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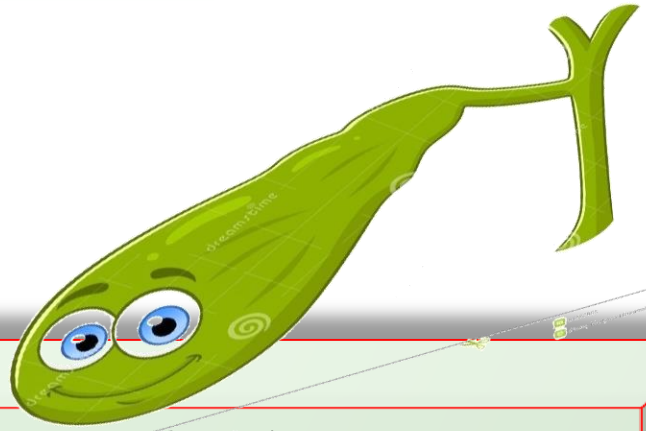

Gallstone Disease and Acute Cholecystitis

Prepared By:

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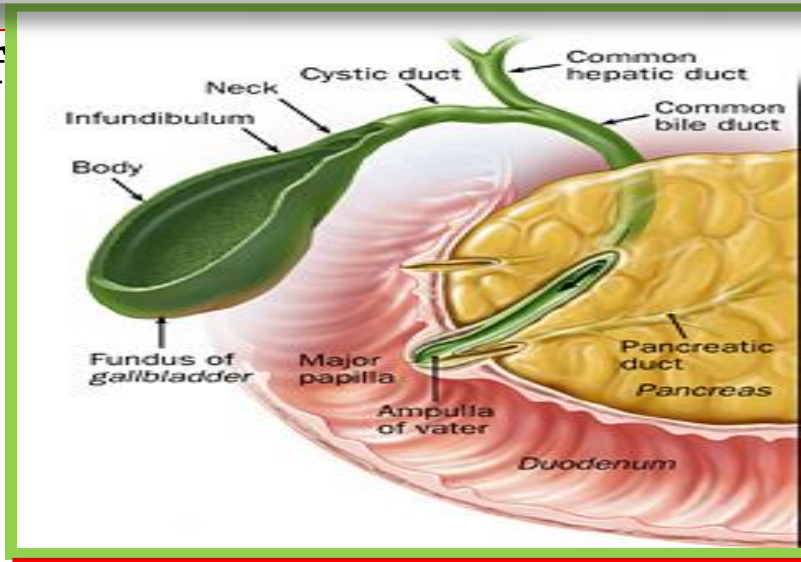
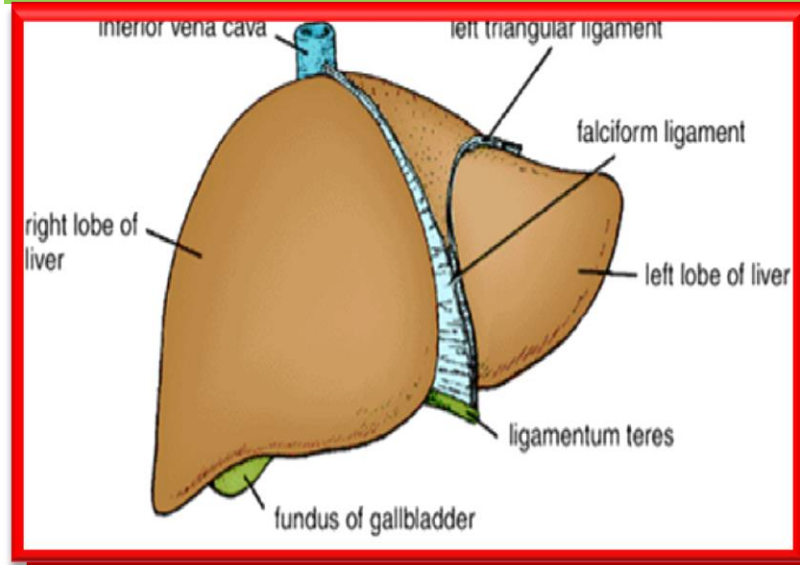
Anatomy Gallbladder

- A pear-shaped sac lying on the undersurface of the liver.
- It has a capacity of 30 to 50 ml and stores bile, which it concentrates by absorbing water.
- The gallbladder is divided into the fundus, body, and neck.
  - **The <u>fundus</u> is rounded and projects below the inferior margin of the liver, where it comes in contact with the anterior abdominal wall at the level of the tip of the ninth right costal cartilage.**
  - **The <u>body</u> lies in contact with the visceral surface of the liver and is directed upward, backward, and to the left.**
  - **The <u>neck</u> becomes continuous with the cystic duct, which turns into the lesser omentum, joins the common hepatic duct, to form the bile duct**



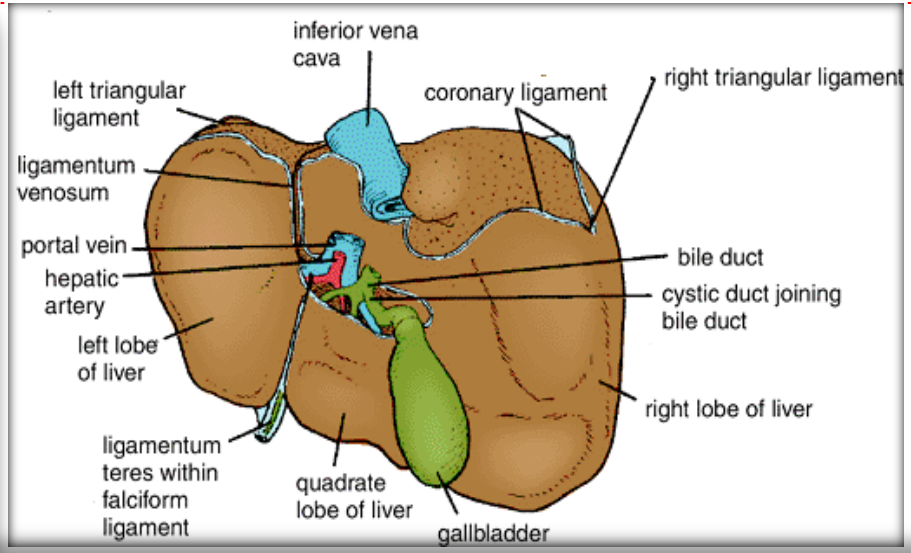


Gallbladder

- The peritoneum completely surrounds the fundus of the gallbladder and binds the body and neck to the visceral surface of the liver.

Relations

- Anteriorly: The anterior abdominal wall and the inferior surface of the liver
- Posteriorly: The transverse colon and the first and second parts of the duodenum



Function of the Gallbladder

- When digestion is not taking place, the sphincter of Oddi remains closed and bile accumulates in the gallbladder. The gallbladder concentrates bile; stores bile; selectively absorbs bile salts, keeps the bile acid; excretes cholesterol; and secretes mucus. To aid in these functions, the mucous membrane is thrown into permanent folds that unite with each other, giving the surface a honeycombed appearance.

Blood Supply

- The cystic artery is a branch of the right hepatic artery.
- The cystic vein drains directly into the portal vein.
- Several very small arteries and veins also run between the liver and gallbladder.

Lymph Drainage

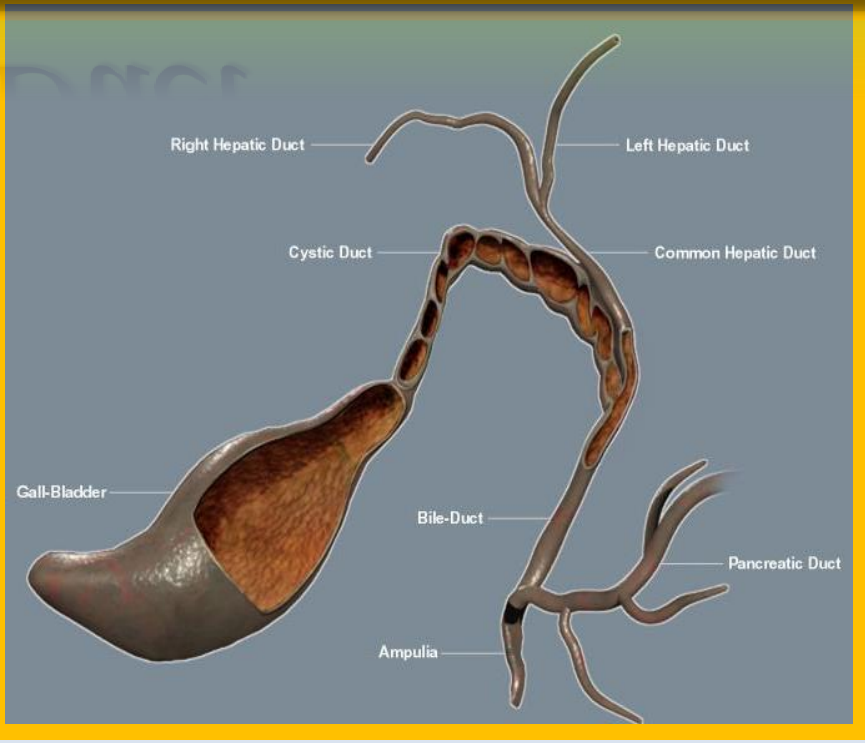
- The lymph drains into a cystic lymph node situated near the neck of the gallbladder. From here, the lymph vessels pass to the hepatic nodes along the course of the hepatic artery and then to the celiac nodes.

