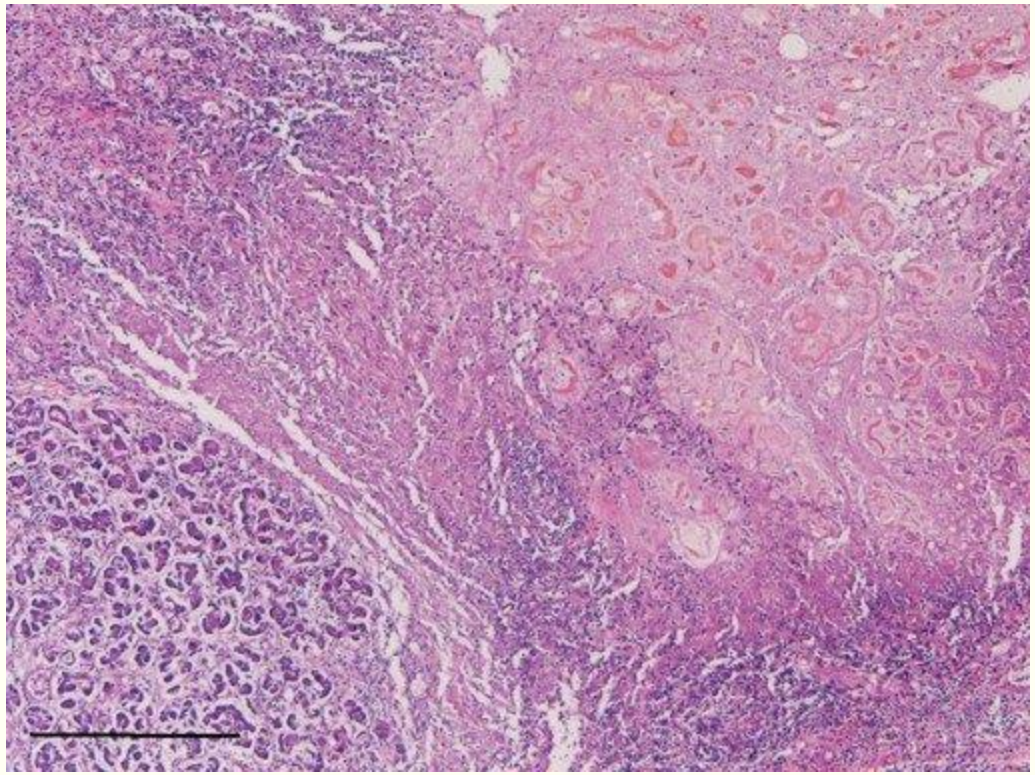
Chronic pancreatitis is characterized by long-standing inflammation that leads to irreversible destruction of the exocrine pancreas, then endocrine function .

- Most common cause of chronic pancreatitis is long-term alcohol abuse.
- characterized by:

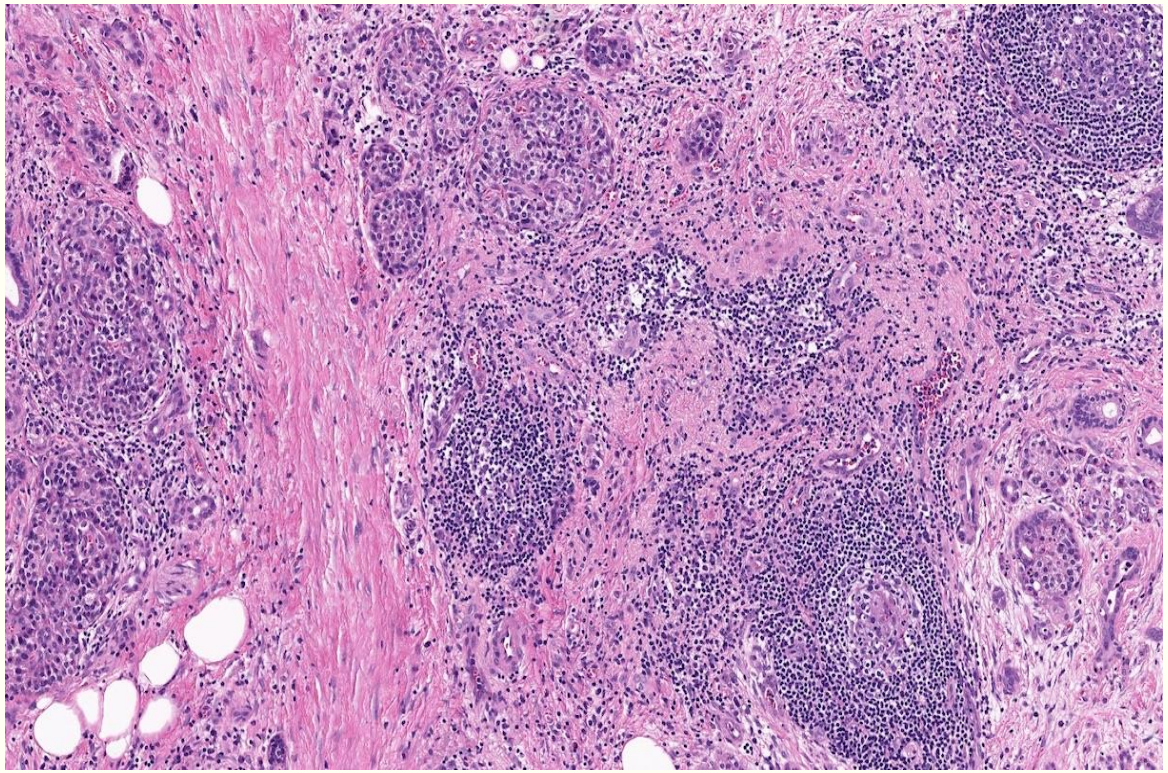
1. Loss of acinar
2. Parenchymal fibrosis

