ACUTE PANCREATITIS

Supervised by : DR.MOHAMMAD ASSEM

DONE BY : NESSREEN KHALDOUN

NEVEEN SALEM

SHAHD AYOUB

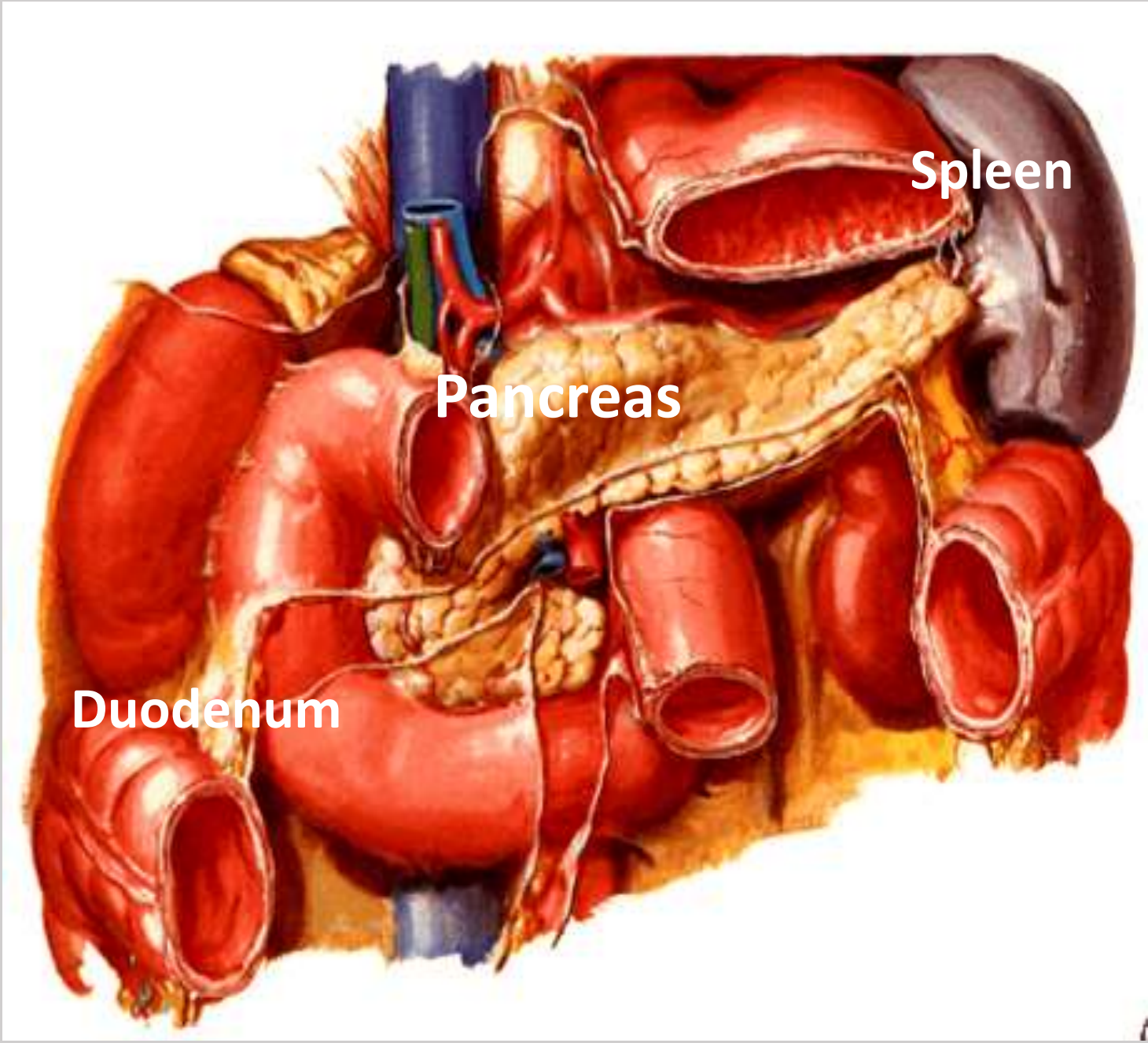
** Pancreas

- is a combined exocrine and endocrine gland

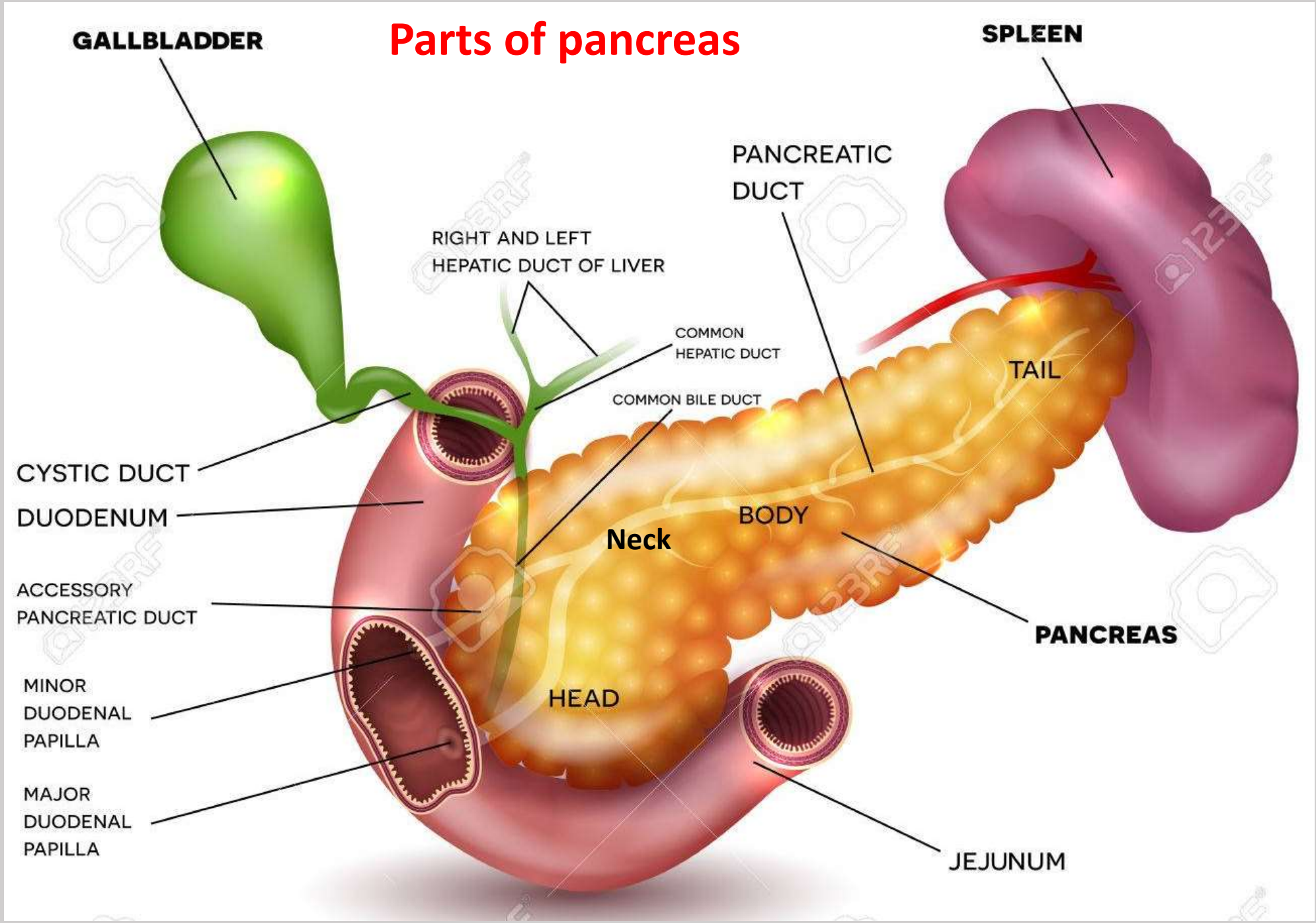
-The pancreas is a retroperitoneal organ that lies in an oblique position, sloping upward from the C-loop of the duodenum to the splenic hilum.