Nerve Supply

- Sympathetic and parasympathetic vagal fibers form the celiac plexus. The gallbladder contracts in response to the hormone cholecystokinin, which is produced by the mucous membrane of the duodenum on the arrival of fatty food from the stomach.

Cystic Duct

- The cystic duct is about 1.5 in. (3.8 cm) long and connects the neck of the gallbladder to the common hepatic duct to form the bile duct.
- It usually is somewhat S-shaped and descends for a variable distance in the right free margin of the lesseromentum.



- The mucous membrane of the cystic duct is raised to form a spiral fold that is continuous with a similar fold in the neck of the gallbladder. The fold is commonly known as the "spiral valve." The function of the spiral valve is to keep the lumen constantly open.

Gallbladder and Bile Ducts

- **Normal size of gallbladder:**

  7~9cm in length ;

  3~4cm in width;

  Wall thickness : 2~3mm

- **Normal size of bile ducts :**

  CBD:≥8mm =dilated

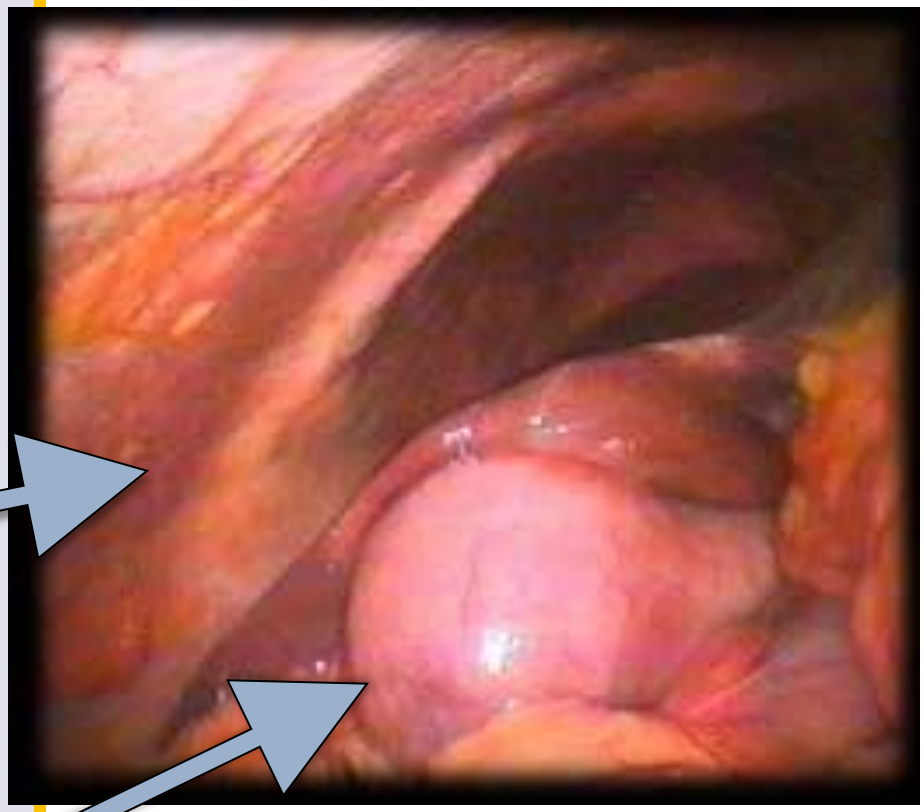
right /left intrahepatic duct just to proximal CHD: 2-3mm ;

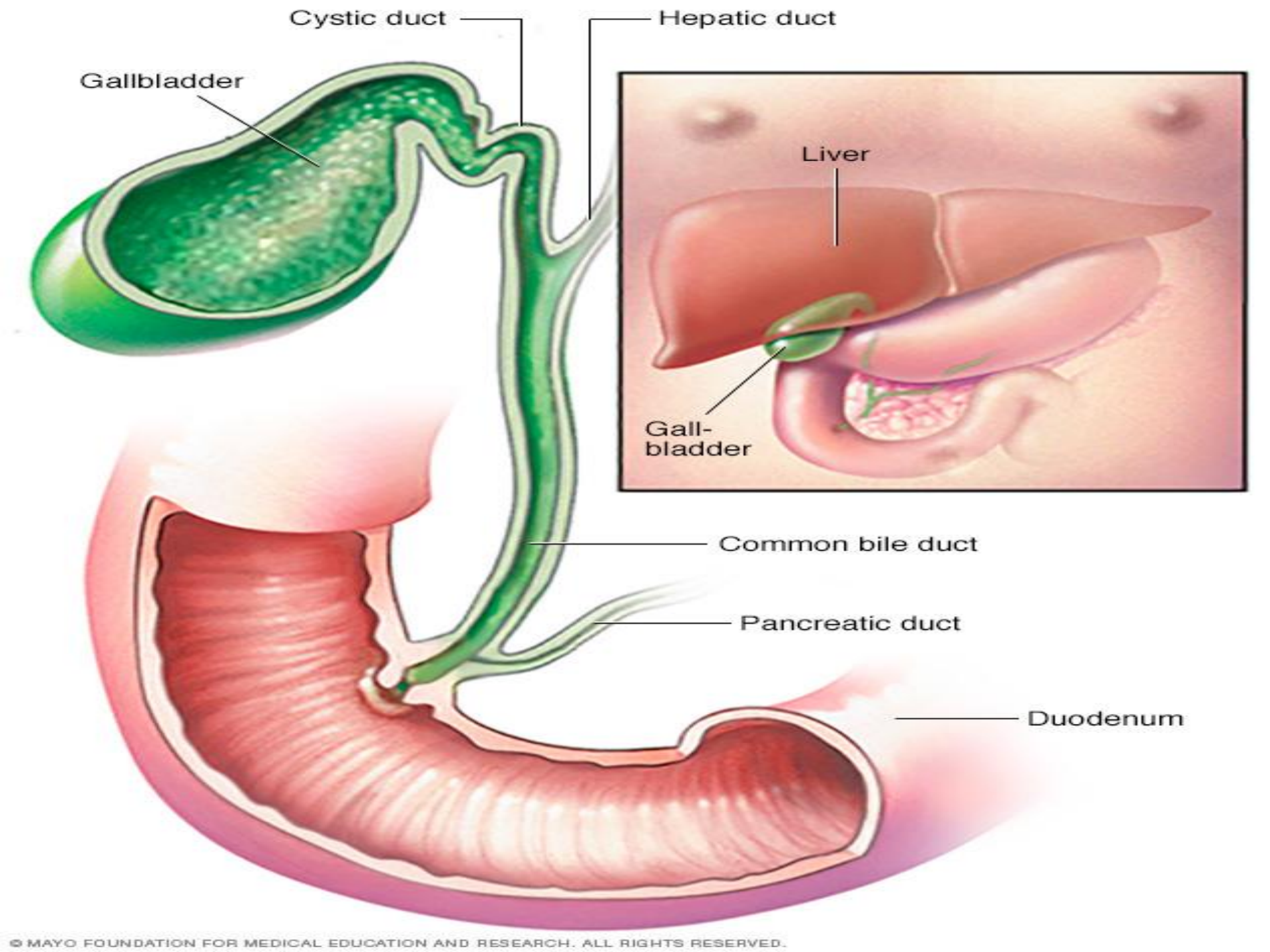
Definition of term

Symptomatic cholelithiasis	Wax/waning postprandial epigastric/RUQ pain due to transient cystic duct obstruction by stone, no fever/WBC, normal LFT
Acute cholecystitis	Acute GB inflammation due to cystic duct obstruction. Persistent RUQ pain +/- fever, ↑WBC, ↑LFT, +Murphy's = inspiratory arrest
Chronic cholecystitis	Recurrent bouts of colic/acute chol'y leading to .chronic GB wall inflamm/fibrosis. No fever/WBC
Acalculous cholecystitis	GB inflammation due to biliary stasis(5% of time) and not stones(95%). Seen in critically ill pts
Choledocho-lithiasis	Gallstone in the common bile duct (primary)means originated there, secondary = from GB
Cholangitis	Infection within bile ducts usu due to obstrux of CBD. Charcot triad: RUQ pain, jaundice, fever (seen in 70% of pts), can lead to septic shock

What is it?