Histology of pancreatitis

Acute pancreatitis



Chronic pancreatitis

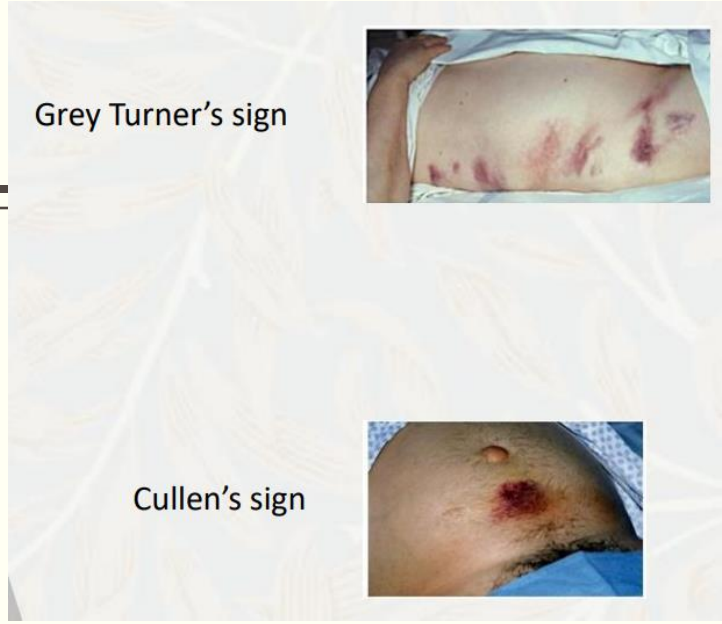


Signs and symptoms

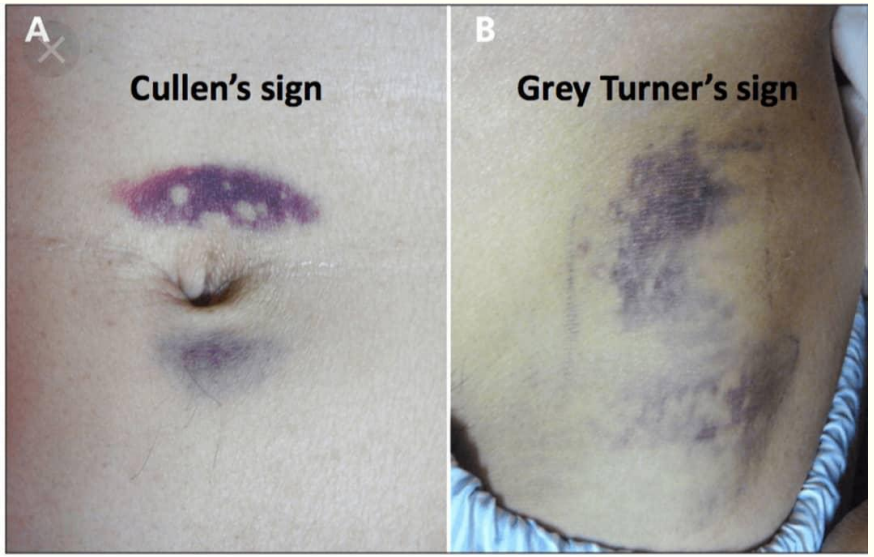
●Symptoms:

- Epigastric abdominal pain, radiating to the back, and relieved by leaning forward (because it's a retroperitoneal organ and leaning forward pulls the peritoneum anteriorly, decreasing the pressure on retroperitoneal organs and relieving the pain).

- Nausea, vomiting, bloating, low grade fevers, and anorexia



- Signs :
 -tachycardia, epigastric tenderness, distension ,
 flank ecchymosis (Grey–Turner sign), periumbilical ecchymosis (Cullen sign), bluish discoloration of the inguinal ligament (Fox's sign) ,
and shock.



HOW TO MAKE A DIAGNOSIS

History and physical examination
Labs
Imaging
Continue until you reach a diagnosis

PRESENTATION

Presentation

- **Pain** is the cardinal symptom.
- Nausea, repeated vomiting and retching are usually marked accompaniments.
- On examination, look for vital signs and signs of dehydration , jaundice , abdominal distention and guarding , gray turner sign and Cullen's sign , signs of pleural effusion and pulmonary edema , CNS exam looking for confusion secondary to hypoxemia)

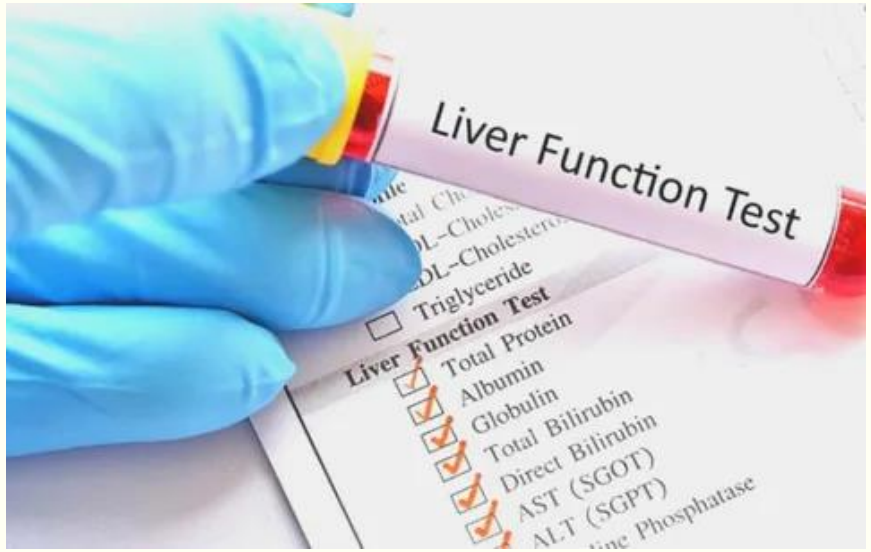
INVESTIGATIONS

· Typically, the diagnosis is made on the basis of the clinical presentation and an **elevated serum amylase level** ·

1. Serum amylase : ▪ Normal level of amylase = 80 U/L. If >3x of normal this is significant for pancreatitis, less than this, it still could be pancreatitis, but it is not specific as it is increased in other pathological causes (Cholecystitis ,IBD, stress, perforated peptic ulcer). ▪ It is elevated only for 2-3 day after the onset.
2. **Serum lipase**: Increased and **more specific than amylase**. ▪ The level of amylase or lipase is not related to severity of pancreatitis .

Lab tests

· CBC- Anemia(hgic), leukocytosis (inflammation, infection)·
Liver enzymes - ALT if increases more that 150 U/L probably dogallstones·
Serum electrolytes, BUN, creatinine- Low Ca2+·
Blood glucose, cholesterol, triglycerides- Blood glucose high to B-cell injury·
ABG- respiratory distress

