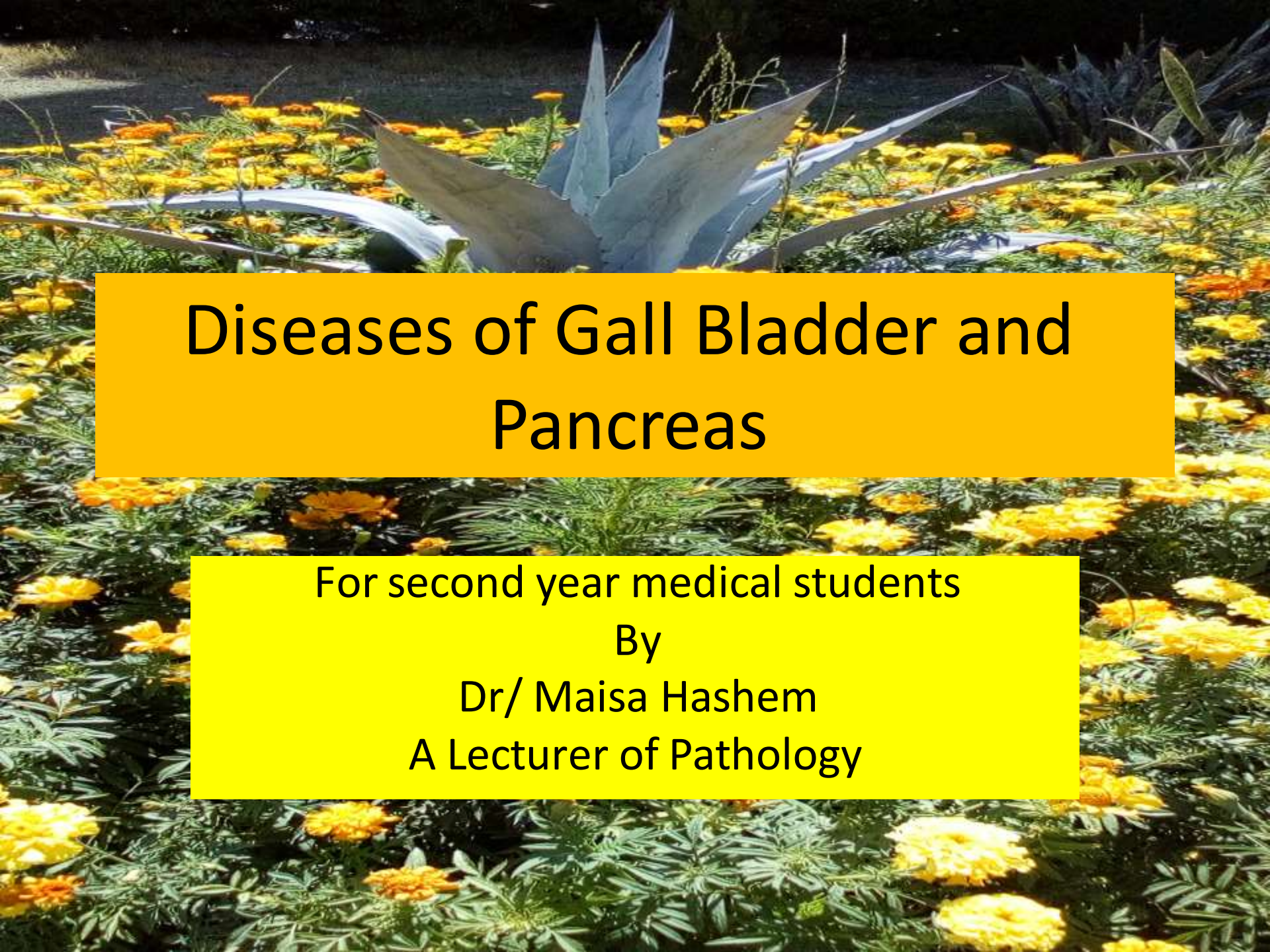

Diseases of Gall Bladder and Pancreas

For second year medical students

By
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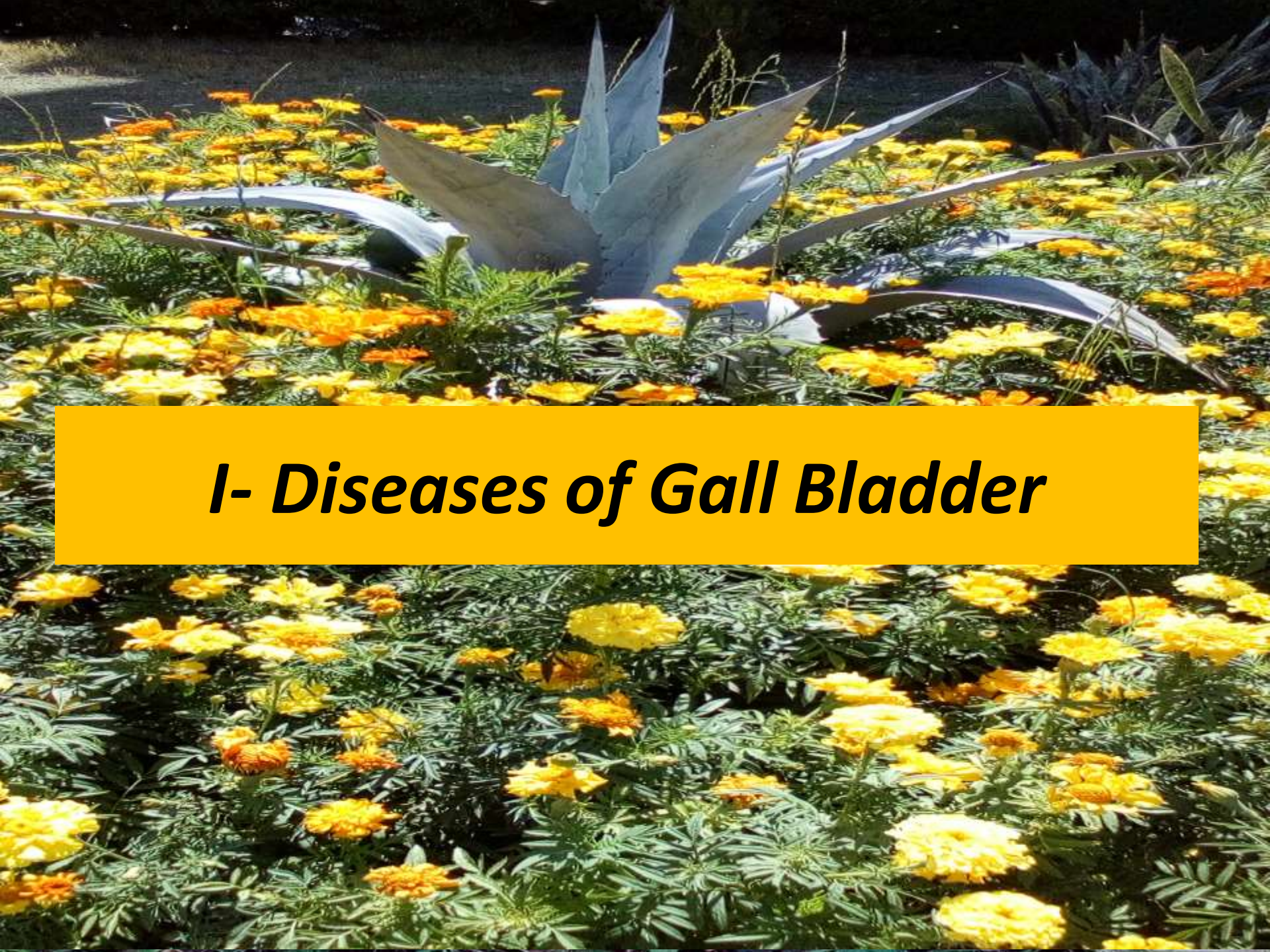
A Lecturer of Pathology