- By definition, **cholecystitis** is an inflammation of the gallbladder wall and nearby abdominal lining.

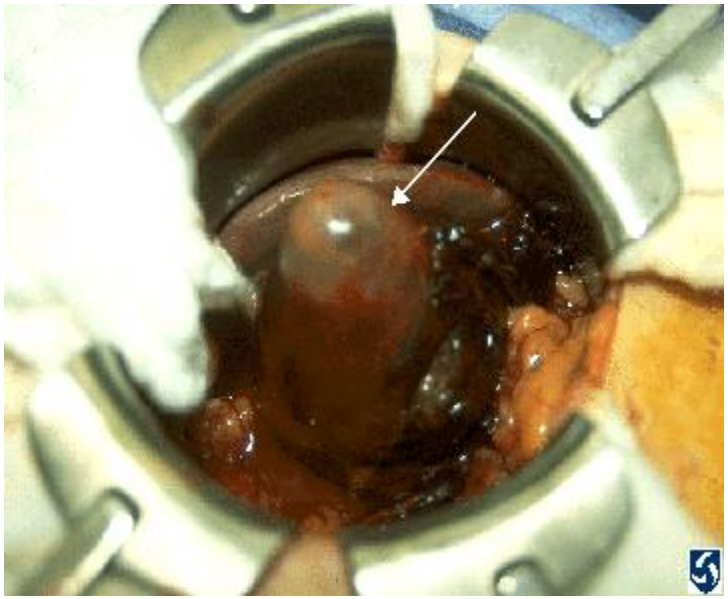




Etiology / Pathophysiology

- Can be caused by an obstruction, gallstone or a tumor.
  - 90% of all cases caused by gallstones.
  - The exact cause of gallstone formation is *unknown*.
- When there is an obstruction, gallstone or tumor it prevents bile from leaving the gallbladder.
  - Bile gets trapped and acts as an irritant which causes cellular infiltration within 3 – 4 days.

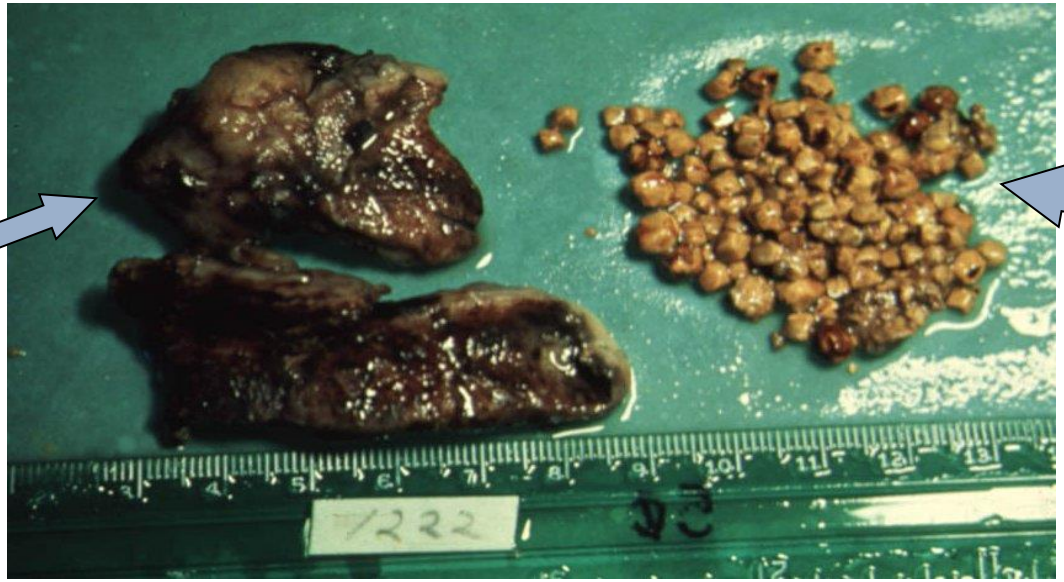
- This infiltration causes an inflammatory process – the gallbladder becomes enlarged and edematous.
  - Eventually this occlusion along with bile stasis causes the mucosal lining of the gallbladder to become necrotic.
  - Bacterial growth occurs due to ischemia.



Necrotic Gallbladder

- Rupture of the gallbladder becomes a danger, along with spread of infection of the hepatic duct and liver.
- If the disease is severe and interferes with the blood supply it can cause the gallbladder to become gangrenous.

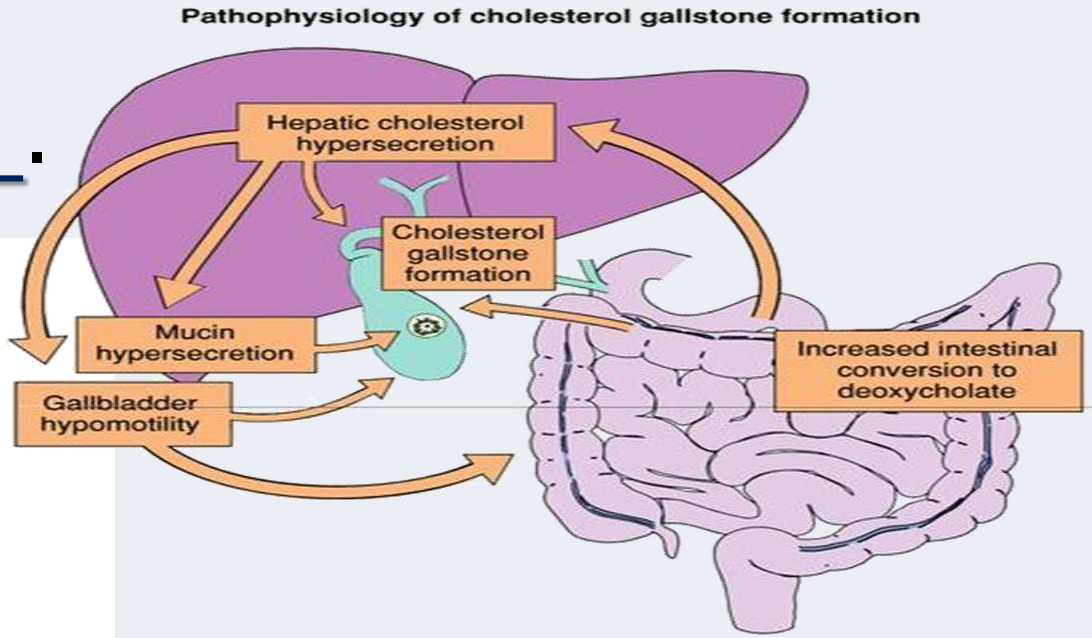
Gangrenous gallbladder

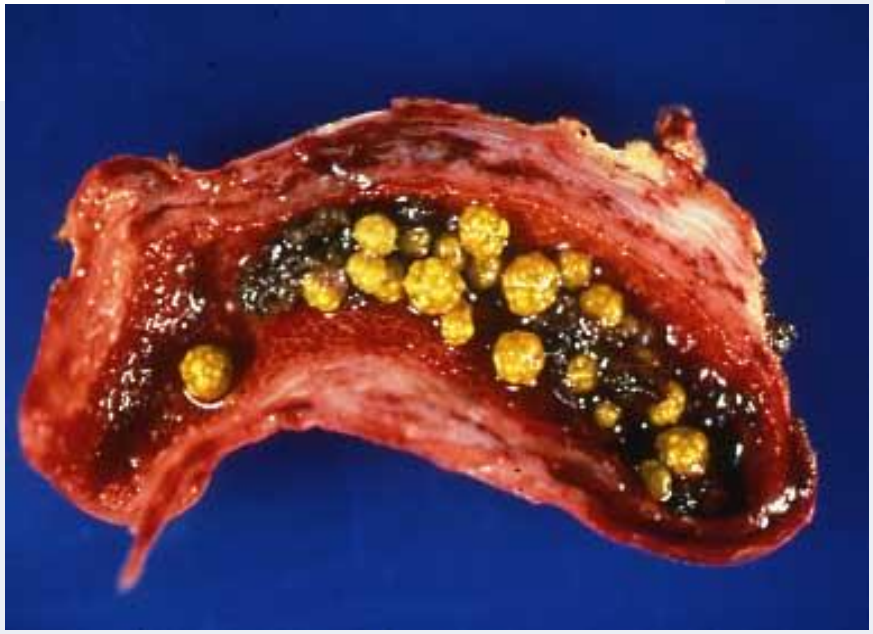


Gallstones

Gallstones .