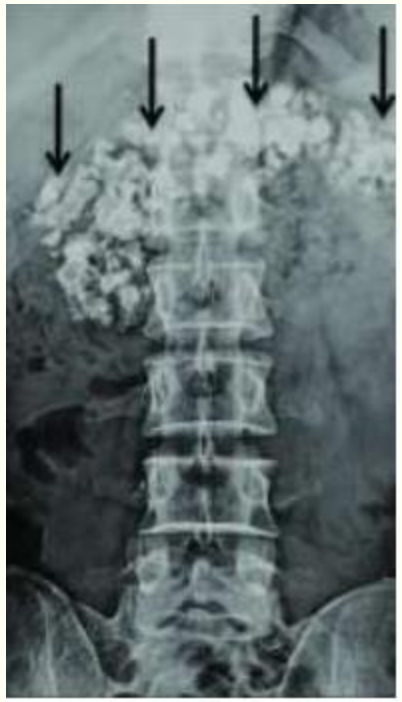


IMAGING

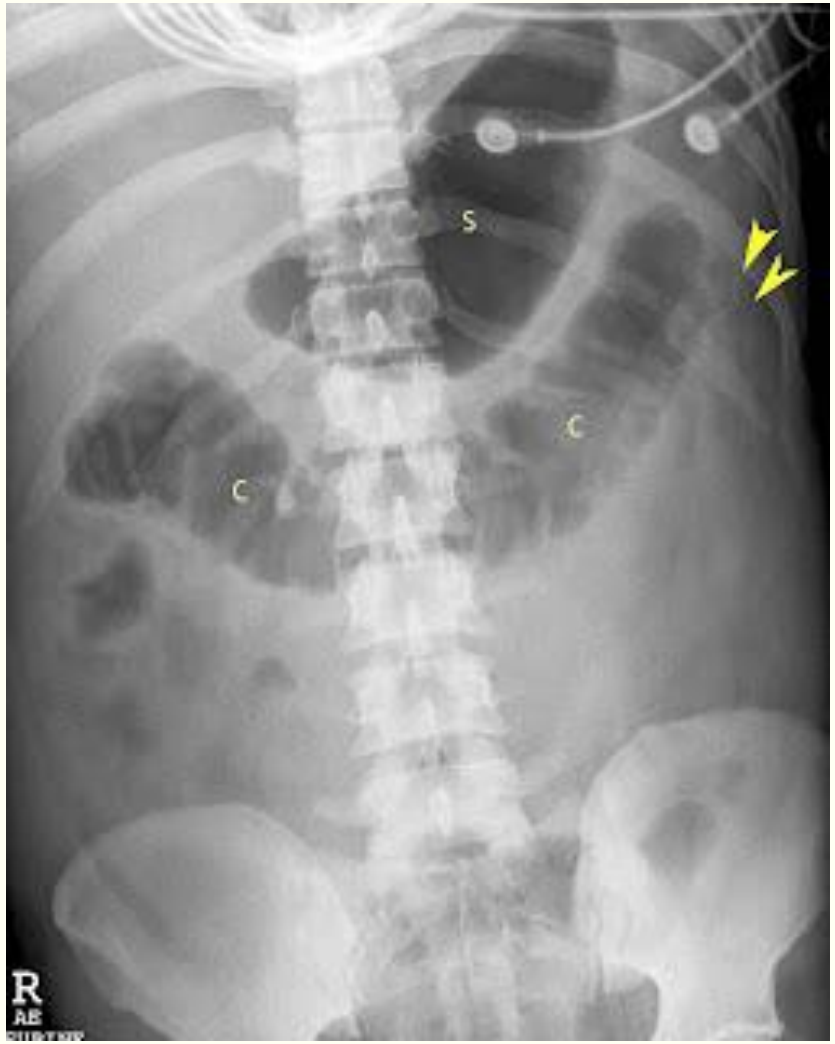
1. CT scan with IV contrast (main investigation in any pancreatic disease) may show pancreatic edema, pancreatic necrosis, gall bladder stones, intra-peritoneal fluid and enlargement of the pancreas & later on pancreatic pseudocyst.
2. Abdominal U/S : show G.B stones and dilatation of biliary passage above CBD stone .
3. Magnetic resonance cholangiopancreatography (MRCP) to detect any pathology in the biliary passage.
4. Diagnostic ERCP after subside of the attack of biliary pancreatitis

X RAY

- Not diagnostic of acute pancreatitis but are useful in the differential diagnosis.

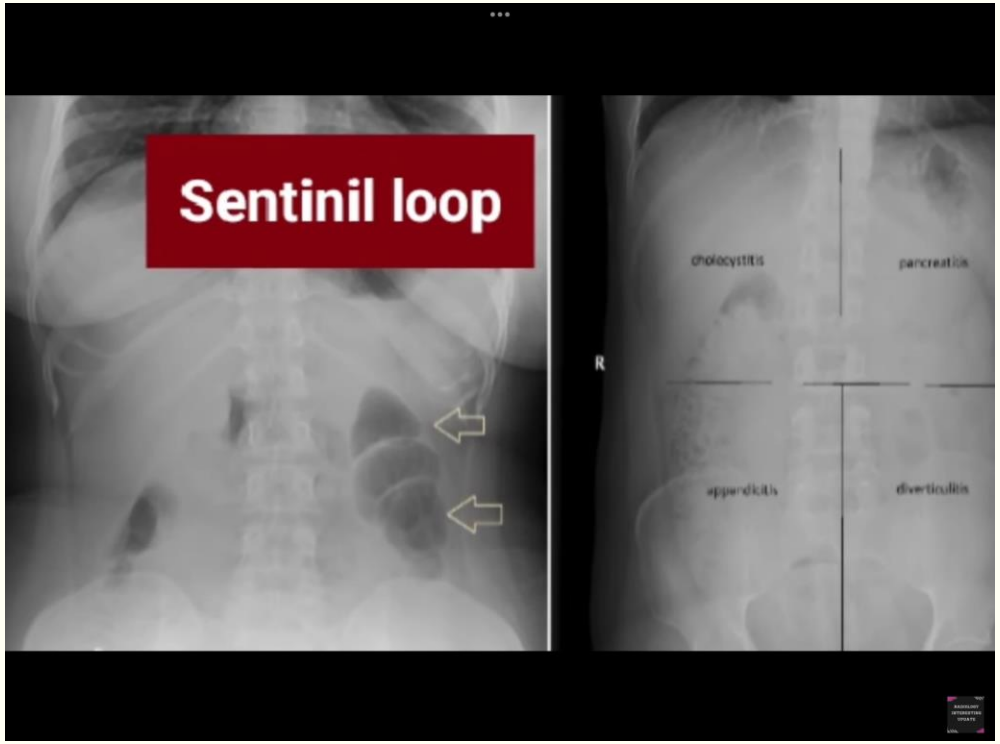


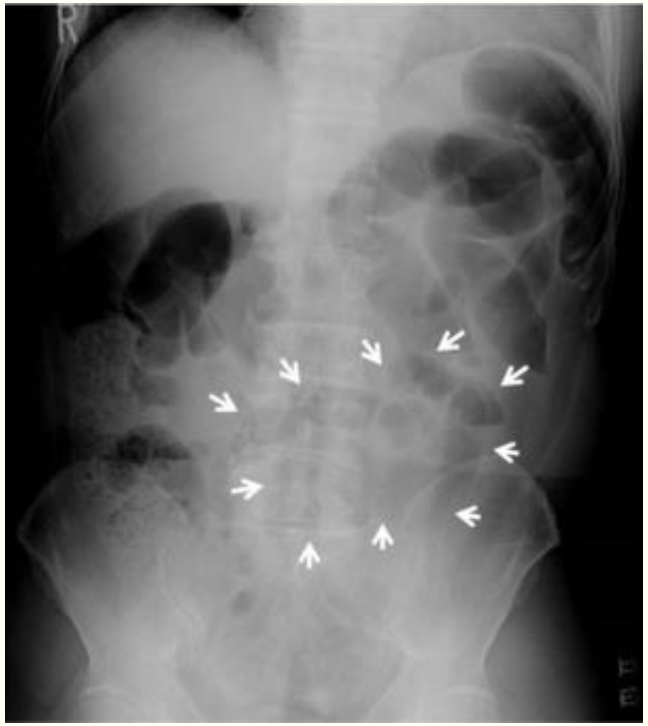
Abrupt cut off of colonic gas column at the splenic flexure (arrow). The colon is usually decompressed beyond this point.



Inflammatory exudate in acute pancreatitis extends into the phrencocolic ligament via lateral attachment of the transversemesocolon
Infiltration of the phrenicocoliic ligament results in functional spasm and/or mechanical narrowing of the splenic flexure at the level where the colon returns to the retroperitoneum.