Aims of this lecture

By the end of this lecture, the student should be able to:

- Identify causes, common types of gall stones and mention their complications.
- Discussing pathology of acute and chronic cholecystitis and mention their complications.
- Determine aetiology, pathology and effects of acute pancreatitis.
- Outline the presenting features and pathology of carcinoma of exocrine pancreas.
- Define ascites and mention its causes.



I- Diseases of Gall Bladder

Gall stones

Gall stones are more common in fatty, fertile females above age of forty. They are formed commonly in the gall bladder, rarely in the intrahepatic bile ducts.

Aetiology of gall stones

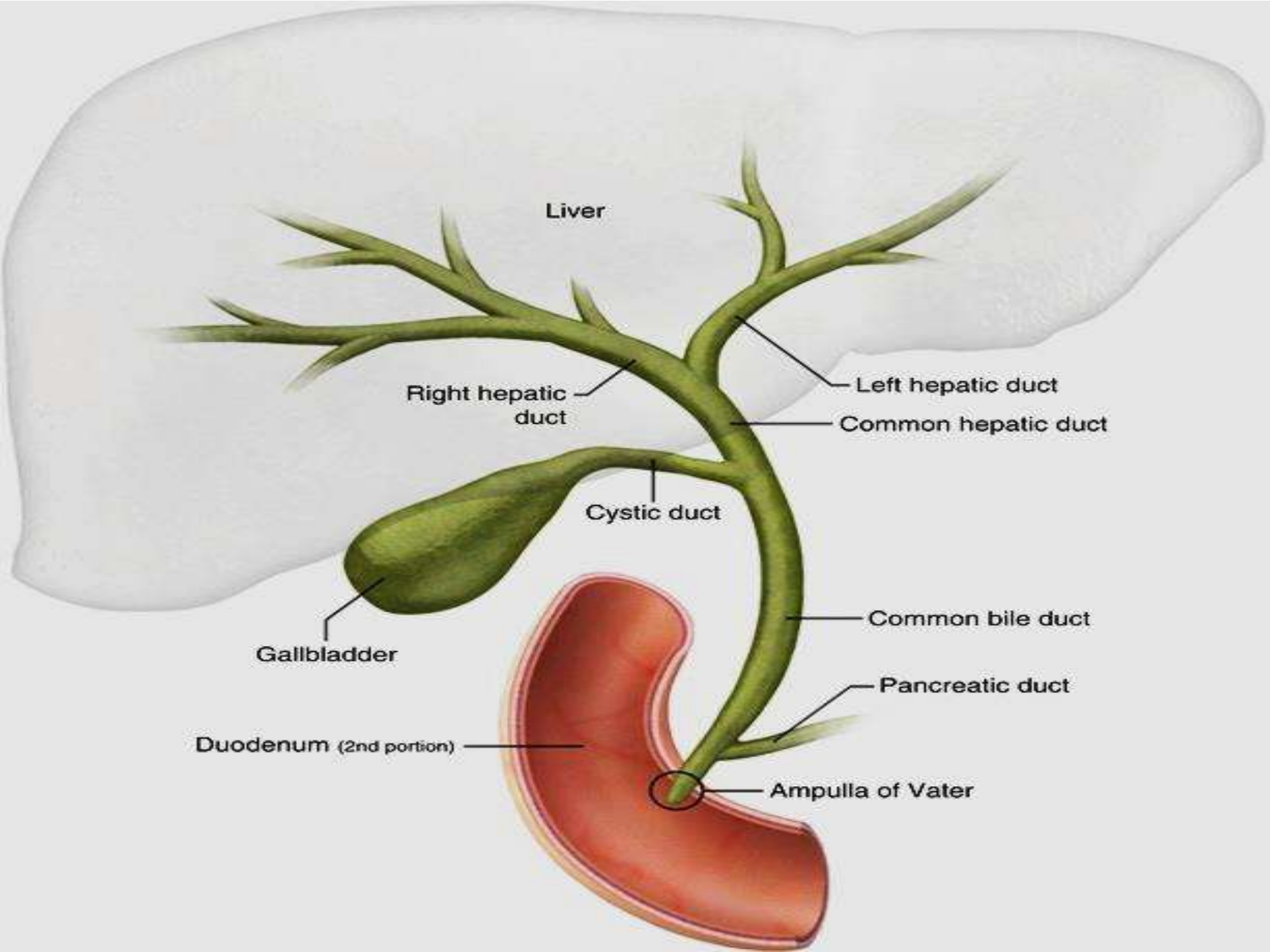
1- <u>Abnormal composition of bile:</u> bile salts act as emulsifying agent for cholesterol and bile pigments keeping them in solution. Precipitation of cholesterol or bile pigments occurs due to

- ✓ Increase cholesterol concentration in hypercholesterolaemia and bile pigments in excessive haemolysis.
- ✓ Decrease concentration of bile salts.

2- <u>Infection:</u>

- ✓ Infection provides a nucleus of bacteria, shedded epithelial cells and blood clots for stone formation.

3- <u>Stasis</u> which is caused by spasm of sphincter of Oddi or organic obstruction of the cystic duct. Stasis causes more water absorption and bile concentration, stasis also favor infection.