- The presence of gallstones in the gallbladder is called Cholelithiasis.





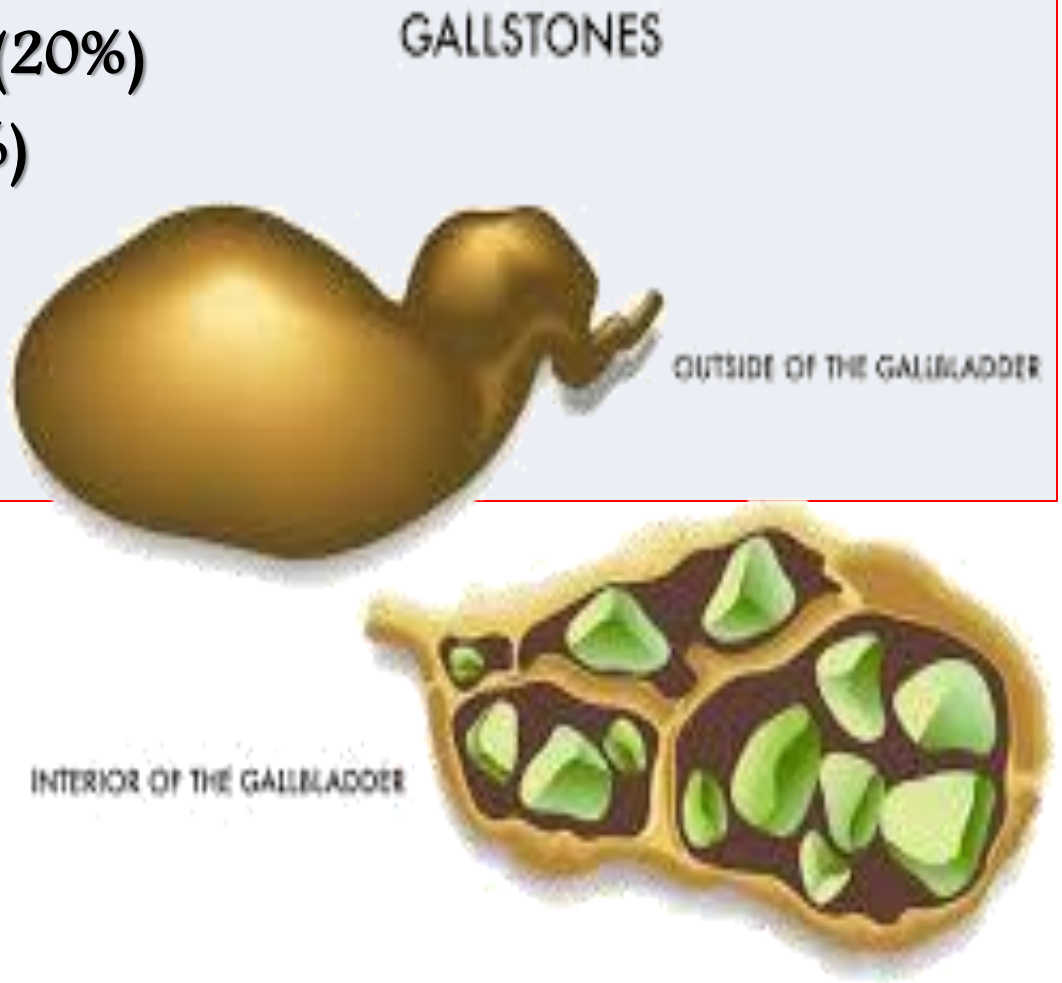
Pathogenesis

Composition of bile:

- Bilirubin (by-product of haem degradation)
- Cholesterol (kept soluble by bile salts and lecithin)
- Bile salts/acids (cholic acid/chenodeoxycholic acid): mostly reabsorbed in terminal ileum(entero-hepatic circulation).
- Lecithin (increases solubility of cholesterol)
- Inorganic salts (sodium bicarbonate to keep bile alkaline to neutralise gastric acid in duodenum)
- Water (makes up 97% of bile)

Types of gallstone

- Cholesterol stones (20%)
- Pigment stones (5%)
- Mixed (75%)



Epidemiology

- Approximately 12% of men and 24% of women of all ages have gallstones
- 80% are asymptomatic
- 2–3% of patients progress per year to symptomatic disease
- 1% of patients with gallstones develop acute complications
- Approximately 12% of patients undergoing cholecystectomy found to have CBD stones

Pathogenesis

Cholesterol

- Imbalance between bile salts/lecithin and cholesterol allows cholesterol to precipitate out of solution and form stones

Pigment

- Occur due to excess of circulating bile pigment (e.g. Heamolytic anaemia)

Mixed

- Same pathophysiology as cholesterol stones

Risk factors for gallstone formation

- **Age**
  - Risk is x4 between the ages of 40–69 compared with younger subjects
  - Due to increased cholesterol content in bile
- **Sex**
  - Higher prevalance in women, up to x3 between ages of 30–39
- **Pregnancies / hormones**
  - Related to frequency and number of pregnancies
  - New biliary sludge may form in up to 30% of women
  - Oestrogens promote cholesterol hypersecretion in bile and reduce bile acid synthesis

Risk factors for gallstone formation

- Progesterones promote stasis and impair contractility

- **Oral contraceptives and HRT**
  - As above
  - Also found to apply to men receiving oestrogen therapy for prostate cancer, compared to those who elected for orchiectomy (small study)
- **Obesity**
  - Enhanced cholesterol synthesis and secretion

Risk factors for gallstone formation

- **Gallbladder stasis**
  - Fasting states
  - Rapid weight loss
  - Major trauma
  - Somatostatin
  - Due to excessive reabsorption of water with resultant cholesterol supersaturation
- **Rapid weight loss**
  - Increases bile calcium concentration
  - Increases bile mucin concentration

Risk factors for gallstone formation

- **Cirrhosis**
  - Overall prevalance approaches 30%
  - Higher incidence with Childs B and C disease
  - High unconjugated bilirubin levels
  - High circulating oestrogen levels (aromatase)

- **Impaired enterohepatic circulation**
  - Small bowel resection
  - Crohn's disease
  - Reduced levels of bile acid content in bile, leading to poor cholesterol solubility

Risk factors for gallstone formation

- **Drugs**
  - Ceftriaxone (biliary excretion, forms a complex with calcium and precipitates)
  - Clofibrate (impairs bile acid formation, leading to supersaturation)

Physical inactivity / sedentary lifestyle

Risk factors for gallstone formation

- **Increased circulating unconjugated bilirubin**
  - Haemolytic states
  - Cirrhosis
  - Hypersplenism
  - High-turnover haematological disease
- **Genetic factors / ethnicity**
  - Pima Native Americans have incidence of up to 75%
  - Chilean
  - Mexican

Gallstone formation – in summary

- **Imbalance of bile content**
  - Cholesterol supersaturation
  - Too much unconjugated bilirubin
  - Inadequate bile salt content
- **Gall bladder stasis**

Protective factors

- Statins
- Aspirin
- Vitamin C (but only for women!)
- Coffee (>3 cups per day), but decaffeinated coffee not protective
- Diet rich in unsaturated fats (mono- and poly-)

Those who are most at risk.

- These are all adjectives to describe the person most at risk of developing symptomatic gallstones.