- Tail is intraperitoneal.

**weighs 75 to 100 g and is about 15 to 20 cm long



Parts of pancreas



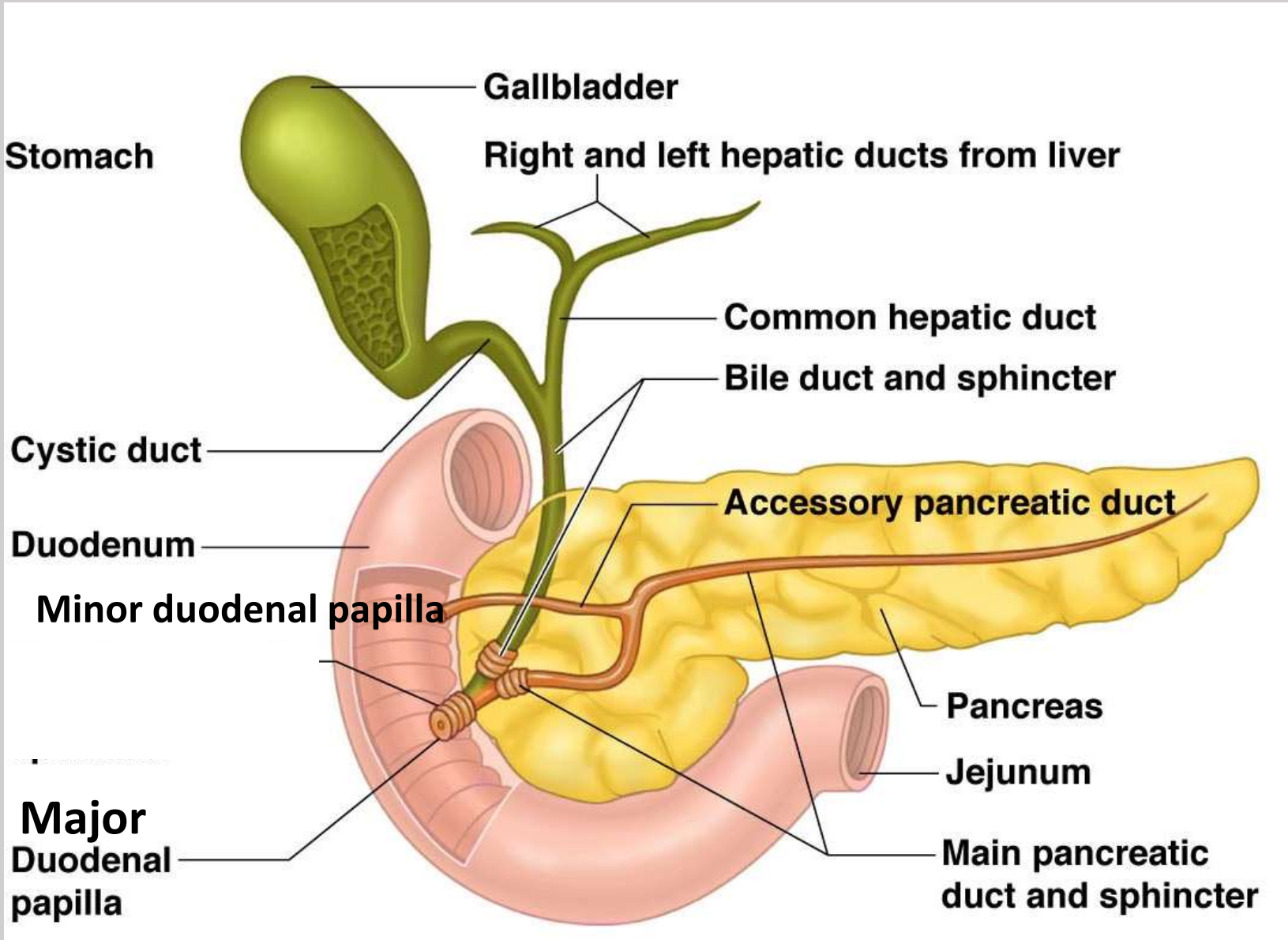
Pancreatic ducts

1- Main pancreatic duct (duct of Wirsung); runs along the long axis of the pancreas from the tail to the head.

- It fuses with **CBD** forming **hepato-pancreatic duct** that opens into the **major duodenal papilla** (ampulla of Vater) in the 2nd part of the duodenum.