# Indicates localized ileus from nearby inflammation

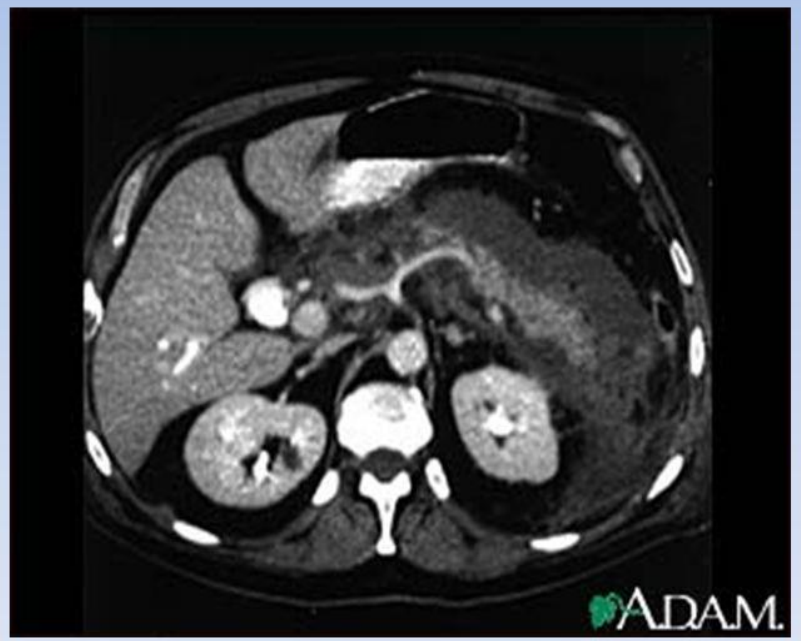


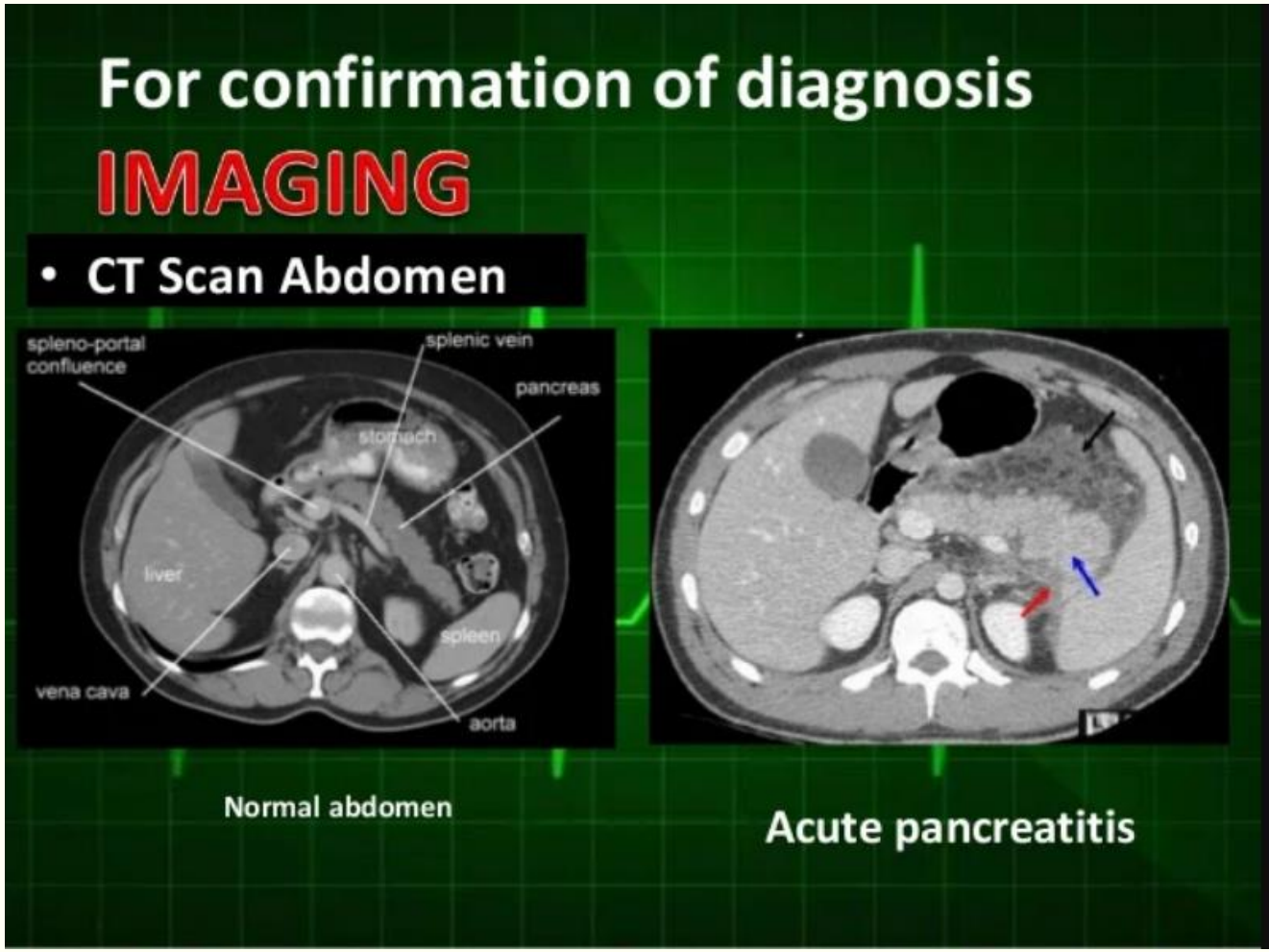


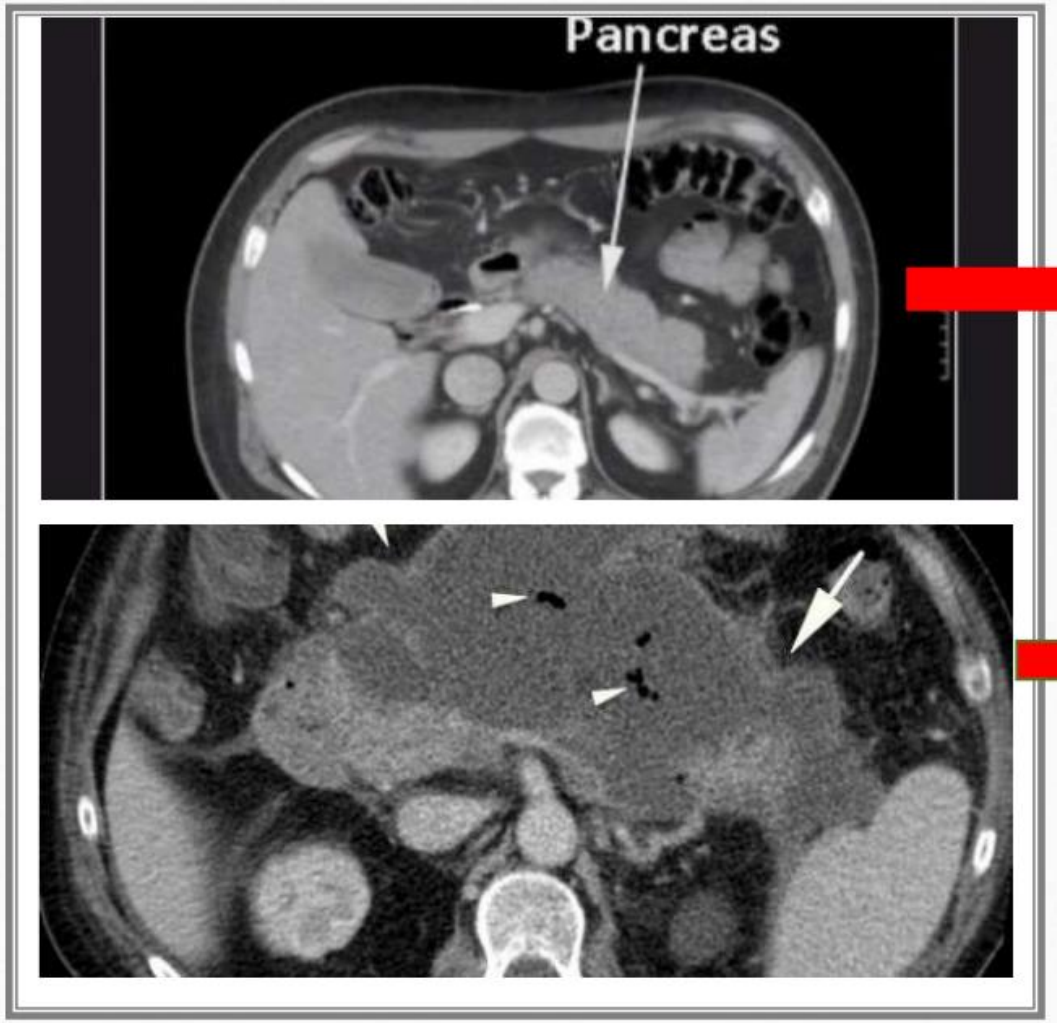
CT scan

CT Scan of acute pancreatitis

- CT shows significant swelling and inflammation of the pancreas







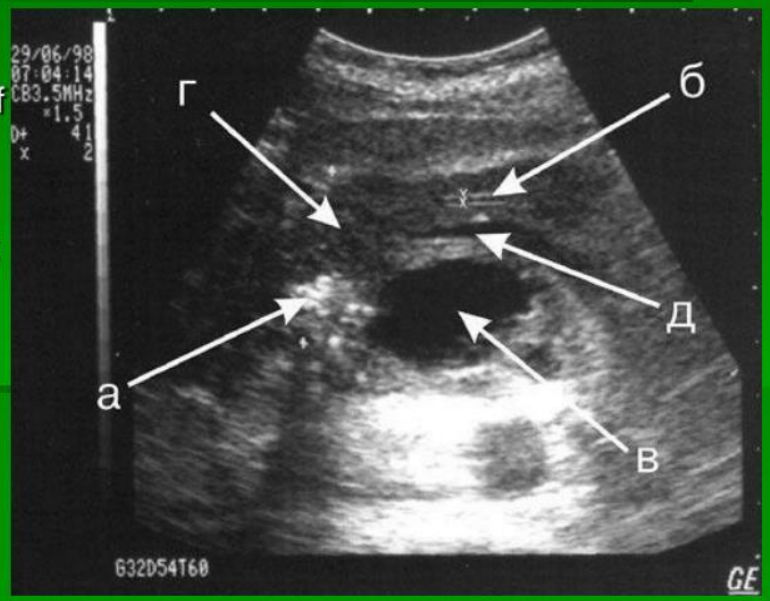
