

THEORY

By Razan Hammad

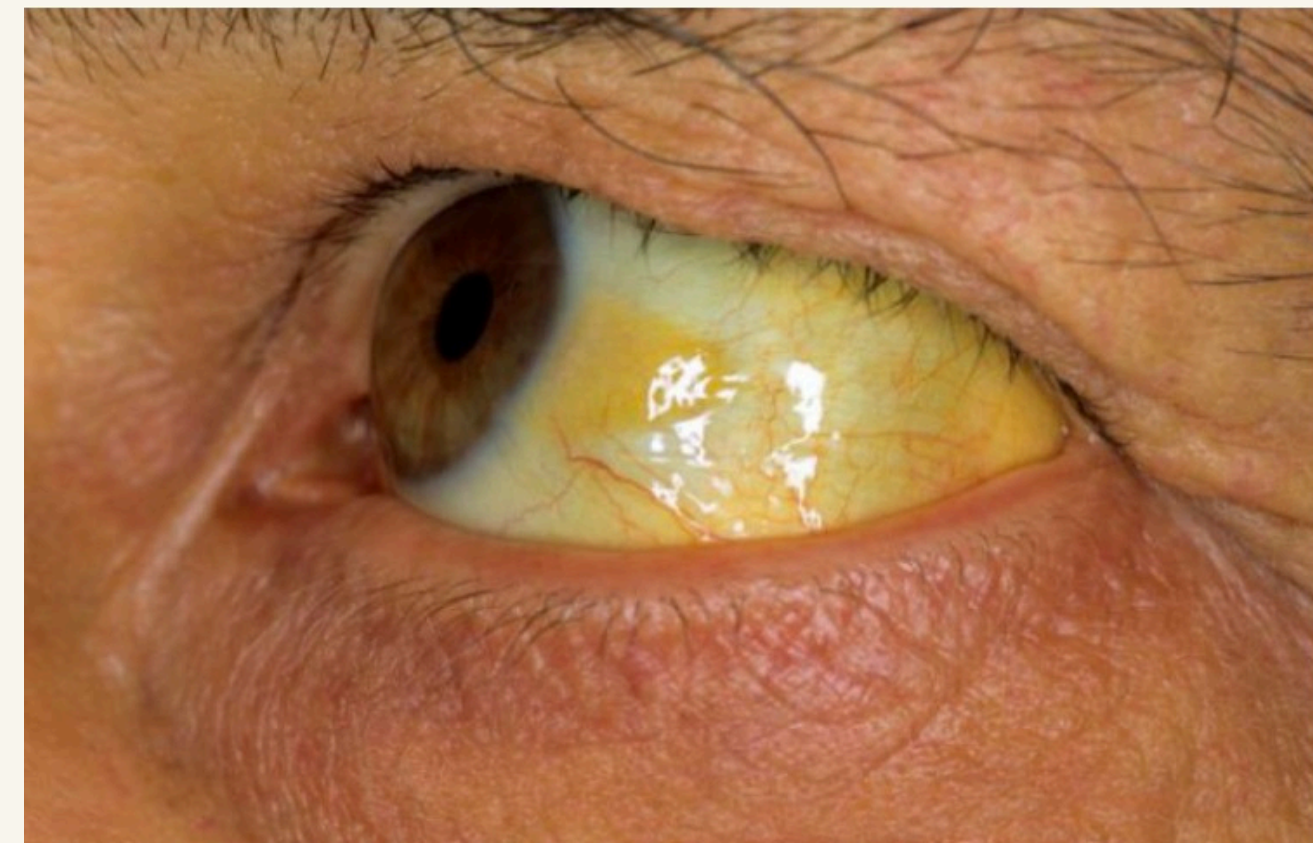
Introduction

Definition

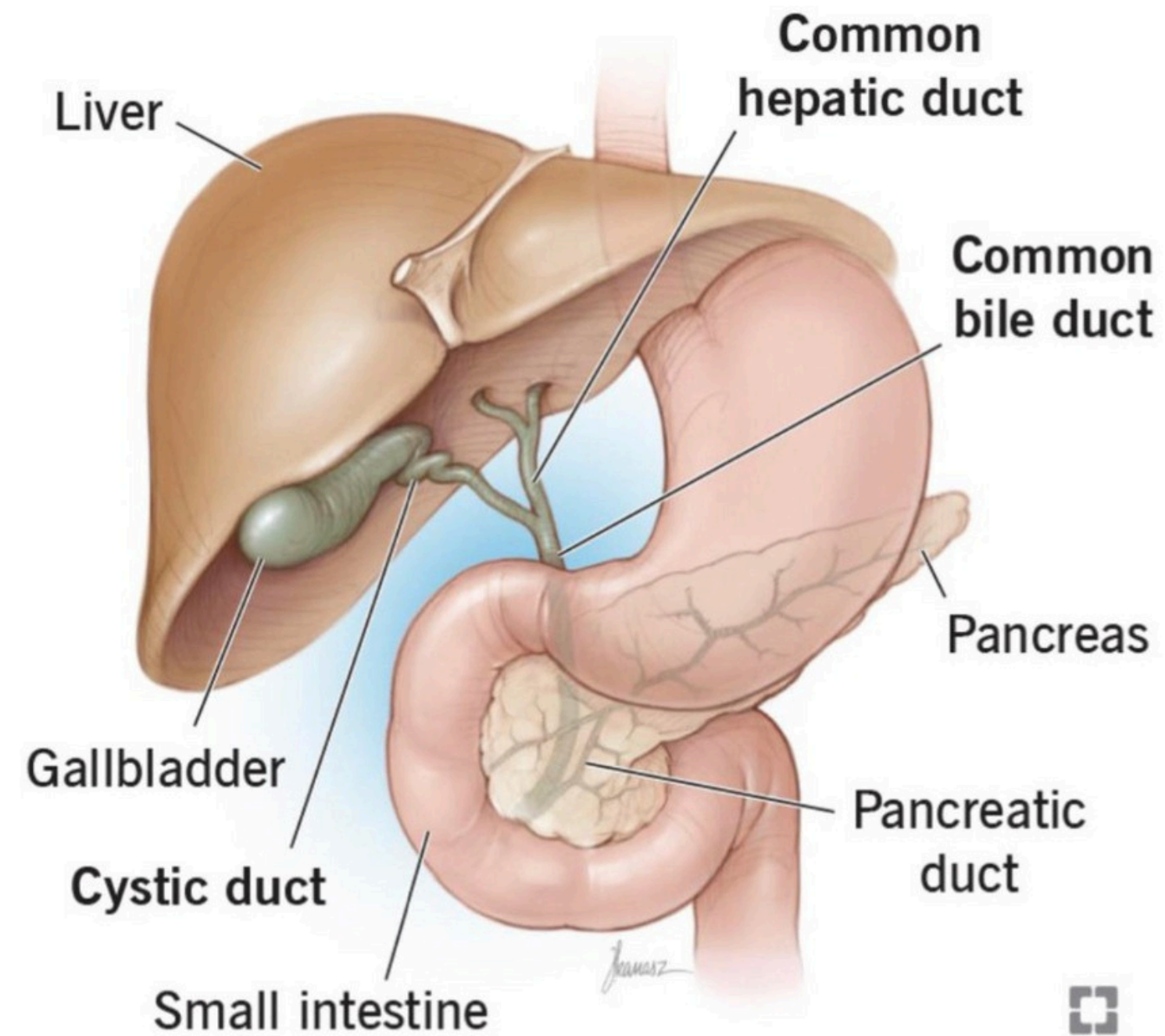
Jaundice (Icterus) is the yellowish discoloration of the skin, sclera and mucous membranes caused by accumulation of Bilirubin.

There is no absolute level at which jaundice is clinically detected but, in good light, most clinicians will recognize jaundice when bilirubin levels exceed 3 mg/dL.