2- Accessory pancreatic duct, begins from the lower part of the head and uncinate process.

- It opens into the 2nd part of the duodenum on the **minor duodenal papilla,** one inch above the major duodenal papilla.



- **Blood supply:**
- **Arteries** : splenic superior and inferior pancreaticoduodenal arteries .

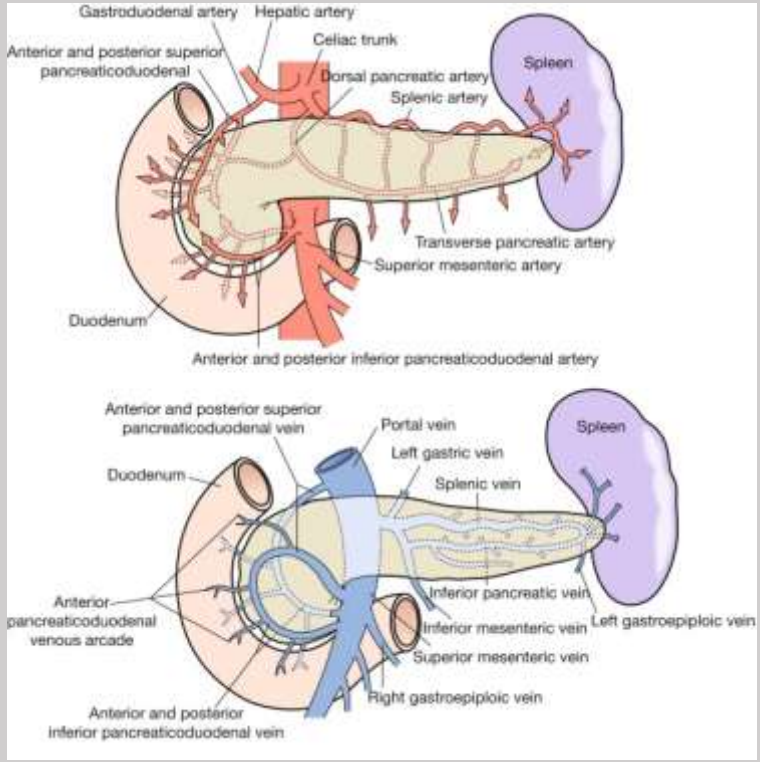
- **Veins** :corresponding veins into portal system.

- **Lymph drainage**:

  celiac and superior mesenteric lymph nodes.

- **Nerve supply:**

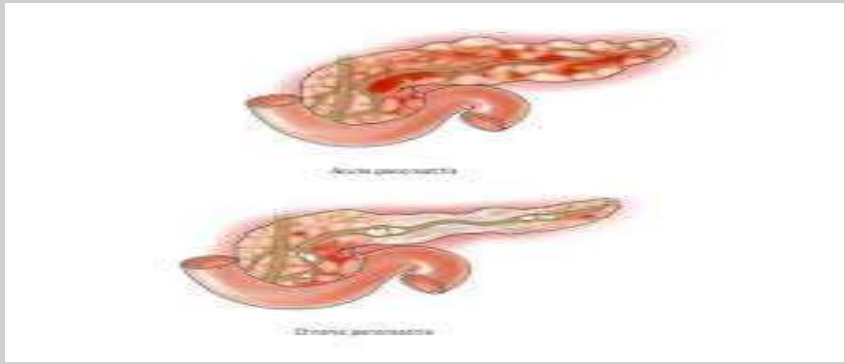
  sympathetic and parasympathetic (vagal) nerve.



Acute pancreatitis

- is an inflammatory disorder of the pancreas that is characterized by edema and, with severe, necrosis, presenting with abdominal pain and is usually associated with raised pancreatic enzyme levels in the blood or urine.

- underlying mechanism of injury in pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of auto-digestion.



Acute pancreatitis may be categorized as mild or severe:

- **Mild acute pancreatitis** is characterized by interstitial edema of the gland and minimal organ dysfunction. 80% of patients will have a mild attack of pancreatitis, the mortality from which is around1%.

- **Severe acute pancreatitis** is characterized by pancreatic necrosis, a severe systemic inflammatory response and often multi-organ failure. In those who have a severe attack of pancreatitis, the mortality varies from 20 to 50%.
- About one-third of deaths occur in the early phase of the attack, from multiple organ failure, while deaths occurring after the first week of onset are due to septic complications.

Incidence

- Worldwide annual incidence may range from 5 to 80 per 100 000. The disease may occur at any age, with a peak in young men and older women.

- The incidence of acute pancreatitis also shows significant variation related to the prevalence of etiological factors and ethnicity and

Smoking is an independent risk factor for acute pancreatitis.

Etiology

- The most common causes are gallstones and alcohol ,accounting for up to 80% of cases.

- The median age at index presentation of acute pancreatitis varies with etiology: with alcohol- and drug-induced pancreatitis presenting in the third or fourth decade compared with gallstone and trauma in the sixth decade.

- The gender difference is probably more related to etiology: in males alcohol is more often the cause while in females it is gallstones.

Table 33-4

Etiologies of acute pancreatitis

Alcohol
Biliary tract disease
Hyperlipidemia
Hereditary
Hypercalcemia
Trauma
External
Surgical
Endoscopic retrograde cholangiopancreatography
Ischemia
Hypoperfusion
Atheroembolic
Vasculitis
Pancreatic duct obstruction
Neoplasms
Pancreas divisum
Ampullary and duodenal lesions
Infections
Venom
Drugs
Idiopathic

Reproduced with permission from Townsend CM, Sabiston DC: Sabiston's Textbook of Surgery: *the biological basis of modern surgical practice*, 16th ed. Philadelphia, PA: Saunders/Elsevier; 2000.

Etiology: (GET SMASHED)

G: Gallstone

E: Ethanol

T: Trauma

S: Steroid

M: Mump

A :Autoimmune

S: Scorpion bits

H: Hyperlipidemia

E: ERCP

D: Drugs

+ Cancer

بشوفهم أكثر شي بالجراحة

1. Gallstones:

- It was proposed that the **“common channel”** hypothesis allowed bile to reflux into the pancreatic duct.