Liver
Right hepatic duct
Left hepatic duct
Common hepatic duct
Cystic duct
Gallbladder
Common bile duct
Pancreatic duct
Duodenum (2nd portion)
Ampulla of Vater

Types of gall stones

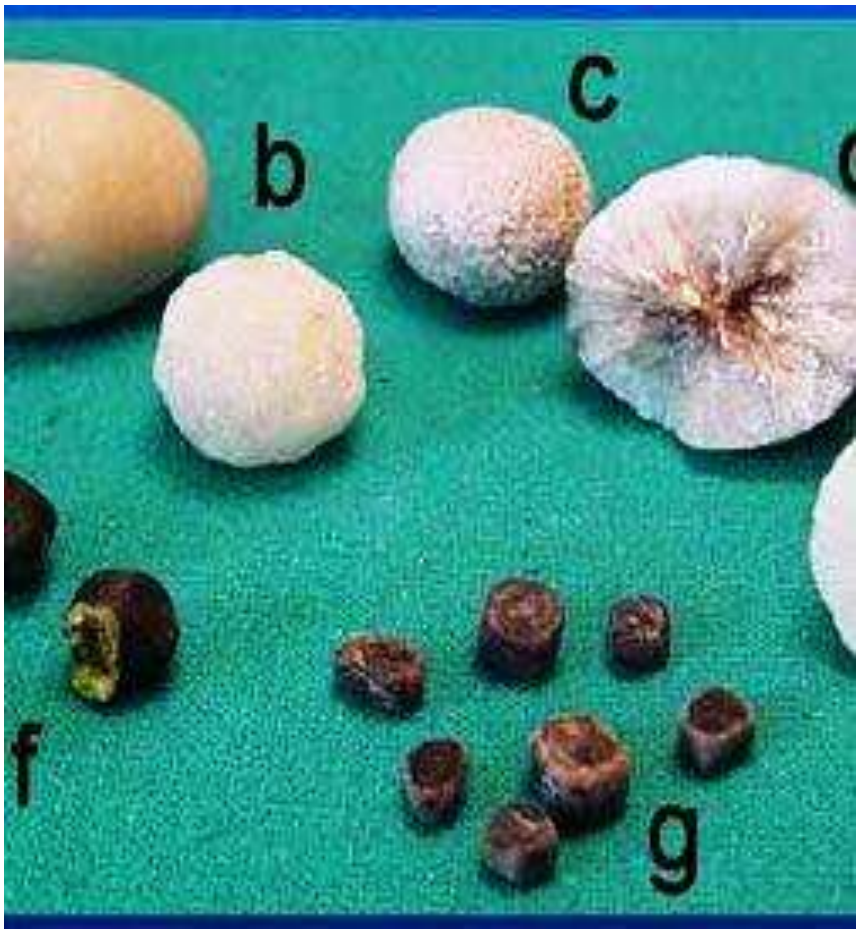
Gall stones are formed of cholesterol, calcium bilirubinate and/ or calcium carbonate.

- **Pure stones (10%):** are composed of only one of the above mentioned components; cholesterol, calcium biliribinate or calcium carbonate.
- **Mixed stones (80%):** composed of a mixture of 2 or 3 constituents.
- **Combined stones (10%): composed** of cholesterol nucleus surrounded by mixed constituents. It complicates a cholesterol stone in the presence of infection.

Examples of Gall Stones

Cholesterol stone

Yellowish- white, usually single, round to oval, has a smooth or finely granular surface. The stone float on water. Cut surface shows a radial glistening pattern.



Pigment stone

Usually multiple, small, soft, round to oval with smooth surface. Its color is dark green to black. It occurs in association with haemolytic anaemia.

Calcium carbonate stone

Rare, greyish-white in color.

Mixed (infective) stones

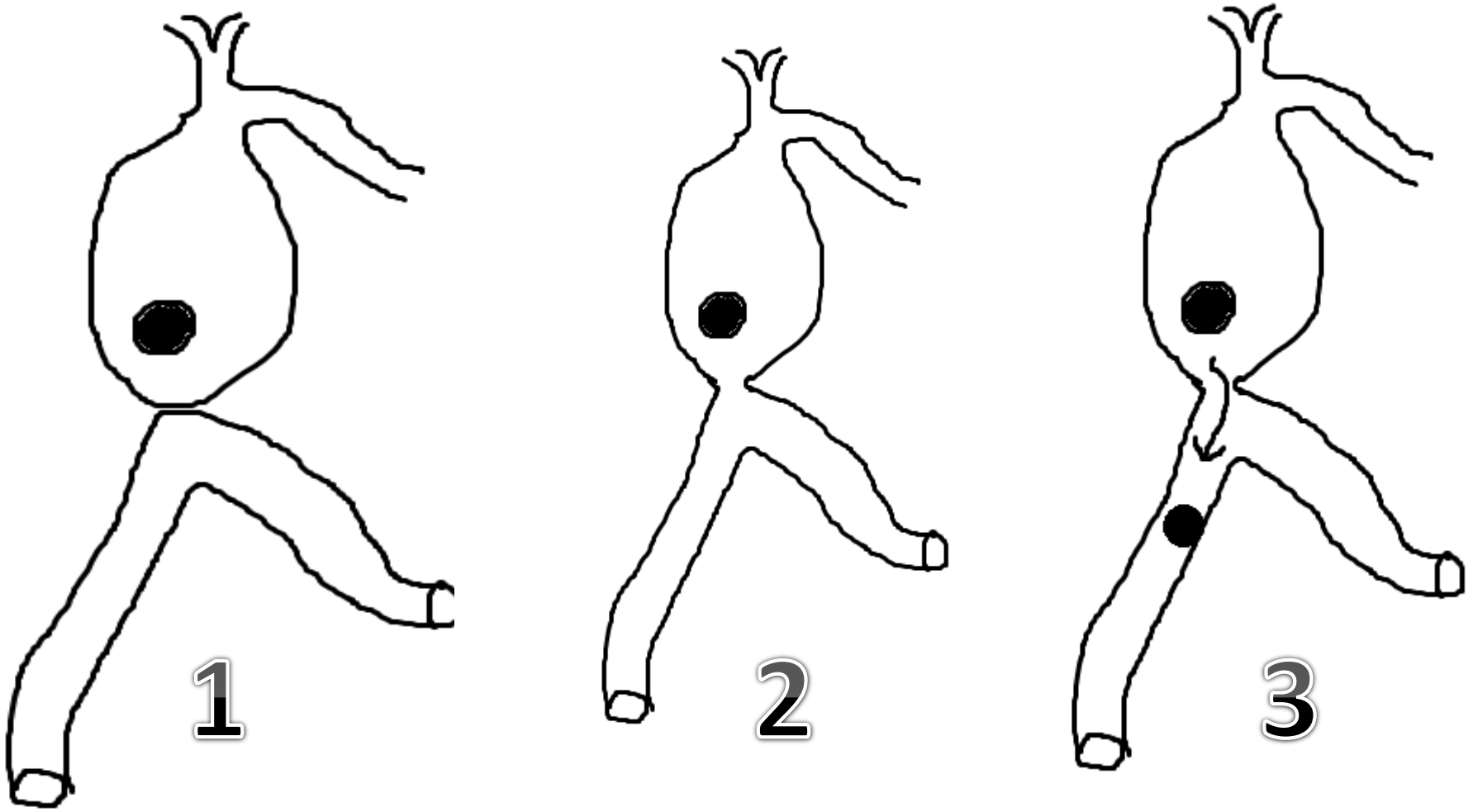
They are multiple, small, any color (whitish, greyish, yellowish, brownish or black) the stone surface is faceted due to pressure of the stones in contact with one another. Cut surface shows concentric layers of cholesterol and protein mixed with bilirubin and calcium salts. They always occur in association with cholecystitis. During an attack of cholecystitis, the gall bladder cavity is closed and contains shedded epithelium, blood clots and pus which act as nuclei. When the bile re-enter after the attack, its constituents precipitate on the performed nuclei forming a group of mixed stones. Repetition of this process results in multiple stones.