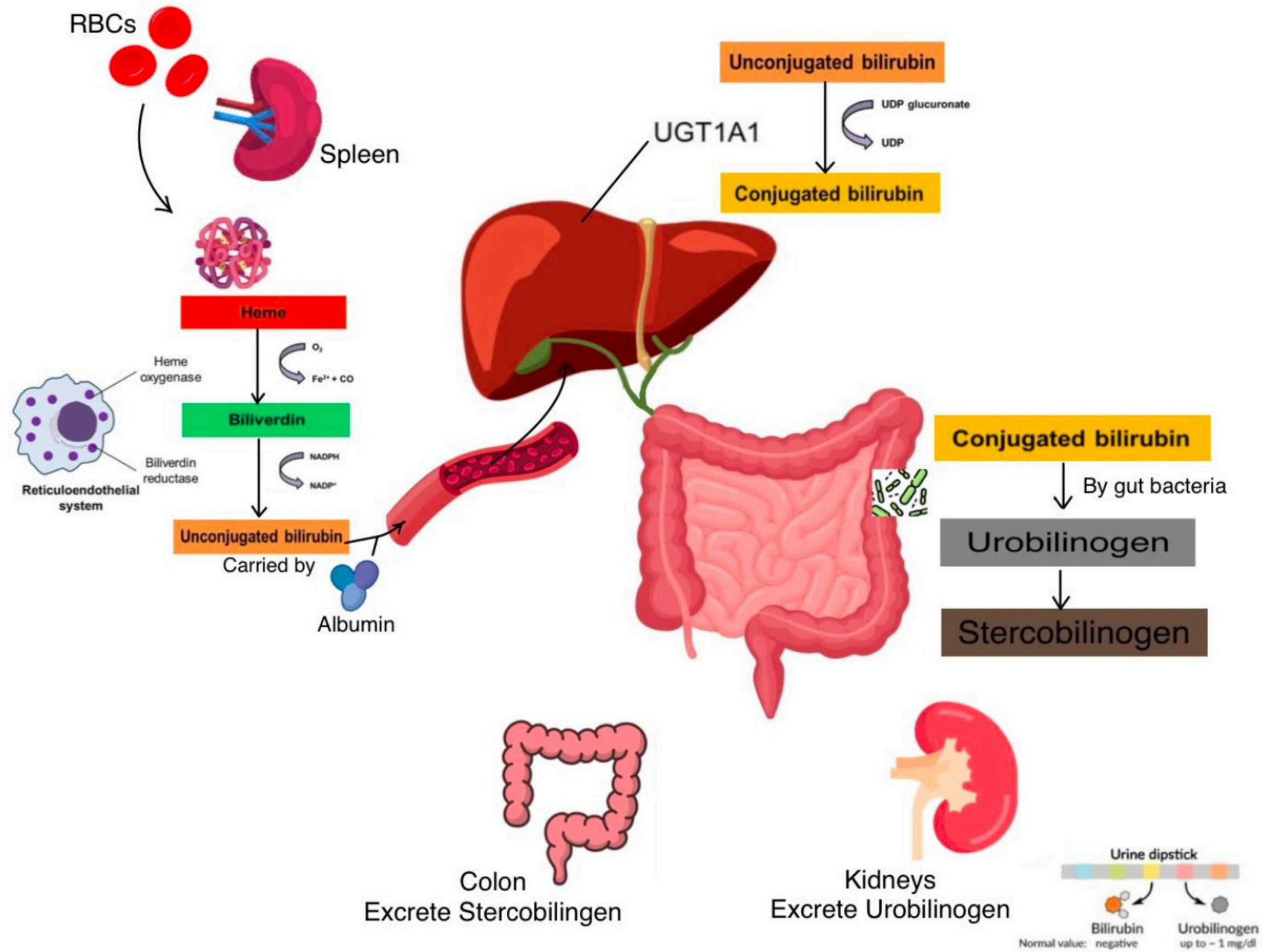
Normal range of Bilirubin is 0.3–1.2 mg/dl.