FAIR

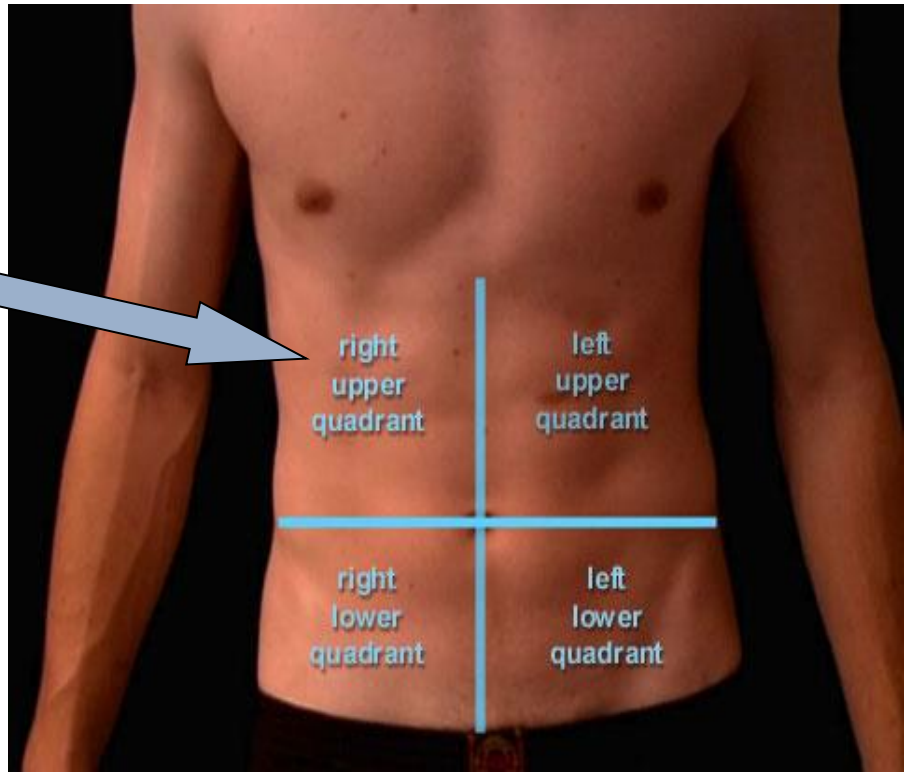
FAT

FORTY

FEMALE

Signs and Symptoms.

- Complaints of indigestion after eating high fat foods.
- Localized pain in the right-upper quadrant epigastric region.
- Anorexia, nausea, vomiting and flatulence.



- Increased heart and respiratory rate – causing patient to become diaphoretic which in turn makes them think they are having a heart attack.

Signs and Symptoms.

- Low grade fever.
- Elevated leukocyte count.
- Mild jaundice.
- Stools that contain fat – *steatorrhea*.
- Clay colored stools caused by a lack of bile in the intestinal tract.
- Urine may be dark amber- to tea-colored.

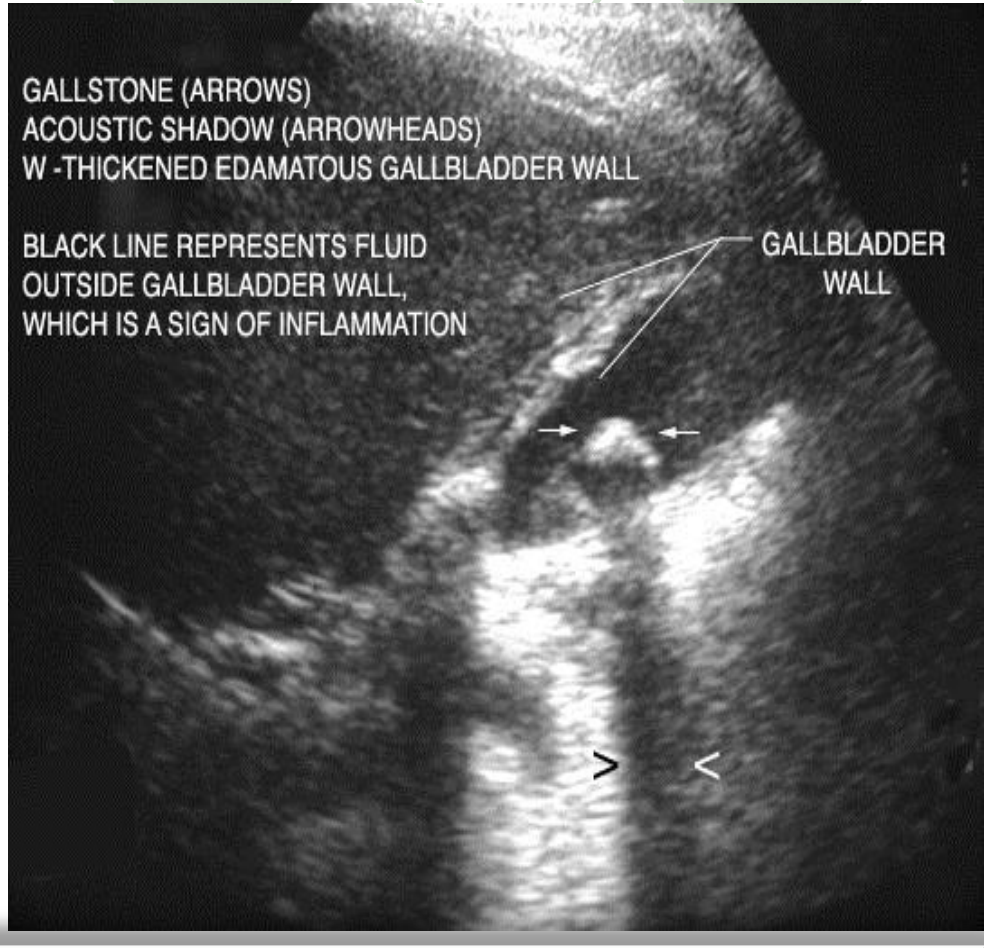
CHOLECYSTITIS

physical exam

Murphy's Sign

Diagnostics

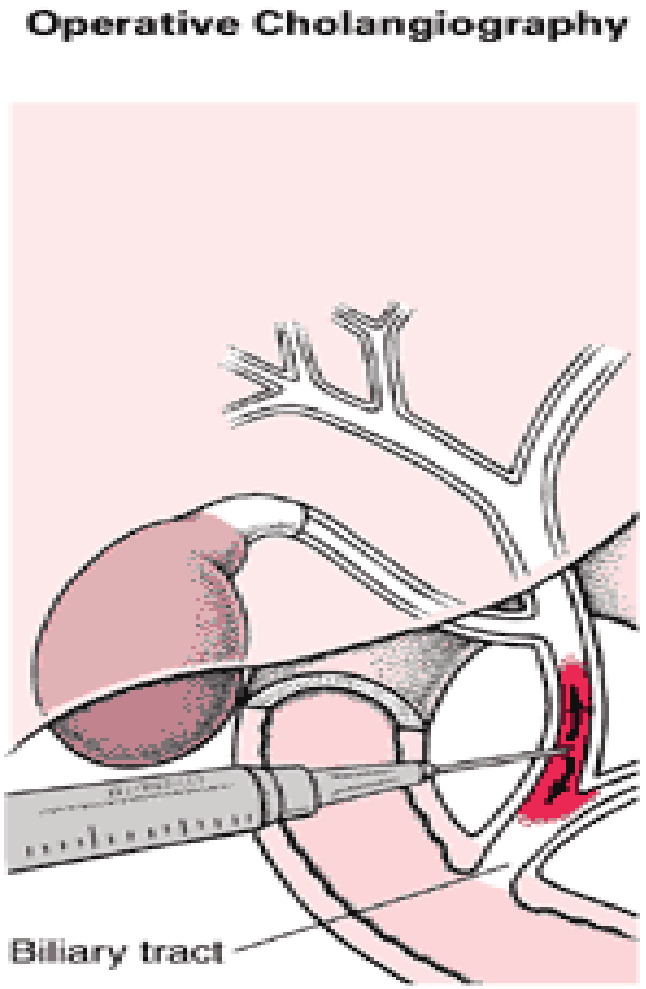
- Fecal studies.
- Serum bilirubin tests.
- Ultrasound of the gallbladder.



USS: first line investigation in gallstone disease □Confirms presence of gallstones □ Gall bladder wall thickness (if thickened suggests cholecystitis) □ Biliary tree calibre (CBD/extrahepatic/intrahepatic) □ if dilated suggests stone in CBD (normal CBD <8mm). Sometimes CBD stone can be seen

Diagnostics

- HIDA scan - imaging test used to examine the gallbladder and the ducts leading into and out of the gallbladder - also referred to as **cholescintigraphy**.
- Oral cholecystogram - the patient takes iodine-containing tablets by mouth - iodine is absorbed from the intestine into the bloodstream - removed from the blood by the liver and excreted by the liver into the bile – it is concentrated in the gallbladder - outlines the gallstones that are radiolucent (x-rays pass through them).
- Operative cholangiography – common bile duct is directly injected with radiopaque dye.