Normal pancrease

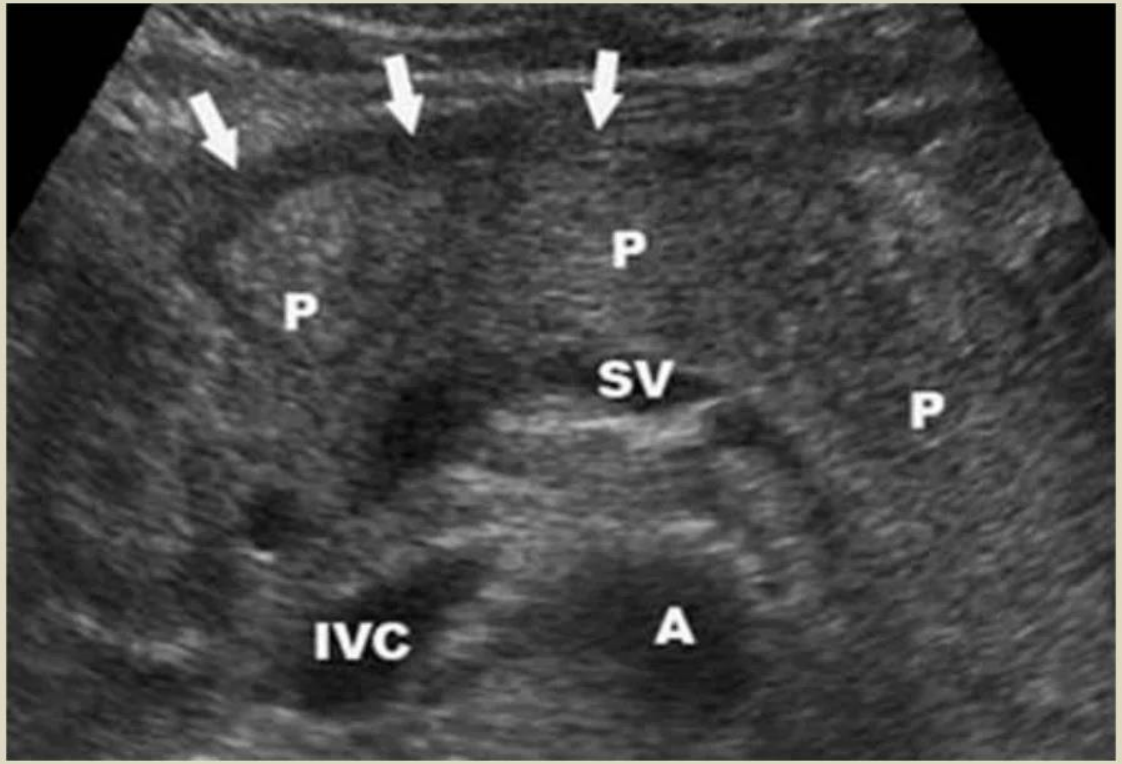
Infected pancreatic necrosis

Ultrasound

Ultrasound investigation. Chronic pancreatitis

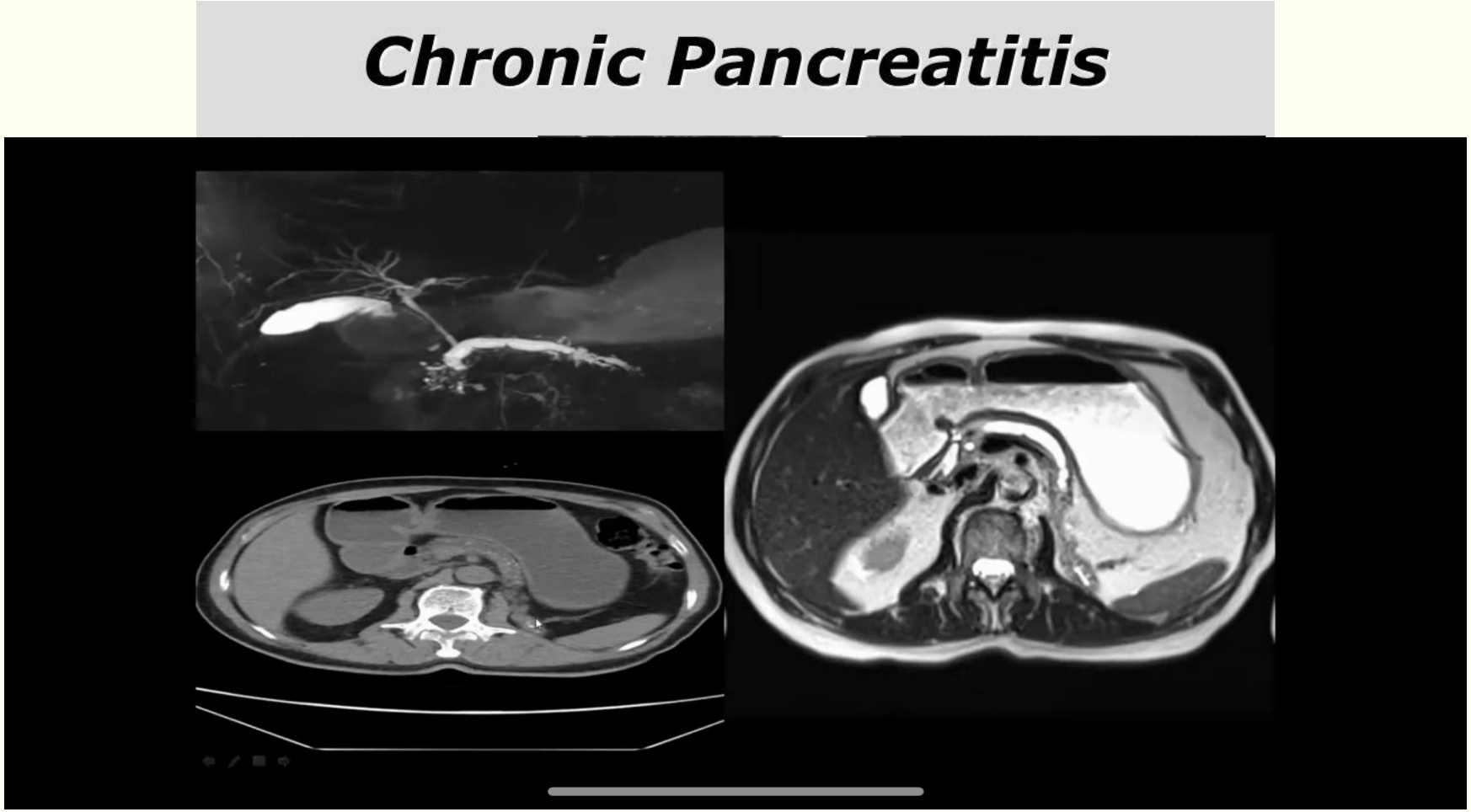
а) calcificates in the head of pancreas;
б) Virsungov's duct;
в) pseudocyst of pancreas;
г) increase of the head of pancreas;
д) spleen vein