Introduction



Bilirubin metabolism



Liver Enzymes

Overview of Liver Enzymes (ALT , AST , ALP , GGT)

ALT (Alanine Aminotransferase):
Found in liver cells and released into the blood when liver cells are damaged, commonly elevated in hepatic jaundice.

AST (Aspartate Aminotransferase):
Also released during liver cell damage, less specific to the liver but elevated in hepatic jaundice.



ALP (Alkaline Phosphatase):
Produced in the liver and bile ducts, elevated in post-hepatic jaundice due to bile duct obstruction.

GGT (Gamma-Glutamyl Transferase):
Found in liver and bile ducts, elevated in post-hepatic jaundice and often used to confirm high ALP levels in Bile duct obstruction and alcoholic liver disease.

Classification of jaundice

Jaundice can be classified into three different categories including: pre-hepatic, intra-hepatic, or post-hepatic.

Pre-hepatic and intra-hepatic causes are known as medical jaundice, while post-hepatic (or obstructive jaundice) is considered surgical jaundice.

Classification of jaundice

Prehepatic jaundice (hemolytic jaundice) occurs when there is excessive breakdown of red blood cells. This leads to a high production of unconjugated bilirubin in blood which the liver cannot process fast enough (Un- conjugated Hyperbilirubinaemia).

- Causes include hemolytic anemia, sickle cell disease, malaria, G6PD deficiency.

Classification of jaundice

Hepatic jaundice (hepatocellular) occurs due to liver damage or dysfunction, affecting the liver's ability to process bilirubin. Both unconjugated and conjugated bilirubin can build up in the blood. Liver enzyme levels are often elevated in hepatic jaundice.

- Causes include Gilbert's syndrome, viral hepatitis, cirrhosis, and liver cancer.

Classification of jaundice

Post-hepatic jaundice occurs due to partial or complete obstruction in the bile ducts, such as from gallstones or tumors. This blockage prevents bile and conjugated bilirubin from being excreted into the intestines. As a result, conjugated bilirubin builds up in the bloodstream, leading to elevated levels (Conjugated Hyperbilirubinemia).

- Causes include choledocholithiasis, Acute cholangitis, chronic pancreatitis, cholangiocarcinoma .

Classification of jaundice

Process	PREHEPATIC OR HEMOLYTIC JAUNDICE e.g., Hemolytic anemia	INTRAHEPATIC JAUNDICE e.g., Hepatitis	POSTHEPATIC OR OBSTRUCTIVE JAUNDICE e.g., Gallstones
Hemolysis of erythrocytes	Excessive	Normal	Normal
↓ Hemoglobin ↓ Heme ↓ Iron ↓ Unconjugated bilirubin in blood	↓	↓	↓
↓ Liver cells	Normal liver cannot process excessive amounts of bilirubin	Hepatocellular damage prevents conjugation and excretion	Normal
↓ Conjugated bilirubin		Increased in blood	Increased in blood
↓ Bile ↓ Intestine ↓ Feces		Inflammation obstructs flow to hepatic duct	Obstructed flow to intestine causes backup to liver and blood
	Normal or darker color	Variable	Light color

6.7 Urine and stool analysis in jaundice

	Urine			Stools
	Colour	Bilirubin	Urobilinogen	Colour
Unconjugated	Normal	—	++++	Normal
Hepatocellular	Dark	++	++	Normal
Obstructive	Dark	++++	—	Pale

Distinguishing surgical jaundice from medical jaundice is just the first step in the diagnosis and treatment .

It is extremely important to obtain a thorough history and physical to help distinguish the underlying pathophysiology of the jaundice.