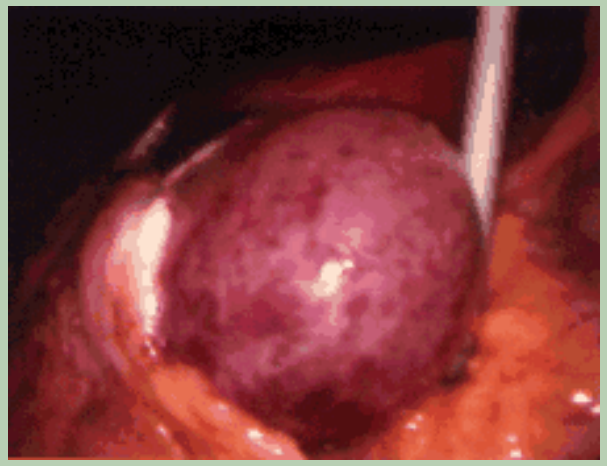


Recap. Stages of Acute Cholecystitis

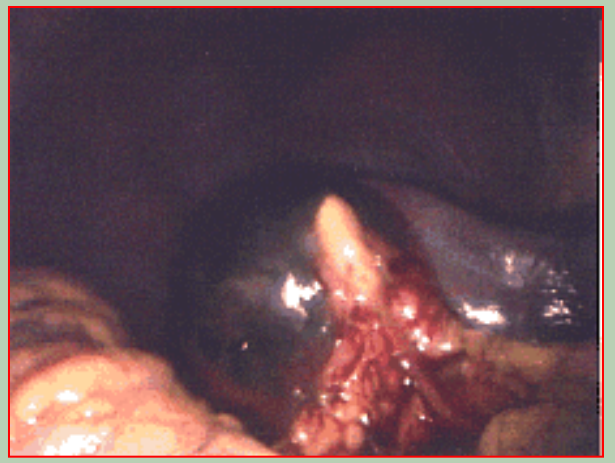
– Gallbladder has a grayish appearance & is edematous.

–There is an obstruction of the cystic duct and the gallbladder begins to swell.

– It no longer has the "robin egg blue" appearance of a normal gallbladder.

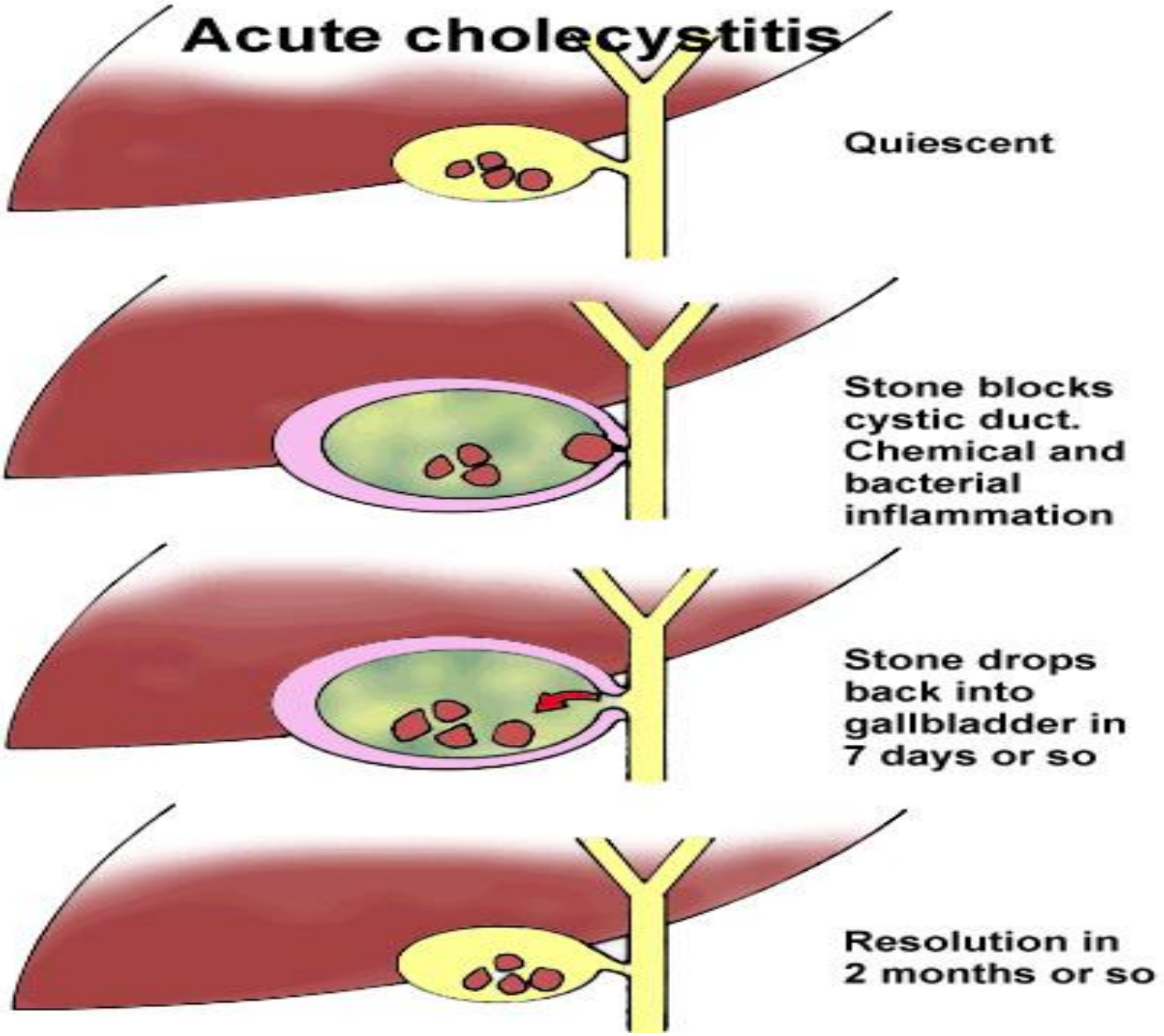


- As acute cholecystitis progresses, the gallbladder begins to become necrotic and gets a speckled appearance as the wall begins to die.

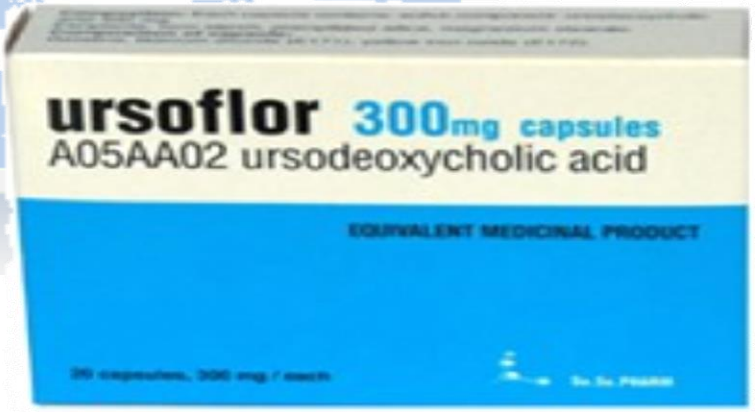


– Gallbladder undergoes gangrenous change and the wall becomes very dark green or black.

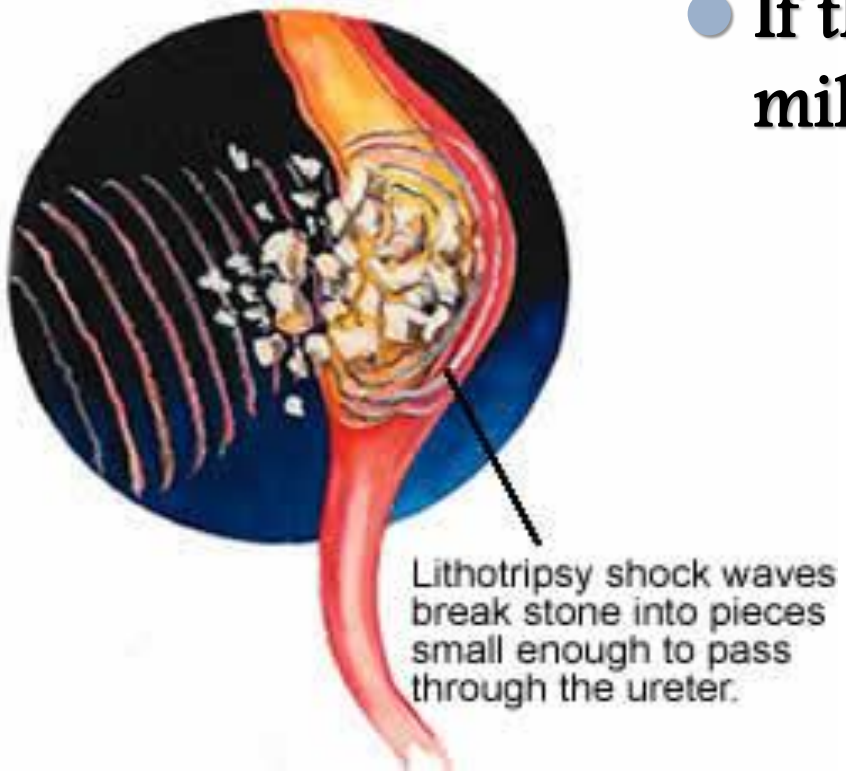
– This is the stage when perforation occurs.