Complications of gall stones

I. Chronic cholecystitis.
II. Squamous metaplasia and squamous cell carcinoma in 3% of cases.
III. The stone may migrate causing:

1. Biliary colic.
2. Obstruction of cystic duct causing acute cholecystitis.
3. The stone may pass through a perforated gall bladder into adherent intestinal loop causing acute intestinal obstruction.
4. Obstruction of common bile duct cause obstructive jaundice.
5. Obstruction of ampulla of Vater cause acute haemorrhagic pancreatitis.

The stone may pass through a perforated gall bladder into adherent intestinal loop causing acute intestinal obstruction.



Acute Cholecystitis

The main clinical symptoms of acute Cholecystitis are pain, in the right upper abdominal quadrant, nusea, vomiting and fever.

There are three types of acute Cholecystitis; calculous, acalculous and emphysematous.

A- Acute calculous Cholecystitis

Acute calculous Cholecystitis: accounts > 90% of cases. The pathogenesis of most cases of acute calculous cholecystitis is stone impacted in cystic duct resulting in changes in concentration and composition of bile as well as obstruction and congestion in venous channels surrounding the cystic duct. The condition is usually not initiated by bacterial infection. However, secondary bacterial infection complicates about 50% of cases of acute calculous cholecystitis.

B- Acute acalculous cholecystitis: representing about 5-10% of cases of acute cholecystitis. It is often associated with serious medical or surgical illness as major cardiac surgery or end-stage renal disease.

C- Acute emphysematous (gaseous) cholecystitis: affection of gall bladder by gas-forming organisms. This condition may affect diabetic patients.

Chronic cholecystitis

Aetiology:

1. It may follow acute cholecystitis.
2. May develop as a complication of stones present in the gall bladder.

Pathological features

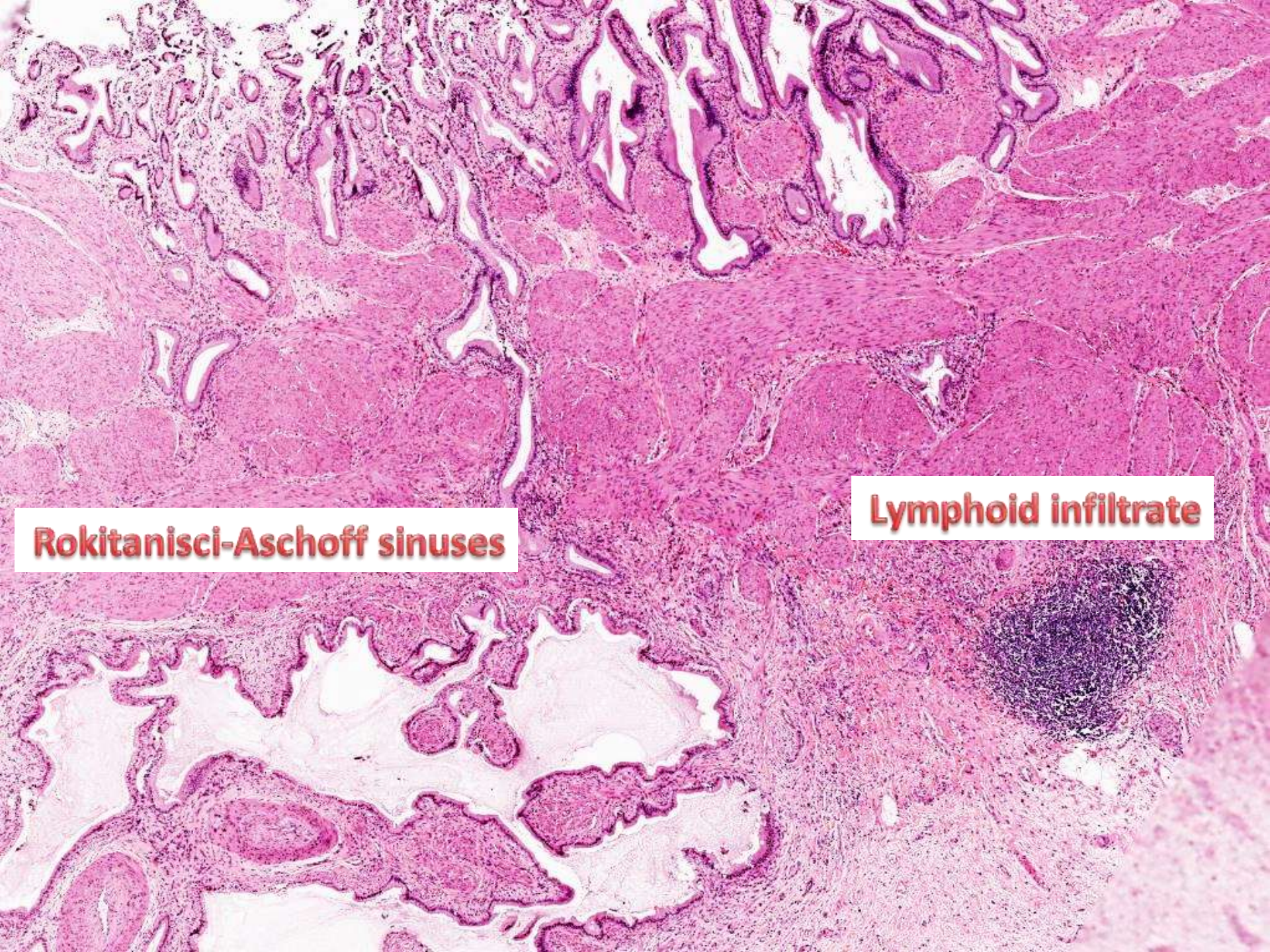
Grossly:

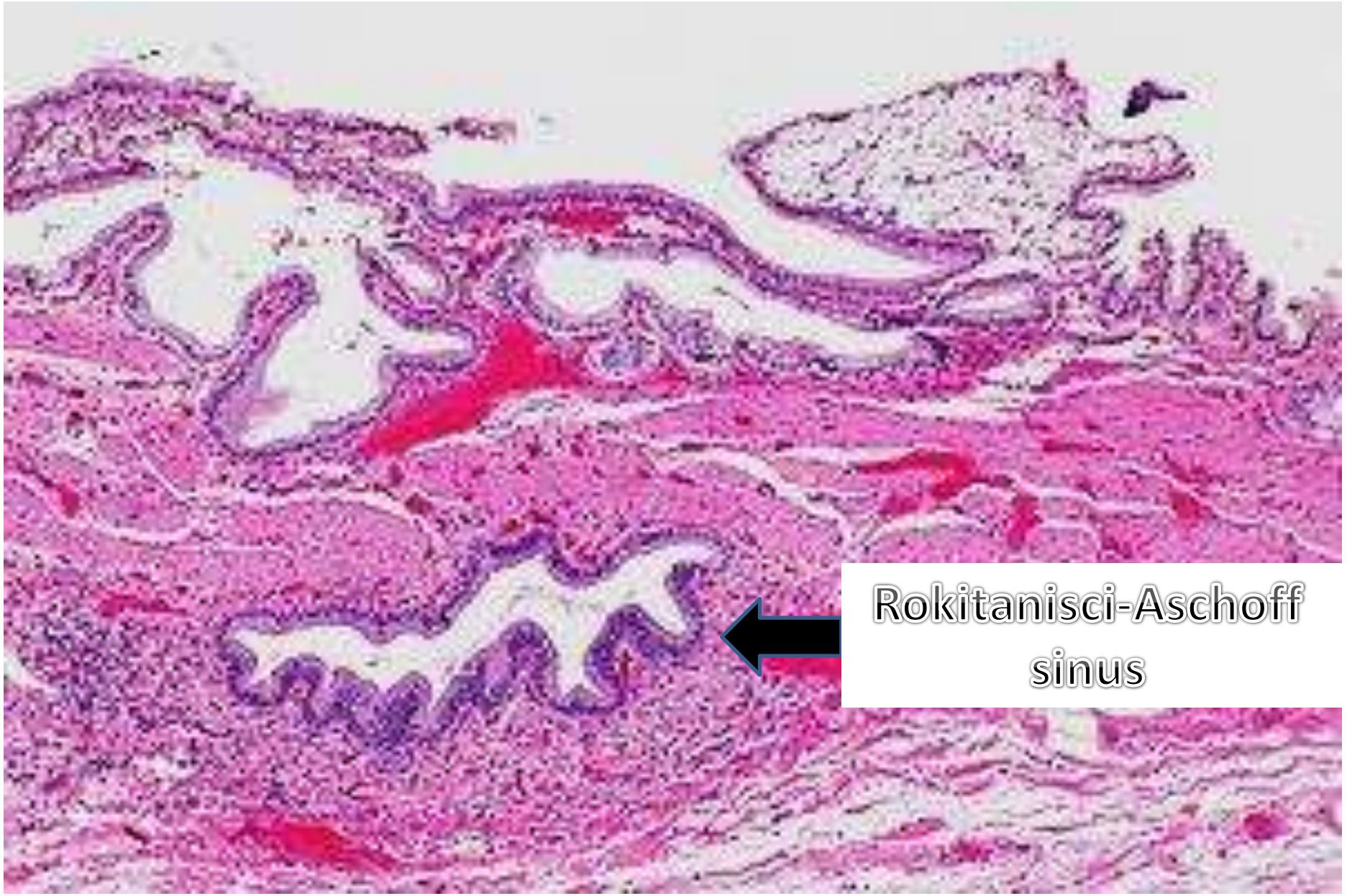
- If the cystic duct is patent; the gall bladder is small and contracted due to fibrosis of its wall. The wall is thick, narrow lumen. The mucosa is thin, atrophic or ulcerated and covered by thick biliary mud. The lumen usually contain mixed stones.
- If the cystic duct is closed by a stone; the gall bladder is distended with thin wall and dilated lumen.