-Another proposal was **that transient incompetence** caused by the passage of a **stone through the sphincter** might allow duodenal fluid and bile to reflux into the pancreatic duct

-A third possibility is that acute pancreatitis is due to the gallstone obstructing the **pancreatic duct** and leading to ductal hypertension. (the most likely one)

2. Alcohol:

There are several mechanisms by which ethanol causes acute pancreatitis by acting on the acinar and stellate cells , The secretory burst coupled with ethanol induced spasm of the sphincter of Oddi probably cause acute pancreatitis

3. Iatrogenic:

The most common iatrogenic cause is ERCP in which acute pancreatitis occurs after about 5% to 10% of procedures , The risk of post-ERCP acute pancreatitis is increased if the contrast agent is infused repeatedly under high pressure by the endoscopist and in patients with sphincter of Oddi dysfunction.

4. Hereditary Pancreatitis:

an autosomal dominant disorder usually related to mutations of the cationic trypsinogen gene (PRSS1). Mutations in this gene cause premature activation of trypsinogen to trypsin and causes abnormalities of ductal secretion, both of which promote acute pancreatitis.

5. Hyperlipidemia:

Patients with types I and V hyperlipoproteinemia can experience episodes of abdominal pain, Lipase is thought to liberate toxic fatty acids into the pancreatic microcirculation, leading to microcirculatory impairment and ischemia.

6. Drugs :

Isolated cases of acute pancreatitis have been associated with exposure to certain drugs, such as **thiazide diuretics, furosemide**, **estrogen replacement therapy**, and **steroid therapy in children**.

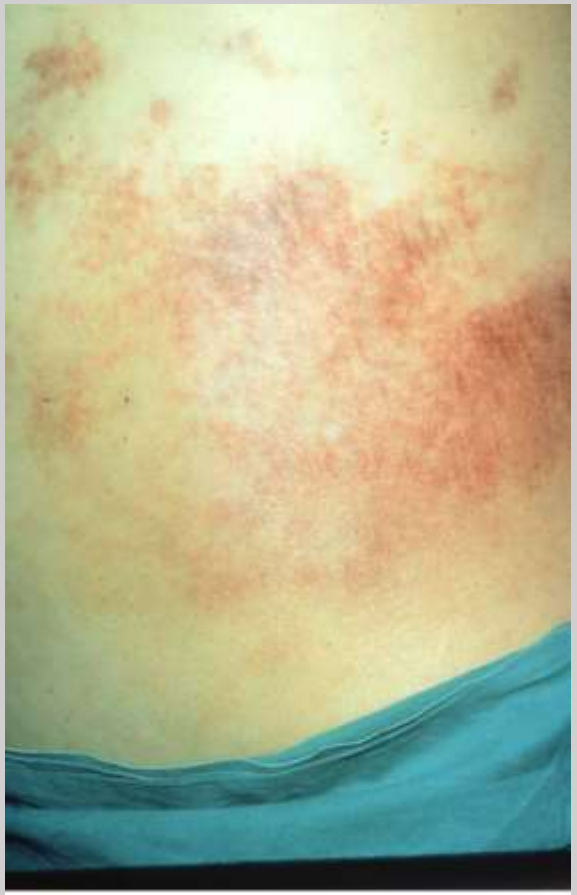
7. tumors:

A pancreatic or periampullary tumor should be considered in patients with idiopathic acute pancreatitis, especially in those over 50 years old. Approximately 1% to 2% of patients with acute pancreatitis have a pancreatic tumor.

Clinical presentation

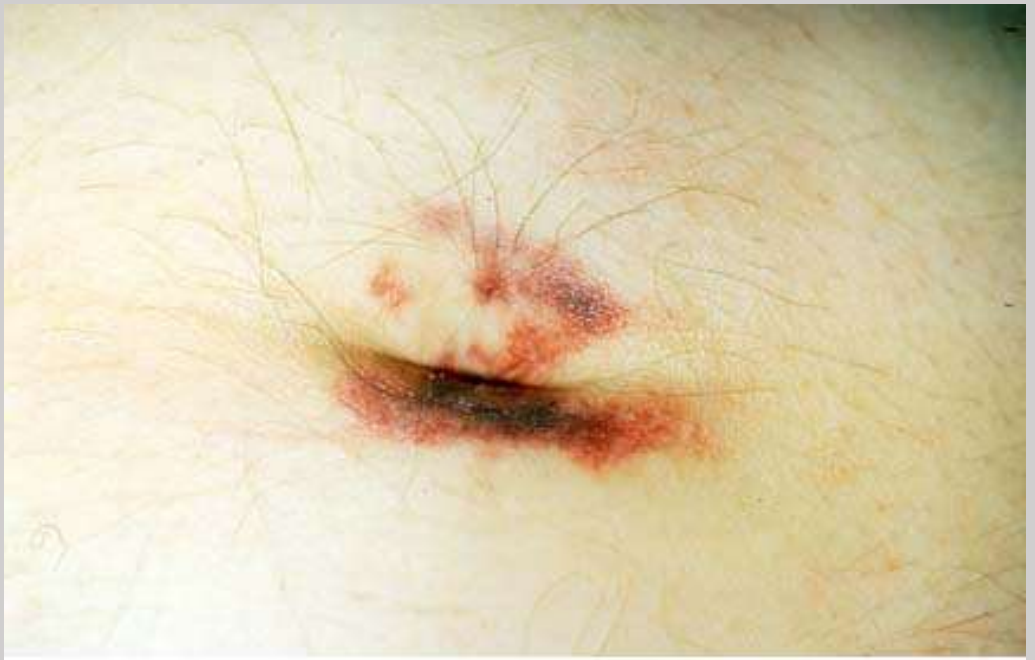
- Epigastric pain that radiates to the back and relived by leaning forward.
- abdominal tenderness, often with signs of peritonitis in the upper abdomen.
- Rarely, pancreatic fluid and bleeding from the pancreas into the retroperitoneum may result in a bruise-like discoloration around the umbilicus (Cullen’s sign) or in the flanks (Grey Turner’s sign).
- Another rare sign is tetany as a result of hypocalcaemia.
- In addition to hemoconcentration, patients with acute pancreatitis often have azotemia with elevated blood urea nitrogen and creatinine levels, hyperglycemia, and hypoalbuminemia.