Acute cholecystitis
Quiescent
Stone blocks cystic duct. Chemical and bacterial inflammation
Stone drops back into gallbladder in 7 days or so
Resolution in 2 months or so

Medical Management.

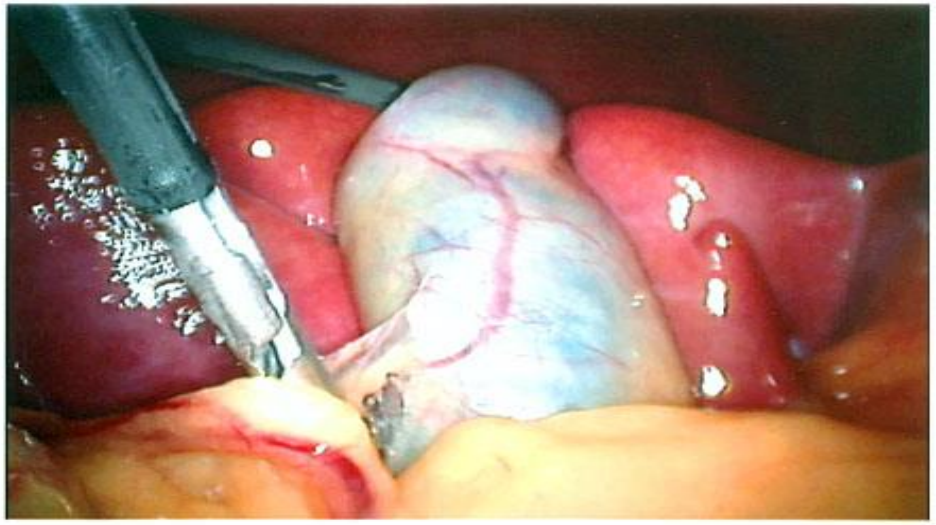


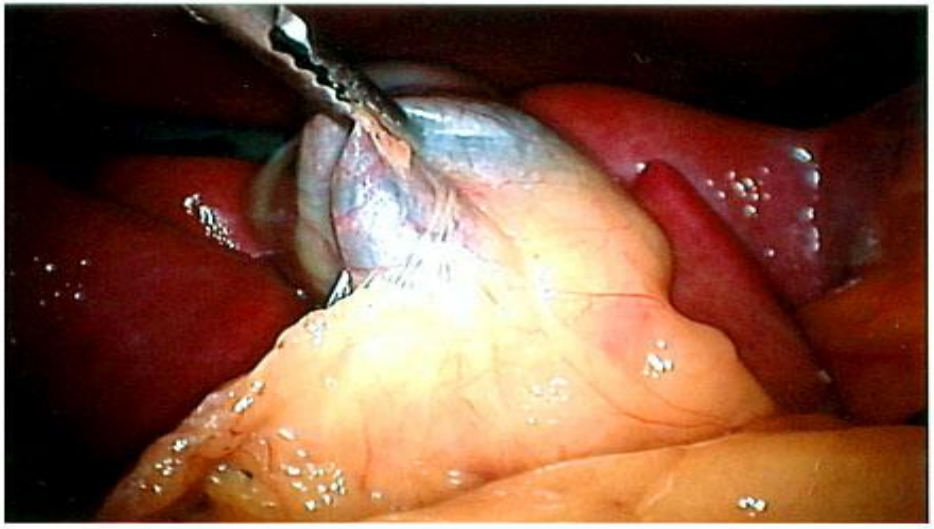
- Lithotripsy
  - for patients with only a *FEW* stones.



- If the attack of cholelithiasis is mild –
  - bed rest is prescribed.
  - patient is placed on NPO to allow GI tract and gallbladder to rest.
  - an NG tube is placed on low suction.
  - fluids are given IV in order to replace lost fluids from NG tube suction.

Surgical Management.





Cholecystectomy

or

Laparoscopic Cholecystectomy

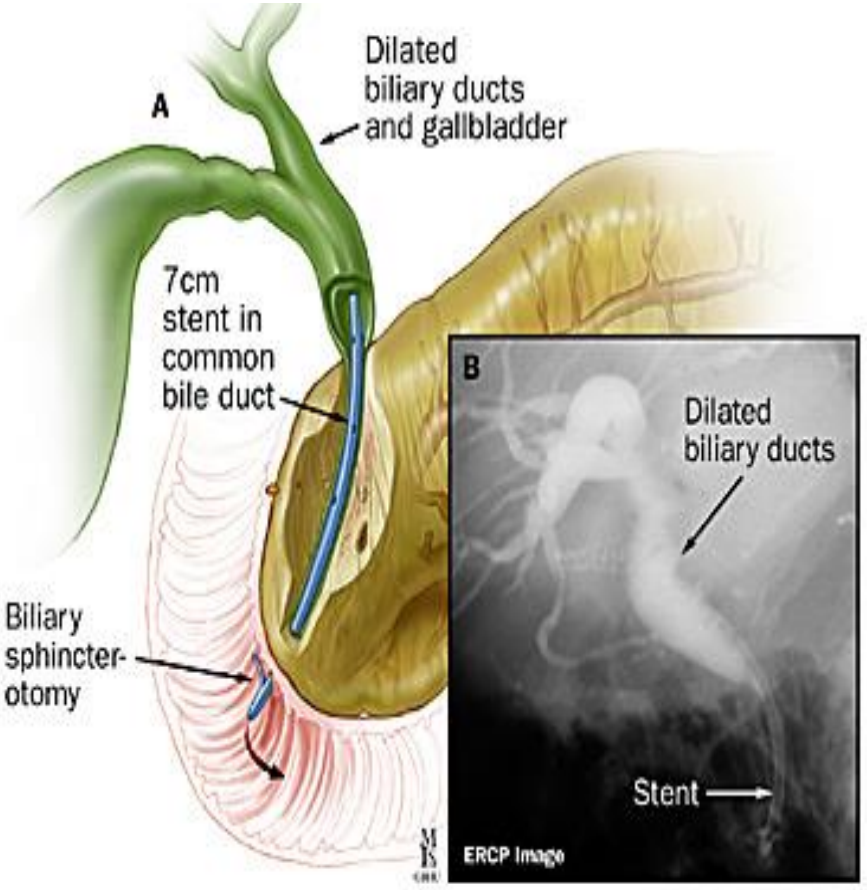
– removal of the gallbladder.

This is the treatment of choice.

The gallbladder along with the cystic duct, vein and artery are ligated.

Medical Management.

- If stones are present in the common bile duct, an **endoscopic sphincterotomy** must be performed to remove them *BEFORE* a cholecystectomy is done.
  - A number of various instruments are inserted through the endoscope in order to "cut" or stretch the sphincter.
  - Once this is done, additional instruments are passed that enable the removal of stones and the stretching of narrowed regions of the ducts.
  - Drains (stents) can also be used to prevent a narrowed area from rapidly returning to its previously narrowed state.



Nursing Interventions
Post Op - Cholesystectomy

1. Administer oral analgesics to facilitate movement and deep breathing – and to stay ahead of pts pain.

2. Observe dressings frequently for exudate and hemorrhage.

3. Vitals are routinely checked.

4. Patient teaching:
-Must understand how to splint the abd. before coughing.
-Report any abnormalities such as, severe pain, tenderness in RUQ, increase in pulse, etc . .
-Instructed that they usually can return to work in 3 days & can resume full activity in 1 week.

5. Fluid balance is maintained IV – potassium added to compensate for loss from surgery.

Nursing Interventions

Interventions center on keeping patient comfortable by carefully administering meds and watching for reactions.