Thick wall, narrow lumen

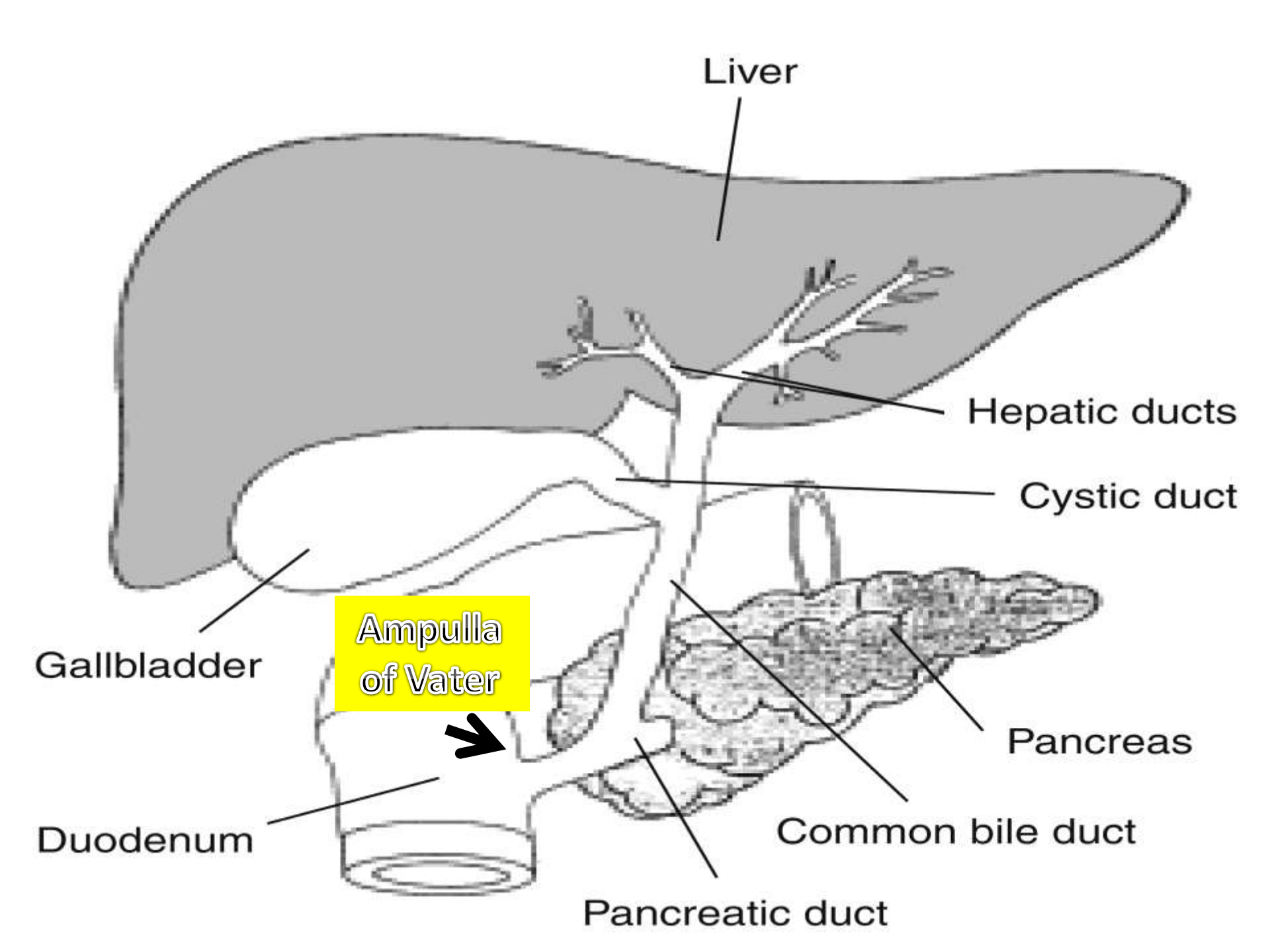
Microscopically

- The lining mucosa showing ulceration or areas of squamous metaplasia.
- The wall get infiltrated by chronic inflammatory cells and fibrosis.
- **Rokitanisky-Aschoff sinuses;** they are irregular-shaped tubular structures present deep in the wall of the gall bladder. They are lined by columnar or cuboidal epithelium and may contain bile or minute stones. These tubular structures are thought to represent herniations from the surface mucosal glands due to increased intraluminal pressure.


Rokitanisci-Aschoff sinuses
Lymphoid infiltrate


Rokitanisci-Aschoff sinus

II- Diseases of the Pancreas

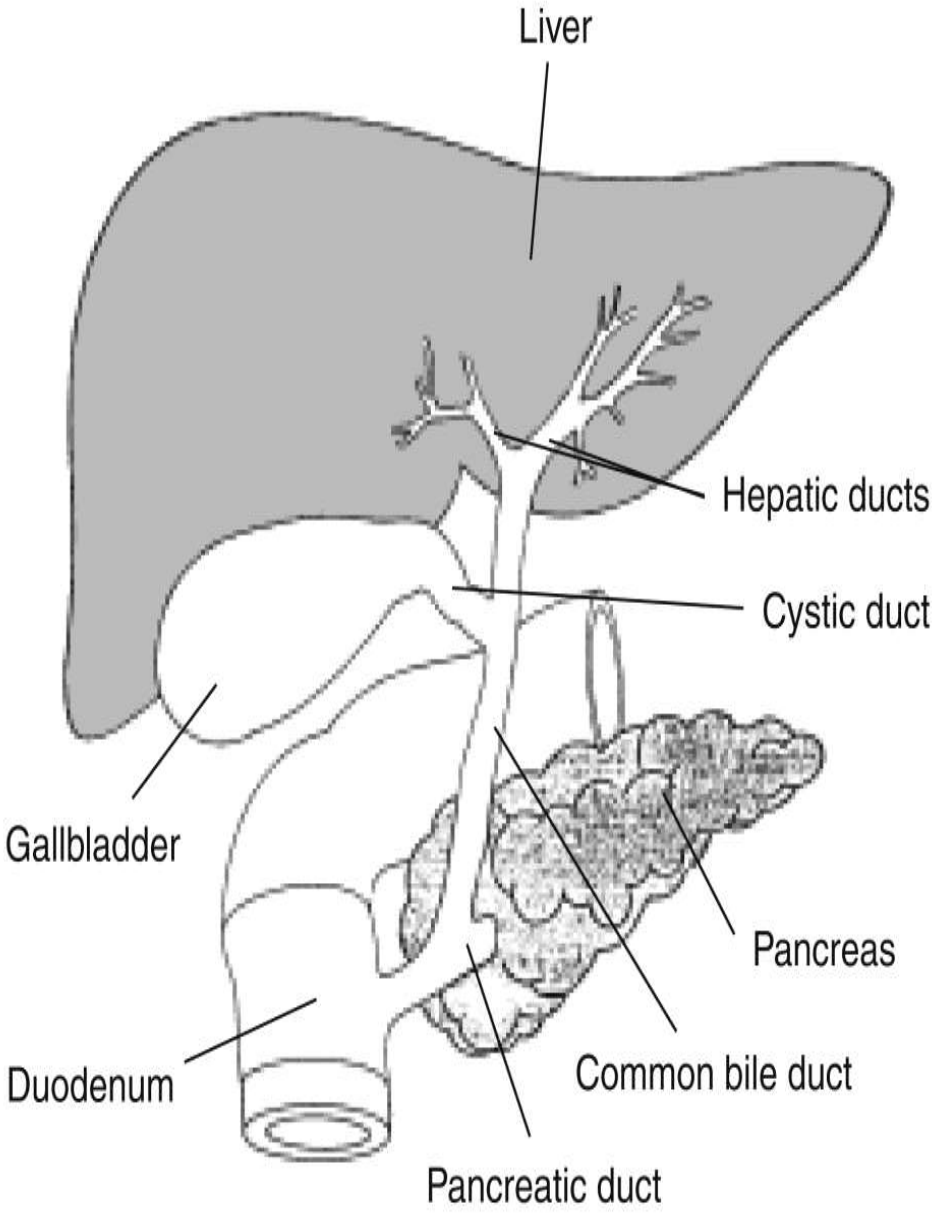

Liver
Hepatic ducts
Cystic duct
Gallbladder
Ampulla of Vater
Pancreas
Duodenum
Common bile duct
Pancreatic duct

Acute pancreatitis

Acute pancreatitis occurs when the common bile duct and pancreatic duct convert into a common channel. This occurs in the following conditions;

1. A migrating gall bladder stone or Biliary sludge impacted in the ampulla of Vater.
2. Tumor or fibrosis developed at site of ampulla of Vater.
3. Invasive pancreaticobiliary procedure.
4. Certain drugs cause spasm at region of ampulla of Vater.
5. Alcohol abuse.
6. Type II diabetes.

All these factors converting common bile duct and pancreatic duct inyo a common channel. Bile active pancreatic trypsinogen into trypsin which results in digestion of pancreatic duct wall, adjacent parenchyma and vessel walls. Activation of pancreatic lipase results in splitting of fat in omentum and mesentry into free fatty acids and glycerol. Glycerol is absorbed while fatty acids are deposited in the peritoneal cavity with calcium forming what's called calcium soaps.