Grey Turner sign



Source: Lichtman MA, Shafer MS, Felgar RE, Wang N:
Lichtman's Atlas of Hematology: http://www.accessmedicine.com

Cullen's sign



Source: Lichtman MA, Shafer MS, Felgar RE, Wang N:
Lichtman's Atlas of Hematology: http://www.accessmedicine.com

Diagnosis

The diagnosis of acute pancreatitis requires the patient to present with 2 of the 3:

1-abdominal pain consistent with acute pancreatitis (acute onset of a severe constant epigastric pain that often radiate through to the mid back) and

2-the elevation of serum amylase or lipase (>3 times upper limit of normal).

↳Be more specific than lipase when it is at least five time greater than the normal value

3-Imaging (usually by contrast-enhanced CT scanning).

Also, CT is only required for the diagnosis

of acute pancreatitis when these diagnostic criteria are not met.

contrast-enhanced CT scan

It may be necessary to perform to diagnose acute pancreatitis in patients who are **severely ill** or in those presenting with **undifferentiated** abdominal pain.

- the diagnosis of **local complications**; in particular, the development and extent of pancreatic necrosis and the different Collections

NOTES

there is no advantage in using CT scanning to predict the severity of acute pancreatitis

There is no correlation between the extent of serum amylase elevation and severity of pancreatitis

normal serum amylase doesn’t exclude acute pancreatitis particularly if the patient has presented a few days later

Don’t forget that hyperamylasemia can also occur in association with other diseases such as duodenal ulcer, small bowel obstruction.

Predicting Severity

pancreatitis severity is important in making triage decisions about whether a patient should be transferred to a tertiary hospital or an intensive care unit and in making decisions about fluid therapy and whether an ERCP is indicated, as well as other issues.

We predict the severity by the most widely used

Ranson's criteria.

Ranson's prognostic signs of pancreatitis

Criteria for acute pancreatitis not due to gallstones

At admission :

-Age >55 y

-WBC >16,000/mm3

-Blood glucose >200 mg/dL

-Serum LDH >350 IU/L

-Serum AST >250 U/dL

During the initial 48 h :

-Hematocrit fall >10 points

-BUN elevation >5 mg/dL

-Serum calcium <8 mg/dL

-Arterial PO2 <60 mmHg

-Base deficit >4 mEq/L

-Estimated fluid sequestration >6L

Criteria for acute gallstone pancreatitis

At admission

Age >70 y

WBC >18,000/mm3

Blood glucose >220 mg/dL

Serum LDH >400 IU/L

Serum AST >250 U/dL

During the initial 48 h

Hematocrit fall >10 points

BUN elevation >2 mg/dL

Serum calcium <8 mg/dL

Base deficit >5 mEq/L

Estimated fluid sequestration >4 L

****Fewer than three positive criteria predict mild, uncomplicated disease, whereas more than six positive criteria predict severe disease with a mortality risk of 50%.**

GLASGOW-IMRIE SCORE

- USED TO ASSESS SEVERITY OF PANCREATITIS AT 48h

*each criteria take one point

* if >3 : sever pancreatitis

P $Pa\,O_2$ < 8 kPa / 60 mmHg

A AGE > 55

N NEUTROPHILS (WBC) > 15×10^9/L

C CALCIUM < 2 mmol/L / 8 mg/dL

R RENAL FUNCTION: BUN > 16 mmol/L / 45 mg/dL

E ENZYMES: LACTATE DEHYDROGENASE > 600 UNITS/L
ASPARTATE TRANSAMINASE > 2000 UNITS/L

A ALBUMIN < 32 g/L

S SUGAR: GLUCOSE > 10 mmol/L / 180 mg/dL

Classification of Severity

For Accurately classifying or staging acute pancreatitis severity

The key determinants of severity are
local complications (absent, sterile, or infected) and
Systemic complications (absent, transient organ Failure, persistent organ failure).
Two classification systems have recently
been proposed: the three grades (mild, moderately severe, and
severe) of the Revised Atlanta Criteria (RAC) and the four
categories (mild, moderate, severe, critical) of the Determinants
Based Classification (DBC)

Imaging: Therapeutic Endoscopic Retrograde Cholangiopancreatography(ERCP)

Randomized trials have demonstrated that early ERCP (within 24 or 48 hours of admission) reduce complications, but not mortality, in patients with predicted severe gallstone associated acute pancreatitis

More recent evidence has suggested that early ERCP confers no benefit in the absence of concomitant cholangitis

Investigations in acute pancreatitis should be aimed at answering three questions

Is the diagnosis of acute pancreatitis correct?

How severe is the attack?

What is the etiology?

Management of Acute Pancreatitis

The essential requirements for the management of acute pancreatitis are:

-accurate diagnosis and **Determining Etiology.**

-appropriate triage(to get the **right patient** to the **right place** at the **right time** with the **right care provider**)

-high-quality supportive care (treatment)

-monitoring for electrolytes(Ca, Na, k), ABG, RFT(urea- creatinine ratio), coagulation factors, PT, PTT INR, etc.....

1)Fluid Resuscitation

-is the **most important** intervention in the early management of acute pancreatitis.

-fluid loss is **due to loss of fluid in third space.**

- it is probably best to resuscitate with a balanced **crystalloid** and aim to restore normal blood volume, blood pressure, and urine output.

-In one study, lactated Ringer's solution was superior to normal saline in reducing the systemic inflammatory Response.

2)Nutritional Support

-HOW?

Parenteral nutrition is now known to be more expensive, riskier, and not more effective than enteral nutrition and should only be offered if the patient's calculated nutritional requirements cannot be achieved by the enteral route.

-WHEN? after48-72

Early initiation of enteral nutrition (within the first 24 hours of admission) is not superior to delaying an oral diet until 72 hours

***Delay** in enteral nutrition may lead to intestinal ileus and feeding intolerance.

***early** enteral feeding, particularly before adequate

resuscitation, may lead to nonocclusive mesenteric ischemia

- NG tube? if after 3 to 5 days there is evidence of feeding intolerance, tube feeding should be commenced.