Acute pancreatitis - Pancreatic gland (P) is edematous and there is a fluid visible in front of the pancreas. (Black anechogenic strip marked by arrows). From other anatomical structures we see splenic vein (SV), aorta (A) and inferior vena cava (IVC).

MRCP



1 Ductal dilatation from head to tail

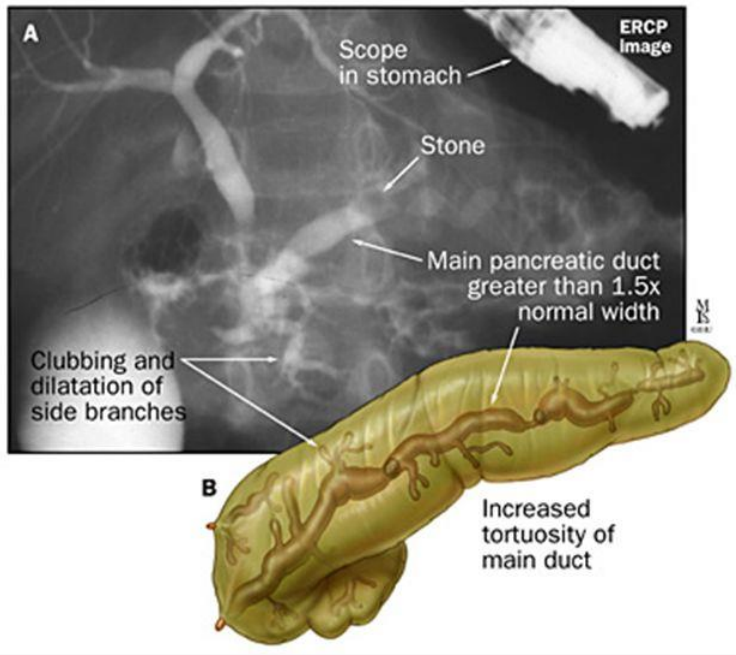
2 Calcifications in the pancreatic parenchyma

3 significant parenchymal loss on either sides of the pancreas with significant atrophy

ERCP

Chronic Pancreatitis

Endoscopic retrograde cholangiopancreatography (ERCP)



A characteristic ***"chain of lakes"*** appearance of the main pancreatic duct can be noted on ERCP in patients with ***severe chronic pancreatitis***.

The main pancreatic duct is enlarged (greater than 1.5 times) with increased tortuosity.

There is severe clubbing and dilation of the side branches.

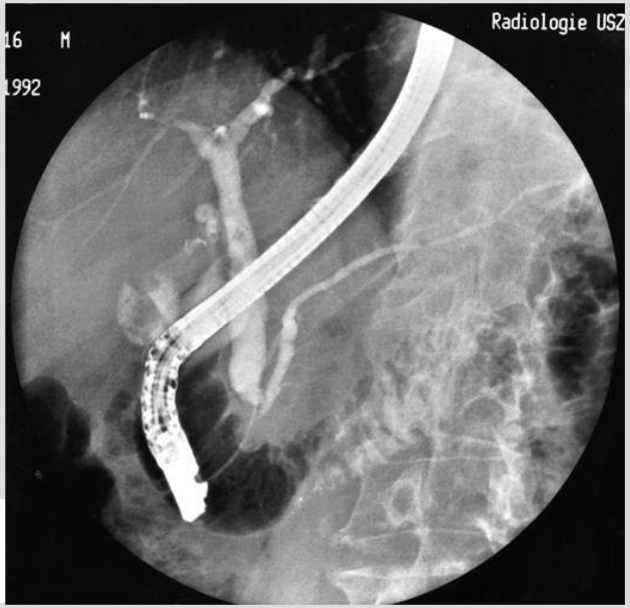
Stone formation and occlusion of the pancreatic duct may occur in this stage of the disease

Acute Pancreatitis

Endoscopic retrograde cholangiopancreatography (ERCP)

ERCP allows identification and removal of common-bile-duct stones in ***suspected gallstone pancreatitis***.

Because of its invasive nature and the inherent risk of worsening pancreatitis, it should be performed only in the setting of ongoing biliary obstruction and cholangitis.



Slightly dilated common bile duct with calculus and normal pancreatic duct are shown

ERCP is diagnostic and therapeutic approach

ERCP

FOR DIAGNOSIS ;
YOU NEED AT LEAST TWO OUT OF THREE DIAGNOSTIC CIRTERIA

1

EPIGASTRIC PAIN

2

Elevated amylase or lipase > 3x upper limit of normal

3

Abnormal pancreatic imaging (CT)

SEVERITY SCORING FPR PANCREATITIS

- Ranson scoring system
- Modified Glasgow Criteria
- Acute physiology and chronic health evaluation (APACHE)-II Scoring System
- CRP (48-hrs. or 24-hrs.) [≥130 mg/ml = severe, <130 mg/ml = mild]
- balthazar Grading System for Acute Pancreatitis (CT scan based)

NOTE : The Ranson and Glasgow scoring systems to predict the severity of acute pancreatitis: in both systems, disease is classified as severe when three or more factors are present

Table 33-7

Ranson's prognostic signs of pancreatitis

Criteria for acute pancreatitis not due to gallstones	
At admission	**During the initial 48 h**
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm^3	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO_2 <60 mmHg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L
Criteria for acute gallstone pancreatitis	
At admission	**During the initial 48 h**
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm^3	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid

Modified Glasgow-Imrie Score	
	Criteria
Within 48 hours of onset of symptoms	1. Arterial Oxygen Partial Pressure < 60 mmHg (8.0 kPa) 2. Age > 55 years 3. White Blood Cell Count >15 x10^3/mm3 4. Serum Calcium <8.0 mg/dl (2.0 mmol/L) 5. Blood Urea Nitrogen 44 mg/dl (16 mmol/L) 6. Blood Glucose >180 mg/dl (10 mmol/L) 7. Serum Albumin <3.2 g/dl (32 g/L) 8. Lactate Dehydrogenase > (600 IU/L)

Interpretation:

- If ≥ 3 criteria are present then severe pancreatitis is likely.