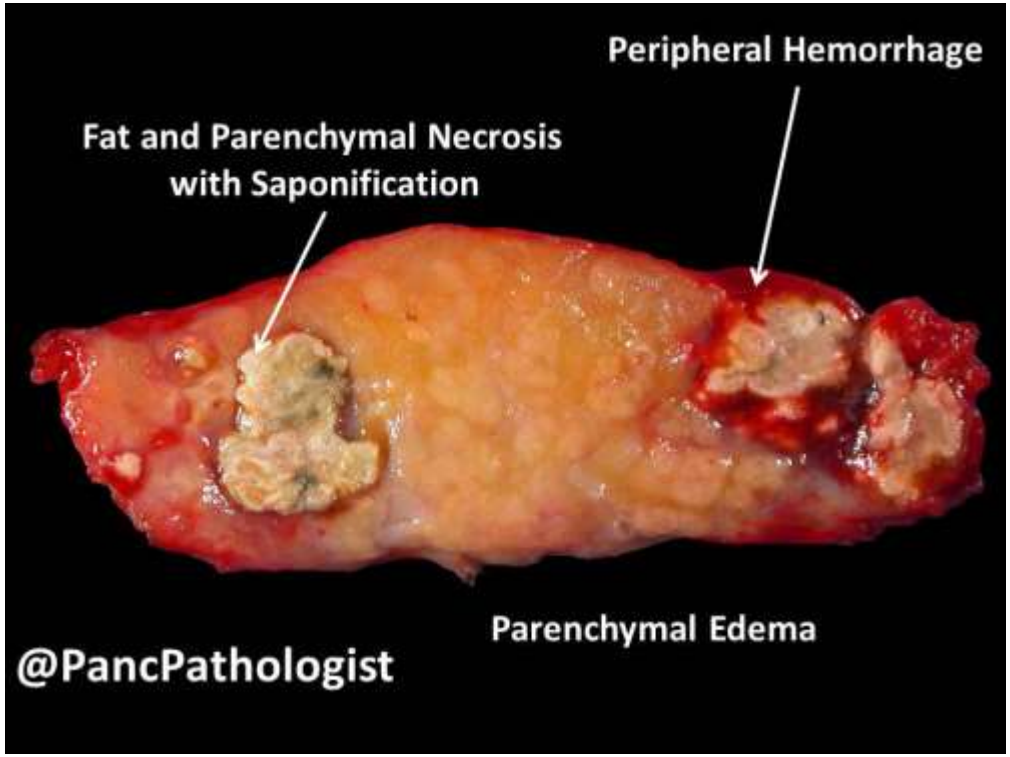
Pathological features

➢ Gross picture:

The gross picture of acute pancreatitis vary from just a swollen and oedematous pancrease to markedly necrotic, destructed and haemorrhagic pancreatic tissue.

yellow plaques representing fat necrosis are seen in the pancreatic and peripancreatic tissues.

The process may spread to the colon.



➢ **Microscopically:**

- The ducts are dilated with degeneration and shedding of the epithelial lining.
- The interstitium shows oedema, leucocytic infiltration, haemorrhage, haemorrhage, necrosis and foci of calcification.

Complications of acute pancreatitis

- The condition may proceed to chronic pancreatitis.
- The necrotic pancreatic tissue may undergo secondary infection, the condition is called infected pancreatic necrosis, which is the most common, most severe and most lethal complication of acute pancreatitis.
- The inflammatory process may progress to the neighboring colon resulting in localized ileus, stenosis, perforation or fistulous formation.

Pancreatic ductal adenocarcinoma

Ductal adenocarcinoma of the exocrine pancreas representing about 90% of all cases of pancreatic malignancy.

Epidemiology:

- ✓ Most patients are elderly.
- ✓ Slight male predominance (1.6:1 ratio).

Risk factors

Risk factors of pancreatic duct adenocarcinoma are poorly understood but the most probable risk factors are **smoking and chronic pancreatitis.**

Presenting features

Symptoms and signs of pancreatic ductal adenocarcinoma depend on location of tumor within the pancreas as well as extent of the disease at time of diagnosis.

- Vague abdominal pain in the upper abdomen radiating to the back, nusea and weight loss.
- Carcinomas located in the head of pancreas may obstruct the common bile duct resulting in progressive obstructive jaundice associated with pain.
- Carcinoma in body and tail usually grows insidiously and already have metastasis at time of diagnosis.
- Pancreatic ductal adenocarcinoma is associated with deep venous thrombosis in about 20% of patients. This sign may be due to release of tumor necrosis factor, interleukin-1 and interleukin-6 by macrophages present in the tumor tissue.

Pathological features

Gross picture:

- Pancreatic duct adenocarcinoma is located in the head of pancreas in two thirds of patients. In the body or tail in the remaining one third of patients.
- The tumor is usually poorly demarcated, firm mass with greyish-yellow cut surface. If the tumor developed on top of chronic pancreatitis; determination of the tumor from the surrounding pancreatic tissue is extremely difficult. The tumor may undergo massive cystic change.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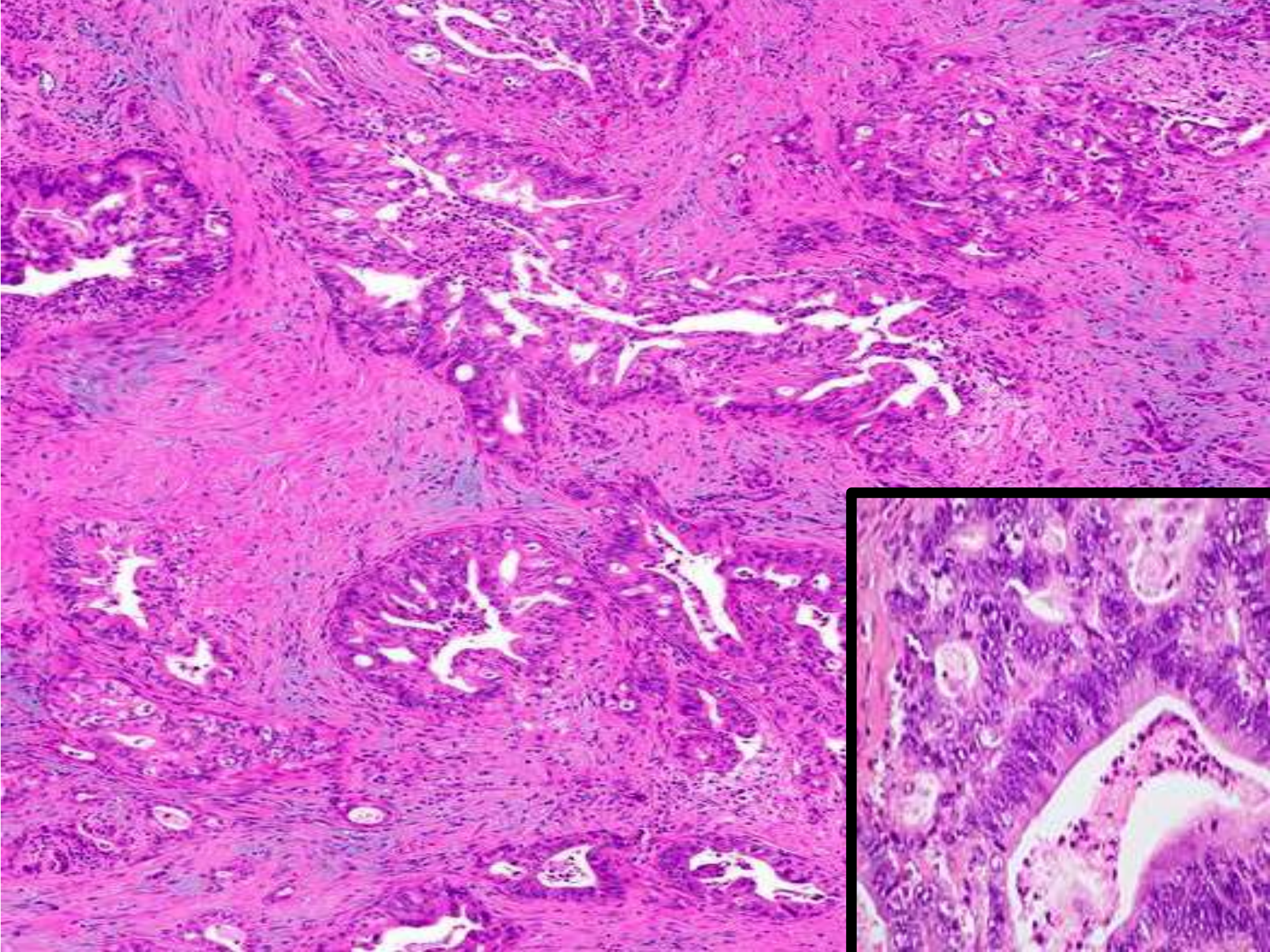
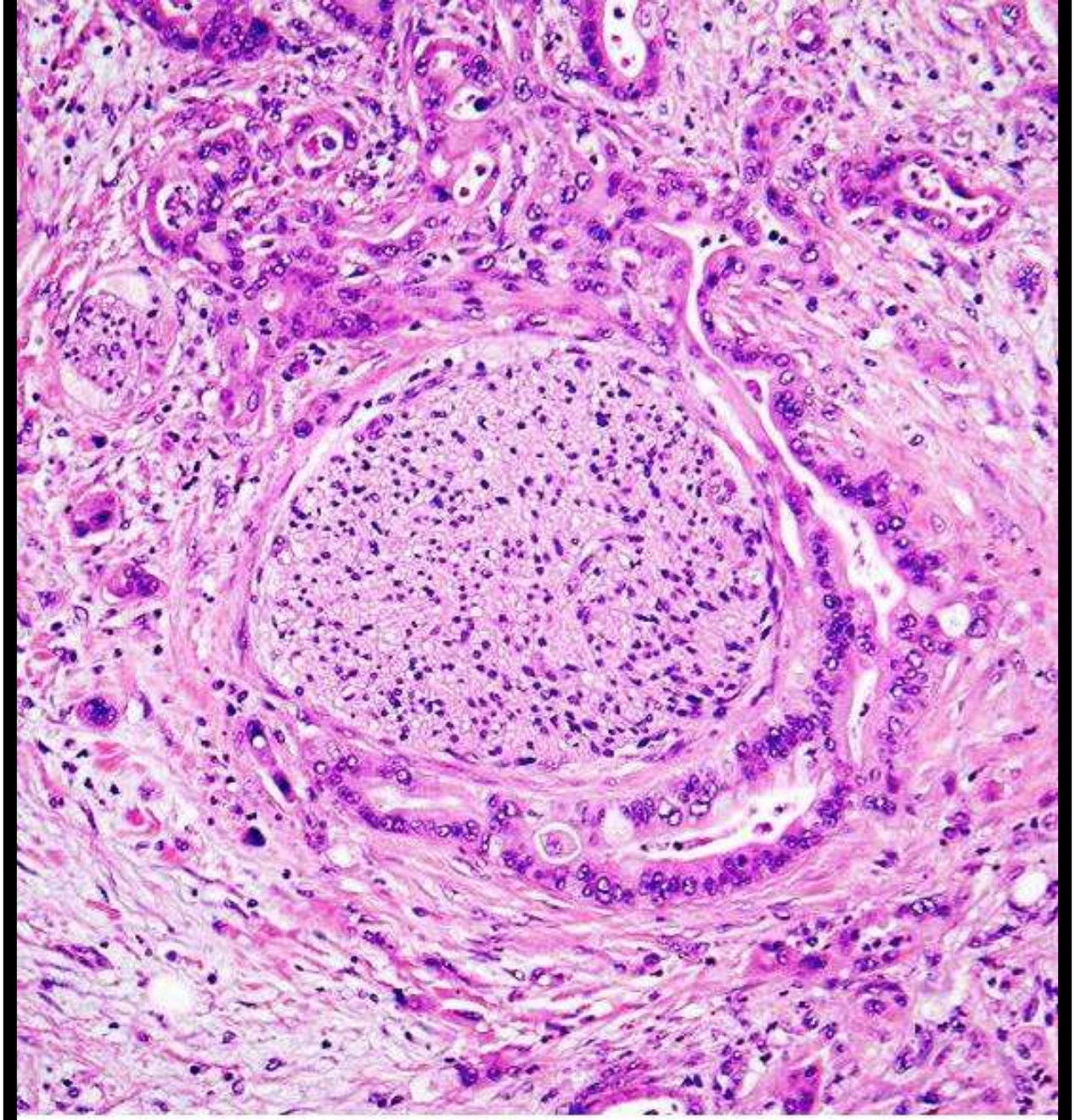
Microscopically:

Pancreatic duct adenocarcinoma is formed of infiltrating, haphazardly distributed glands and ducts surrounded by desmoplastic stroma. They are lined by neoplastic epithelial cells showing criteria of malignancy (pleomorphism, hyperchromatism, abnormal mitosis).

- **Perineural invasion is present in about 90% of cases, vascular invasion in 50% of cases.**





Perineural invasion

Ascites

Ascites means accumulation of fluids usually serous in the peritoneal cavity. The fluid may be a transudate or exudate.

- **Transudative ascites:** it is caused by;
  - As a part of generalized oedema (cardiac, renal or nutrional).
  - Portal hypertension secondary to liver cirrhosis, bilharzial hepatic fibrosis.
  - Constrictive pericarditis.
- **Exudative ascites:**
  - As in cases of tuberculosis of serous sacs.
  - Malignancy in abdominal organs, here the ascetic fluid is usually haemorrhagic.

Reference books

- Elsevier's Integrated Pathology (236- 244), (244- 246)
- USMLE Step 1: Pathology (15-1: 15-20) (16-1: 16-5)
- Robbins and Cotran Pathologic Basis of Disease


Thank you