3)Pain management

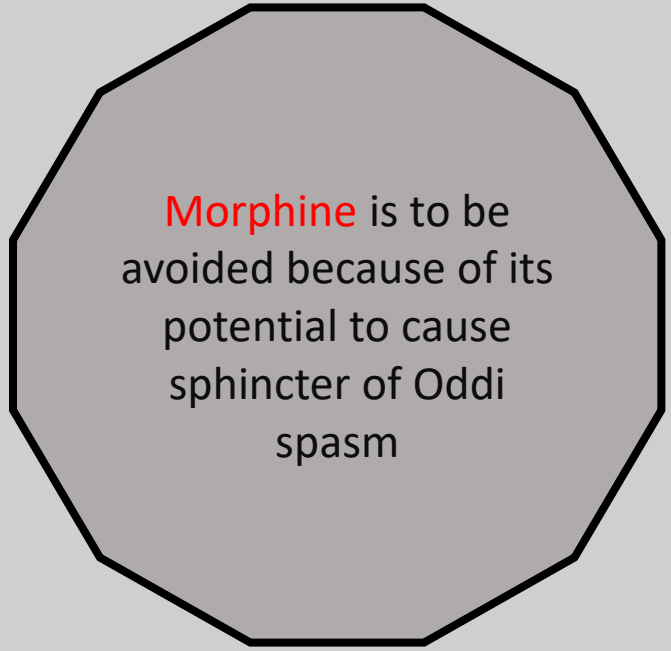
Pain is the cardinal symptom of acute pancreatitis, and its relief is

a clinical priority.

-mild pain can usually be managed with a NSADs drugs (e.g., metamizole 2 g/8 h IV),

-while those with more severe pain are best managed with opioid analgesia (e.g buprenorphine 0.3 mg/4 h IV).

Also, Administration of **pentazocine**, **procaine hydrochloride**, and **meperidine** are all of value in controlling abdominal pain.



Antibiotics

Overall, it appears that the most recent and generally better designed studies **do not support the use** of prophylactic antibiotics **to** reduce the frequency of pancreatic **infectious complications**, **surgical intervention**, and **death.**

-PPI, H2 blockers are usually not helpful, unless the patient in a coma in the ICU to prevent stress ulcer.

Complications of acute pancreatitis And their management

Table 68.5 Complications of acute pancreatitis.

Systemic	Local
(*More common in the first week*)	(*Usually develop after the first week*)
Cardiovascular	Acute fluid collection
Shock	Sterile pancreatic necrosis
Arrhythmias	Infected pancreatic necrosis
Pulmonary	Pancreatic abscess
ARDS	Pseudocyst
Renal failure	Pancreatic ascites
Haematological	Pleural effusion
DIC	Portal/splenic vein thrombosis
Metabolic	Pseudoaneurysm
Hypocalcaemia	
Hyperglycaemia	
Hyperlipidaemia	
Gastrointestinal	
Ileus	
Neurological	
Visual disturbances	
Confusion, irritability	
Encephalopathy	
Miscellaneous	
Subcutaneous fat necrosis	
Arthralgia	

ARDS, acute respiratory distress syndrome; DIC, disseminated intravascular coagulation.

A)Systemic complications

Pancreatitis may involve all organ systems and place demands on the surgeon beyond his or her skills. Patients with systemic complications should be managed by a **multidisciplinary team that includes intensive care specialists**. When there is organ failure, appropriate supportive therapies may include

1) **inotropic support** for hemodynamic instability.
2)**hemofiltration** in the event of renal failure.
3)**ventilatory support** for respiratory failure.
4)**and correction of coagulopathies** (including DIC).
5) There is no role for **surgery** during the initial period of resuscitation and stabilization; surgical intervention is contemplated only in the patient who deteriorates as a result of **local complications** following successful stabilization.

B)Local complications

- Usually by the end of the first week, major organ failure is under control, then local complications become pre-eminent in the management of these patients.
- The course of the patient should be followed carefully and, if clinical resolution does not take place or signs of sepsis develop, a **CT scan should be performed.**
- Local complications in pancreatic disease are serious and **carry a significant mortality**.
- The management approach is conservative on the whole, with ***surgery*** **restricted to situations in which conservative management has failed.**
- **So, if the patient is Unstable, infective, necrotic = needs Surgery**

1)Acute fluid collection

-this occurs **early** in the course of acute pancreatitis and is **located** in or near the pancreas. The **wall** encompassing the collection is ill defined. The **fluid** is sterile.

- most such collection resolve. No intervention is necessary unless a large collection causes symptoms or pressure effects, in which case it can be percutaneously **aspirated under ultrasound or CT** guidance. **Trans gastric drainage under EUS** guidance is another option.

-An acute fluid collection that does not resolve can evolve into a **pseudocyst** or an **abscess** if it becomes infected.

2)Sterile and infected pancreatic necrosis

-The term **'pancreatic necrosis'** refers to a diffuse or focal area of **non-viable parenchyma** that is typically associated with peripancreatic **fat necrosis**.

-Necrotic areas can be identified by an absence of contrast enhancement on **CT.**

-These are **sterile** to begin with, but can become subsequently **infected**, probably due to translocation of gut bacteria.

- Infected necrosis is associated with a **mortality rate of up to 50 %.**

What to do with necrotic tissue?

1-Sterile necrotic material **should not be drained** or interfered with.

2- if the patient shows signs of **sepsis,** CT scan should be performed, and a needle passed into the area under CT guidance. This may be done under ultrasonographic guidance as well. If the aspirate is purulent, percutaneous drainage of the infected fluid should be carried out , and appropriate antibiotic should be commenced .

3-If the sepsis worsens despite this, then a pancreatic necrosectomy should be considered.

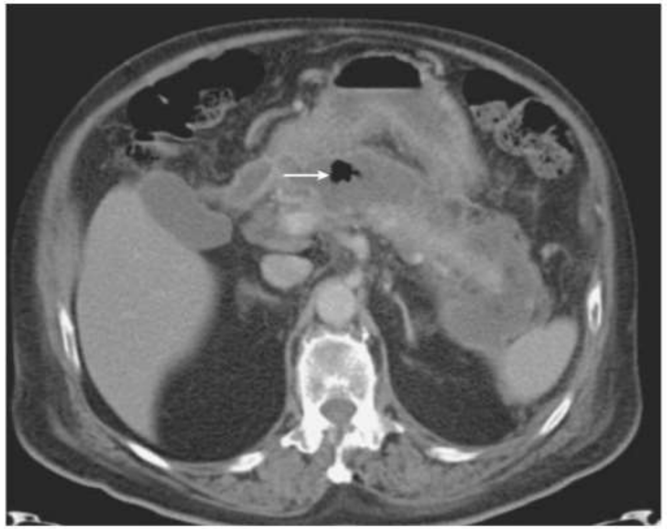


Figure 68.24 Infected pancreatic necrosis in an elderly patient. Note the areas of reduced enhancement in the pancreas and the peripancreatic fluid collection with pockets of gas within it (arrow). This resolved after percutaneous drainage and antibiotic therapy.