The Glasgow Score for acute pancreatitis was initially devised by the surgeon Dr. Clem William Imrie at the Royal Glasgow Infirmary in 1978, and later modified to better account for gallstone related causes in 1981.

Imrie, C. W., et al. "A single-centre double-blind trial of Trasylol therapy in primary acute pancreatitis." *British journal of surgery* 65.5 (1978): 337-341.

Osborne DH, Imrie CW, Carter DC. Biliary surgery in the same admission for gallstone associated acute pancreatitis BrJ Surg 1981; 68: 758-61

MANAGEMENT

·NPO (nil per os) = fasting >> no stimulation of pancreas by CCK.

·NGT if vomiting present (symptomatic relief)

·Aggressive IV fluid resuscitation and Foley catheter.

Due to NPO, and since the pancreas is retroperitoneal there might be third-space loss (fluid goes to the interstitium).

·Daily fluid needs calculated as following: Deficit fluid = (0.5 for female, 0.6 for male) * cofactor * weight * 10 | this is replaced by Ringer lactate (50% on 8-hrs. and 50% on 16hrs.) ,The Cofactor for (mild =5, moderate =10, severe =15). ·

*Maintenance fluid replaced by glucose saline.

·Pain control (no morphine because it causes spasm of sphincter of Oddi or the pancreatic duct)

·ICU monitoring for severe cases

·In case of biliary pancreatitis, laparoscopic cholecystectomy should be performed prior to discharge because likelihood of recurrence is high, unless pancreatitis is severe in which case delayed cholecystectomy (after 6 weeks) is recommended.

IV FLUIDS

Ringer lactate

Glucose Saline

TREATMENT

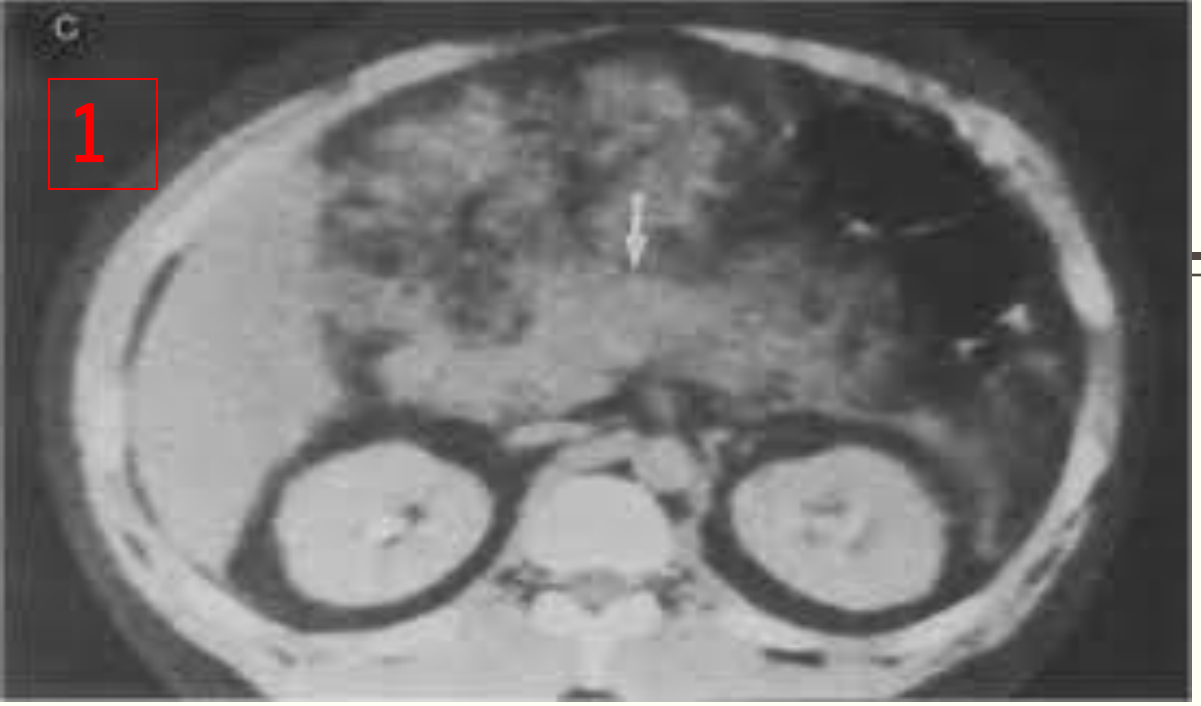
ANTIBIOTICS ??

Antibiotic in pancreatitis only if there is infected pancreatic necrosis or other documented infection (UTI , chest infection , cholangitis , acute cholecystitis ...etc

COMPLICATION

I. Local
   A. Pancreatic phlegmon
   B. Pancreatic abscess
   C. Pancreatic pseudocyst
   D. Pancreatic ascites
   E. Involvement of adjacent organs, with hemorrhage, thrombosis, bowel infarction, obstructive jaundice, fistula formation, or mechanical obstruction