1. Urine and stool should be observed for alterations in the presence of bilirubin.

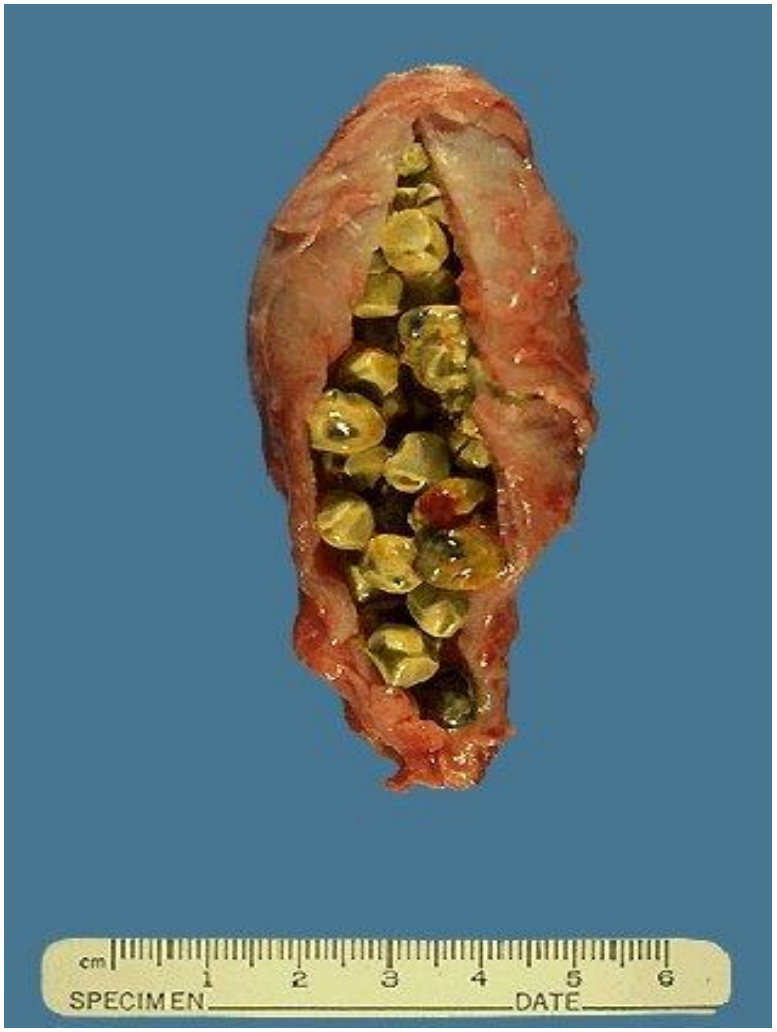
2. NG tube must be monitored for amount, color & consistency of output.
Also, tube must be on LOW suction and nasal area should be monitored for irritation and necrosis.

3. Anti-emetics may be administered if nausea persists.

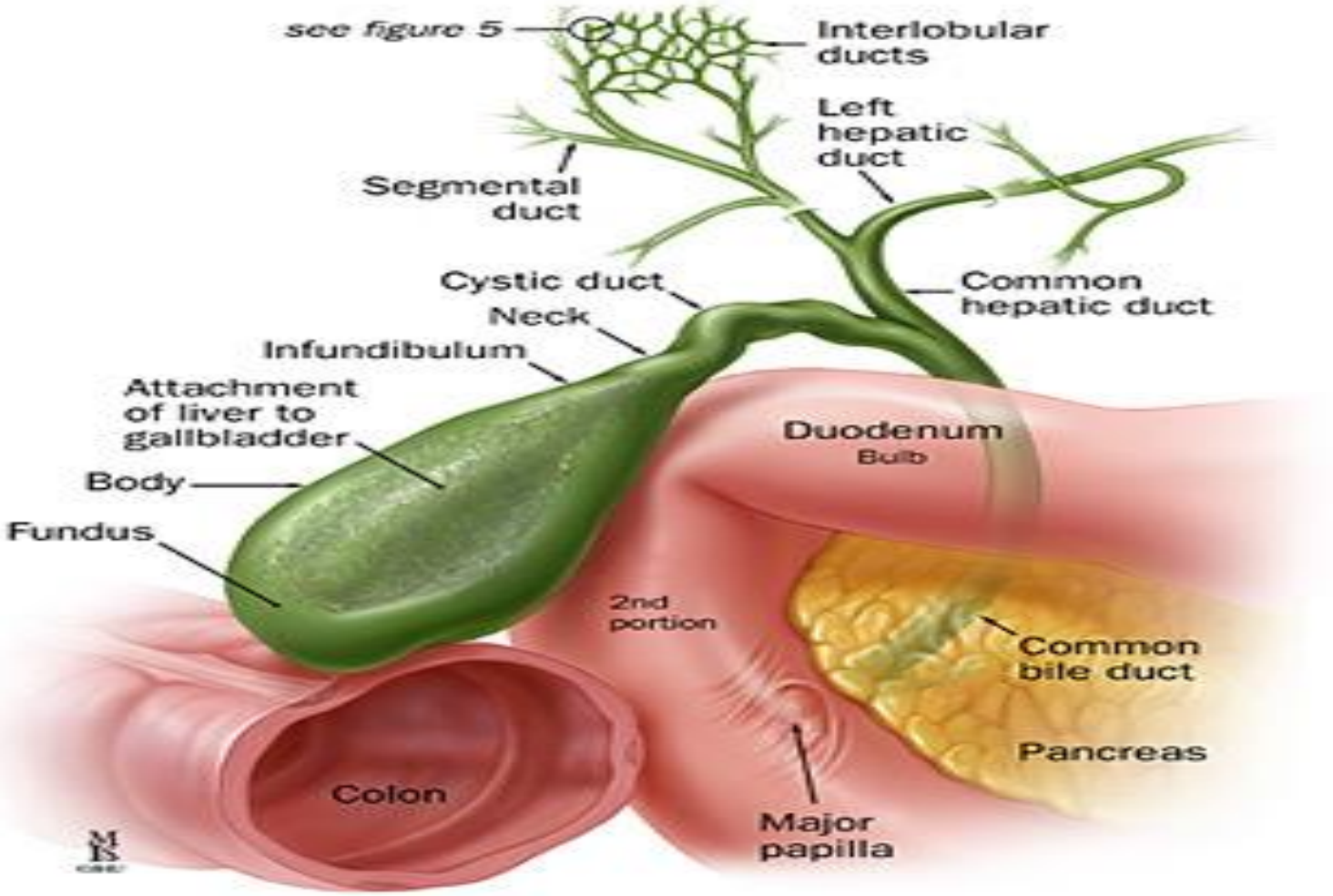
4. I & O are measured and described carefully.

5. Pt. must understand how to splint the abdomen for post op coughing, turning and deep breathing.

Eww!



Complications of Gallstones



Obstructive Jaundice

Pathogenesis:

Stone obstructing CBD (bear in mind there are other causes for obstructive jaundice) – danger is progression to ascending cholangitis.

USS

Will confirm gallstones in the gallbladder
CBD dilatation i.e. >8mm (not always!)
May visualise stone in CBD (most often does not)

MRCP

In cases where suspect stone in CBD but USS indeterminate
E.g.1 obstructive LFTs but USS shows no biliary dilatation and no stone in CBD

Obstructive Jaundice

ERCP

If confirmed stone in CBD on USS or MRCP proceed to ERCP which will confirm this (diagnostic) and allow extraction of stones and sphincterotomy (therepeutic)

Treatmen

Must unobstruct biliary tree with ERCP to prevent progression to ascending cholangitis Whilst awaiting ERCP monitor for signs of sepsis suggestive of cholangitis

Ascending Cholangitis

Pathogenesis:

- Stone obstructing CBD with infection/pus proximal to the blockage

Treatment

- ABC
- Fluid resuscitation (clear fuids and IVF, catheter)
- Antibiotics (Augmentin)
- HDU/ITU if unwell/septic shock
- Pus must be drained* – this is done by decompressing the biliary tree
  - Urgent ERCP
  - Urgent PTC – if ERCP unavailable or unsuccesful

Acute Pancreatitis

Pathogenesis

- Obstruction of pancreatic outflow
  - Pancreatic enzymes activated within pancreas
  - Pancreatic auto-digestion

USS: to confirm gallstones as cause of pancreatitis

- USS not good for visualising pancreas

CT: gold standard for assessing pancreas.

- Performed if failing to settle with conservative management to look for complications such as pancreatic necrosis

Treatment

- Analgesia
- **Fluid resuscitation**
- Pancreatic rest – clear fluids initially
- Identify underlying cause of pancreatitis

- 95% settle with above conservative management
- 5% who do no settle or deteriorate need CT scan to look for pancreatic necrosis

Gallstone ileus

Pathogenesis:

- Gallstone causing small bowel obstruction (usually obstructs in terminal ileum)
- Gallstone enters small bowel via cholecysto-duodenal fistula (**not** via CBD)

AXR – dilated small bowel loops

- May see stone if radio-opaque

Treatment

- NBM
- **Fluid resuscitation** + catheter
- NG tube
- Analgesia
- **Surgery** (will not settle with conservative management) – enterotomy + removal of stone

Diagnosis of gallstone ileus usually made at the time of surgery.