Figure 68.25 Necrotic body and tail of the pancreas removed as an intact specimen rather than piecemeal. The patient had suffered severe necrotising gallstone pancreatitis complicated by persistent pancreatic sepsis. Necrosectomy was carried out through a left flank retroperitoneal approach.

↳ has high mortality Rate

لأنه بنفتح ال Pancreas وبنقيم ال necrotic tissue وبنخليه فاتح لتاني يوم وبنقيم كمان ال necrotic tissue واليوم الثالث نفس الإشي علماً إنه ال Pancrease ← Retro Peritoneal organ

3)Pancreatic abscess

- This is a circumscribed intra-abdominal collection of pus, usually in proximity to the pancreas. It may be an acute fluid collection or a pseudocyst that has become infected.
- **Percutaneous drainage** with the widest possible **drains** (usually pigtail) placed under imaging guidance is the treatment, along with appropriate **antibiotics** and **supportive care**. **Repeated scans** may be required depending on the progress of the patient, and drains may need to be flushed, repositioned or reinserted. Very occasionally, open drainage of the abscess may be necessary.

4)Pancreatic ascites

- This is a chronic, generalized, peritoneal, enzyme-rich effusion usually associated with **pancreatic duct disruption**.
- Paracentesis will reveal turbid fluid with a high amylase level.
- Adequate **drainage** with wide-bore drains placed under imaging guidance is essential. Measures that can be taken to suppress pancreatic secretion include **parenteral feeding** and administration of **octreotide**. An **ERCP** may allow demonstration of the duct disruption and placement of a pancreatic stent.

5)Pancreatic effusion

- This is an encapsulated collection of fluid in the pleural cavity, arising as a consequence of acute pancreatitis.
- Concomitant pancreatic ascites may be present, or there may be a communication with an intra-abdominal collection.
- **Percutaneous drainage under imaging guidance is necessary.**

6)Hemorrhage

- Bleeding may occur into the gut, into the retroperitoneum or into the peritoneal cavity.
- Possible causes include bleeding into a pseudocyst cavity, diffuse bleeding from a large raw surface, or a pseudoaneurysm. The last is a false aneurysm of a major peri- pancreatic vessel confined as a clot by the surrounding tissues and often associated with infection.
- Recurrent bleeding is common, often culminating in fatal hemorrhage.
- CT, angiography or magnetic resonance angiography (MRA) helps to make the diagnosis**.**
- **Treatment involves embolisation or surgery**.

7)Pseudocyst (most common one)

-shape? pseudocyst is a collection of amylase-rich **fluid** enclosed in a **wall** of **fibrous or granulation tissue**.

-etiology? Pseudocysts typically arise following an attack of acute pancreatitis, but can develop in chronic pancreatitis or after pancreatic trauma.

-when? Formation of a pseudocyst **requires 4 weeks** or more from the onset of acute pancreatitis .

-number? They are often single but, occasionally, patients will develop multiple pseudocysts.

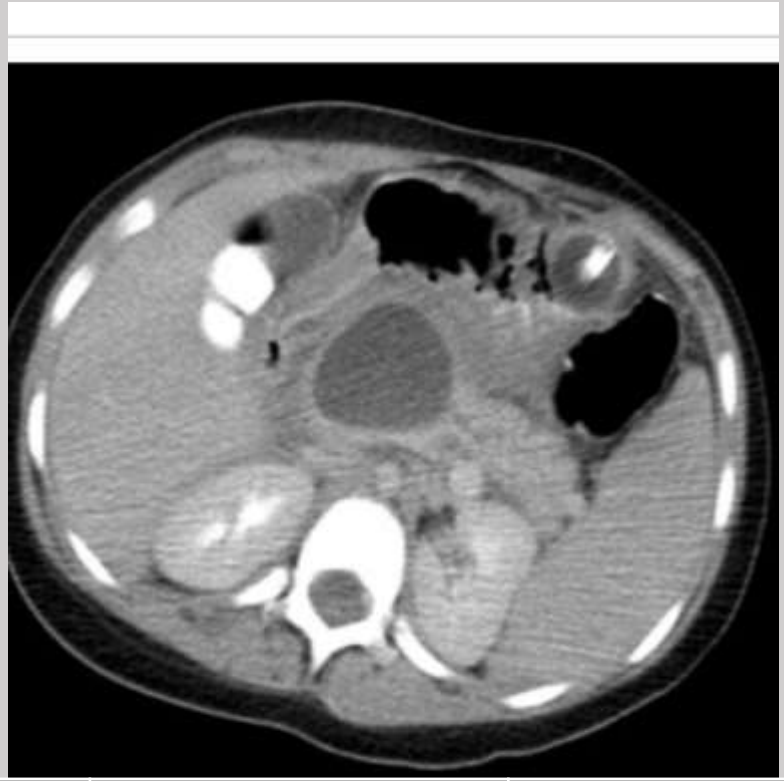
-cause? If carefully investigated, more than half will be found to have a communication with the main pancreatic duct.

-Site? of pseudocyst in the **lesser sac** between the pancreas and stomach .

-Clinical presentation?

1. A **small pseudocyst is painless** and may be discovered by follow up ultrasonography.

2. **A large cyst causes discomfort and manifests as an upper abdominal swelling** that may reach the size of a melon



	pseudocyst	Acute fluid collection
Wall?	fibrous or granulation tissue.	ill defined.
Fluid?	amylase-rich	sterile
Time?	4 w	early
Most common?	The most common complication	

- **Complication?**

infection , hemorrhage, rupture .