II. Systemic
   A. Pulmonary
      1. Pneumonia, atelectasis
      2. Acute respiratory distress syndrome
      3. Pleural effusion
   B. Cardiovascular
      1. Hypotension
      2. Hypovolemia
      3. Sudden death
      4. Nonspecific ST-T wave changes
      5. Pericardial effusion
   C. Hematologic
      1. Hemoconcentration
      2. Disseminated intravascular coagulopathy
   D. GI hemorrhage
      1. Peptic ulcer
      2. Erosive gastritis
      3. Portal vein or splenic vein thrombosis with varices
   E. Renal
      1. Oliguria
      2. Azotemia
      3. Renal artery/vein thrombosis
   F. Metabolic
      1. Hyperglycemia
      2. Hypocalcemia
      3. Hypertriglyceridemia
      4. Encephalopathy
      5. Sudden blindness (Purtscher's retinopathy)
   G. Central nervous system
      1. Psychosis
      2. Fat emboli
      3. Alcohol withdrawal syndrome
   H. Fat necrosis
      1. Intra-abdominal saponification
      2. Subcutaneous tissue necrosis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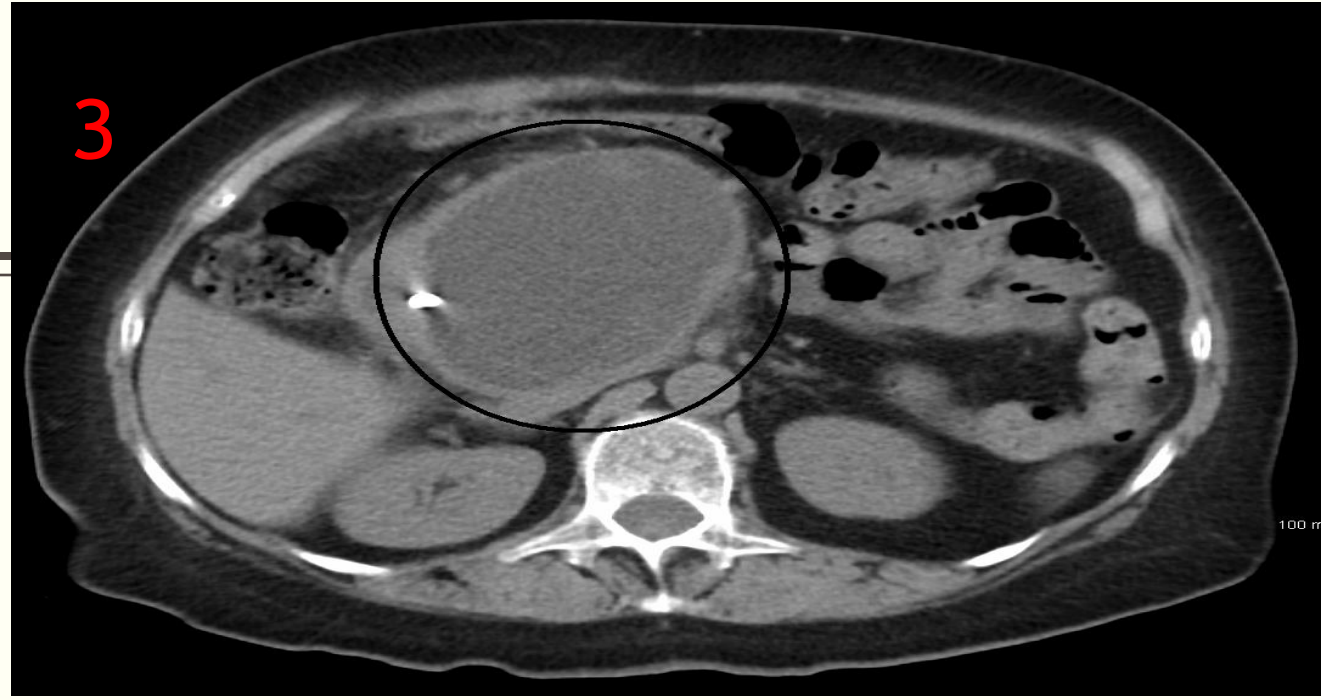
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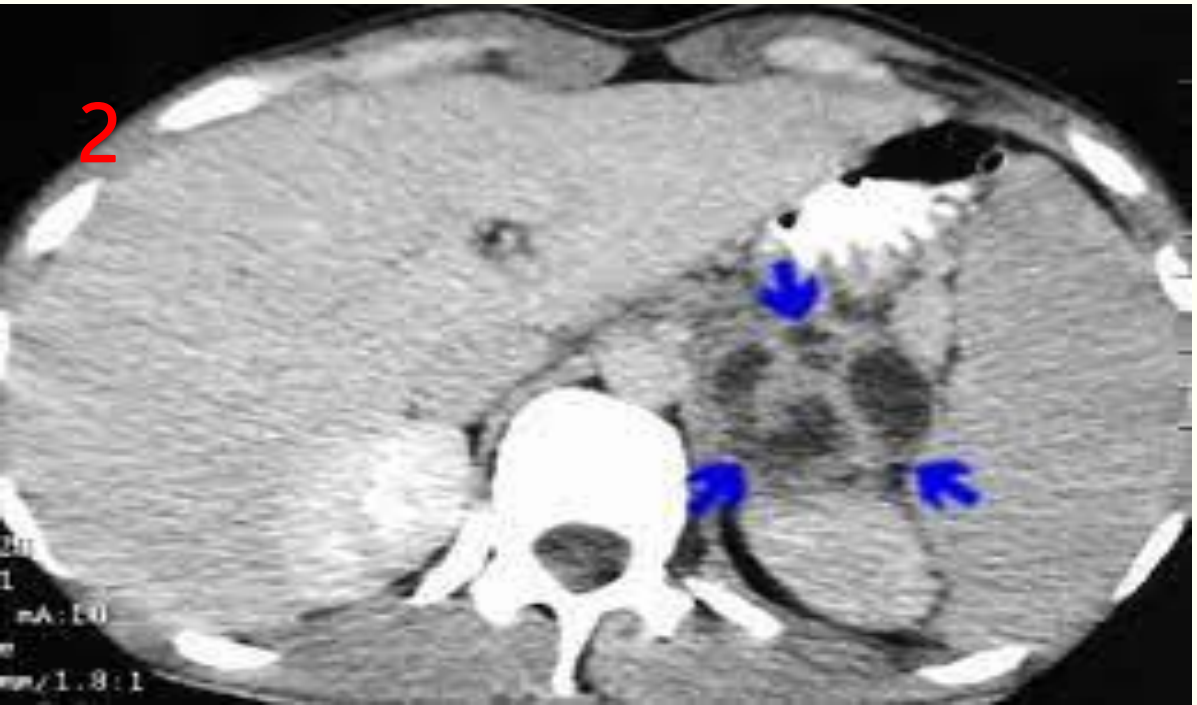
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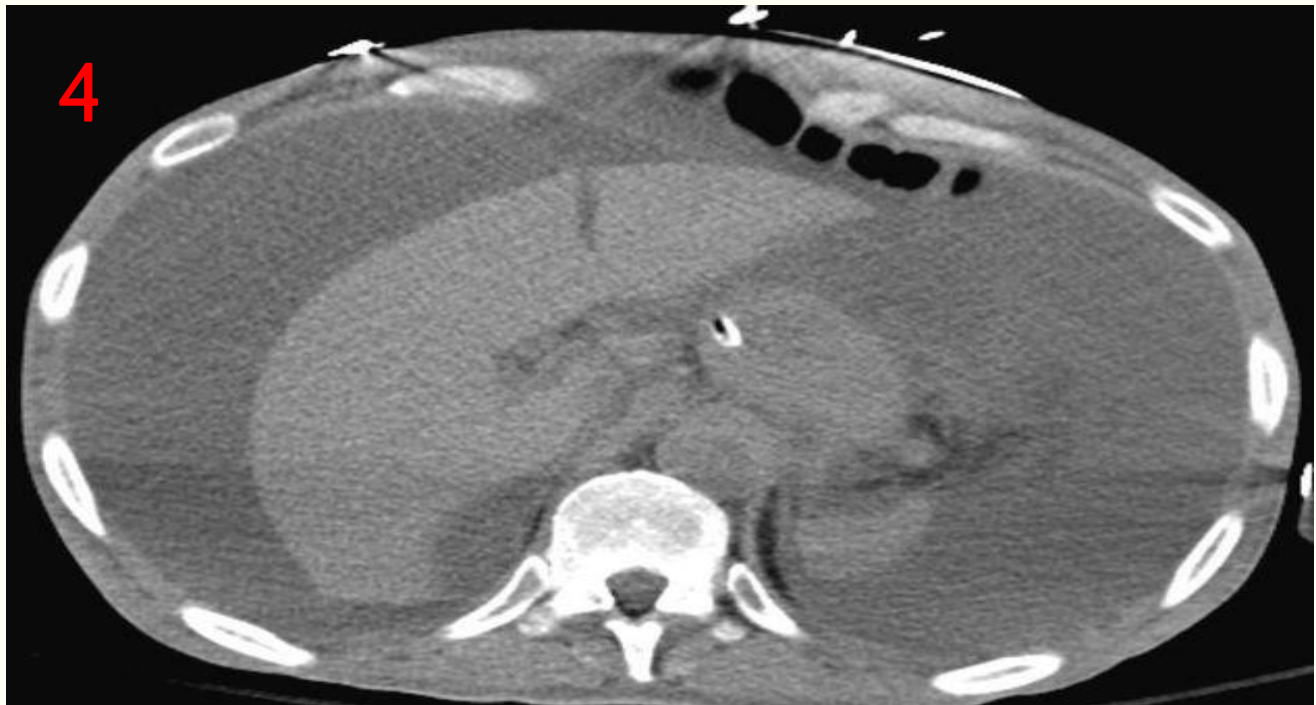
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