- **Treatment ?**

1-Most of these cysts resolve spontaneously –

clinical and ultrasound follow-up is needed –

2-A persistent pseudocyst for 6 weeks → is surgically drained. -Waiting for 6 weeks to allow the development of a strong cyst wall that can hold sutures. -The cyst is internally drained to the stomach (Cystogastrostomy) or to a jejunal loop (cystojejunostomy)

also if complicated or multiple

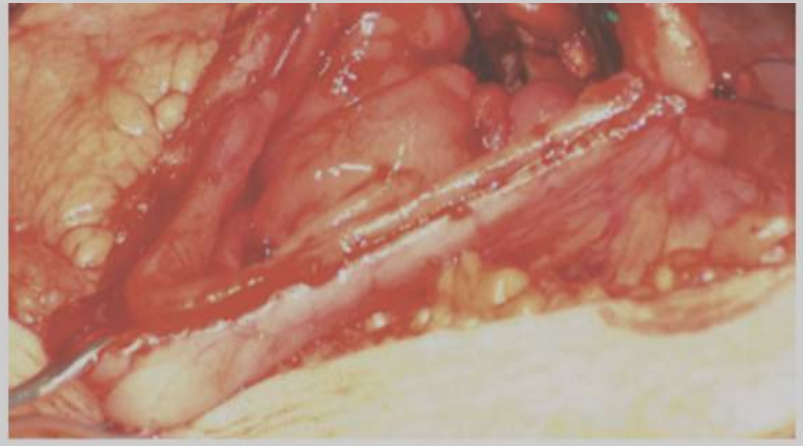
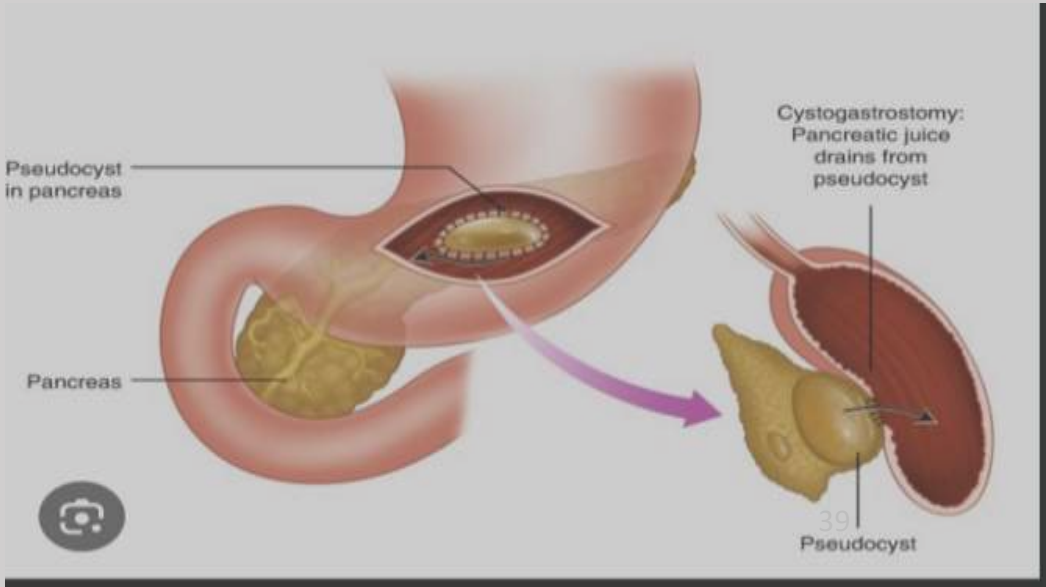
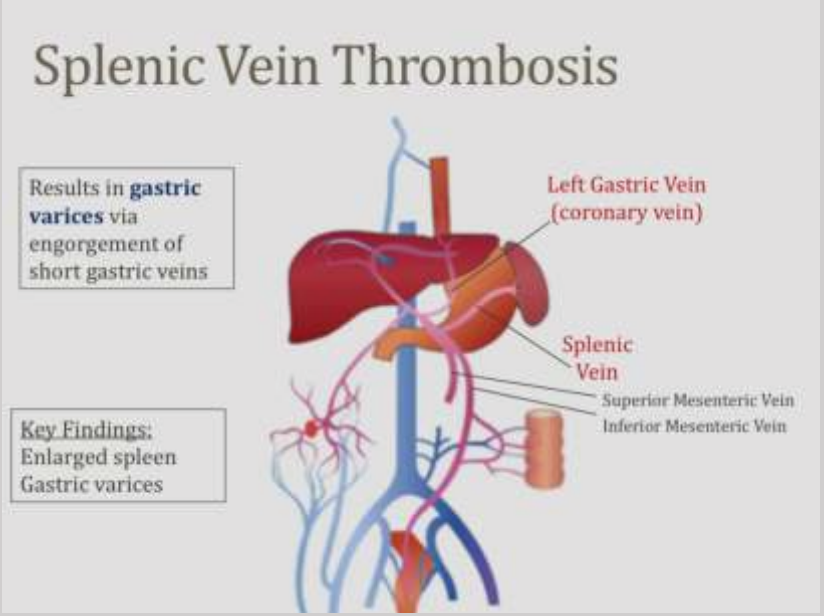


Figure 68.28 Cystgastrostomy for the pancreatic pseudocyst shown in Figure 68.10. The anterior wall of the stomach has been opened and the edges drawn back, held by Babcock's forceps. An opening has been made through the posterior wall of the stomach into the pseudocyst, and the tips of the dissecting forceps are in the cavity of the pseudocyst, which is lined by slough and granulation tissue. The tip of a nasoaastric tube is visible. A runnina stitch will next be placed alona the



8)Portal or splenic vein thrombosis

- **diagnoses?** This may often develop silently and is identified on a **CT scan**. A **marked rise in the platelet count** should raise suspicions
- **Treatment?**
- In the context of acute pancreatitis, **treatment is usually conservative.** The patient should be screened for pro-coagulant tendencies.
- **If varices or other manifestations of portal hypertension develop**, they will require treatment, such as endoscopic injection or banding, B-blockade, etc.
- **Thrombocytosis** may mandate the use of aspirin or other anti-platelet drugs for a period.
- **Systemic anticoagulation**, if instituted early in the process, may achieve recanalization of the vein, but it is not routinely used as it carries considerable risks in a patient with ongoing pancreatitis.



Thank you

- Resources :

  Schwartz’s Principles of Surge eleventh edition

  Bailey & Love's Short Practice of Surgery, 26th Edition