- Clinical approach to jaundice:

- History
- physical examination
- Investigations

HISTORY TAKING

By Demah ALzoubi

History

- Patient's profile
- Chief complaint + duration
- History of presenting complaint
- Past history (medical , drug , surgical , family, social)
- Systemic review

Patient's profile

- Name
- Gender
- Age
- Neonate : neonatal jaundice (physiological, pathological)
- Children : infections (viral hepatitis), genetic disorders (Gilbert's syndrome , congenital (biliary atresia)
- Adults : liver diseases (hepatitis, cirrhosis) , gallstones, alcohol-related liver damage.
- Elderly: chronic liver disease , cancer (pancreatic, hepatic) , obstruction due to tumor .
- Pregnant women: Intrahepatic cholestasis of pregnancy, HELLP syndrome .

Patient's profile

- Marital status
- Occupation:
 - Health care workers: exposure to infected blood or bodily fluids (hepatitis A, B or C).
 - Travel related jobs : hepatitis A , malaria.
- Address .

Chief Complaint

Main problem:

- Jaundice (yellow discoloration of eyes , mucosal membrane, skin)
- Abdominal pain
- Itching
- Dark urine , pale stools

Duration : for how long ?

- If Acute , the following diseases are commonly suspected:
 - Acute viral hepatitis, hemolytic Anemia , Gallstones.
- If Chronic :
 - Chronic hepatitis, liver cirrhosis , primary biliary cholangitis (PBC) , Gilbert's syndrome, chronic bile duct obstruction from strictures or tumors.

History of presenting complain :

1. Since when? Today, a week ago?

2. Did it appear sudden or gradually?

A. Days to a week-----acute hepatitis

B. 2 weeks: subacute hepatitis or biliary obstruction.

C. More than two weeks : It may be : malignant biliary obstruction, chronic hepatitis (cirrhosis) , chronic pancreatitis, stricture in the common bile duct, or toxin exposure (alcohol).

History of presenting complain :

3. Had this problem happen to you before? Recurrent short episodes over months to years + RUQ abdominal pain, implicate gallstones, chronic hepatitis, cirrhosis.

4. How did you noticed it? (friends, family, you noticed it?)

5. Is it continuous?

A. Fluctuating intensity: gallstones, ampullary carcinoma, or drug-induced hepatitis.

questions to differentiate between jaundice causes

obstructive jaundice (post-hepatic)

Pale (clay colored) stool?
Dark urine?
Itching?



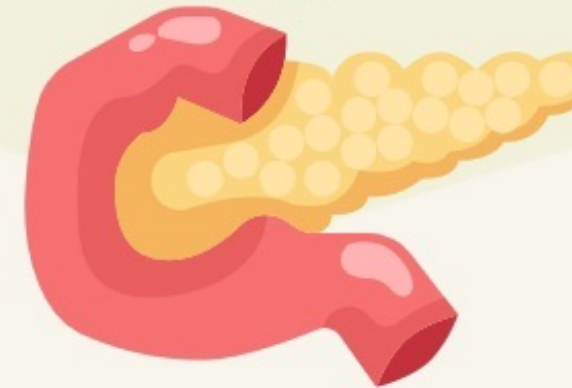
hemolytic anemia

Pallor
Fatigue
Shortness of breath
Dizziness



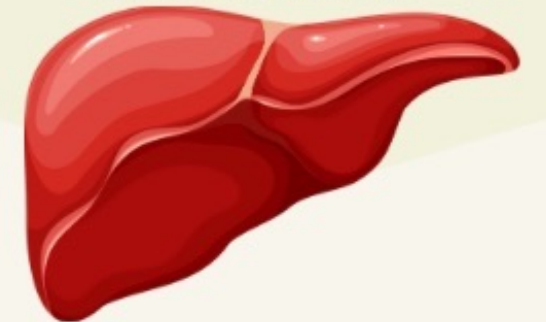
pancreatic head cancer

Anorexia (loss of appetite)?
Weight Loss? Fatigue?
Bloating?
Steatorrhea?
Diarrhea?



Liver disease complications

Change in bowel habits?
Nausea, vomiting, and
distention (ascites)?
Melena, hematemesis,
hematochezia?



questions to differentiate between jaundice causes

cholangitis

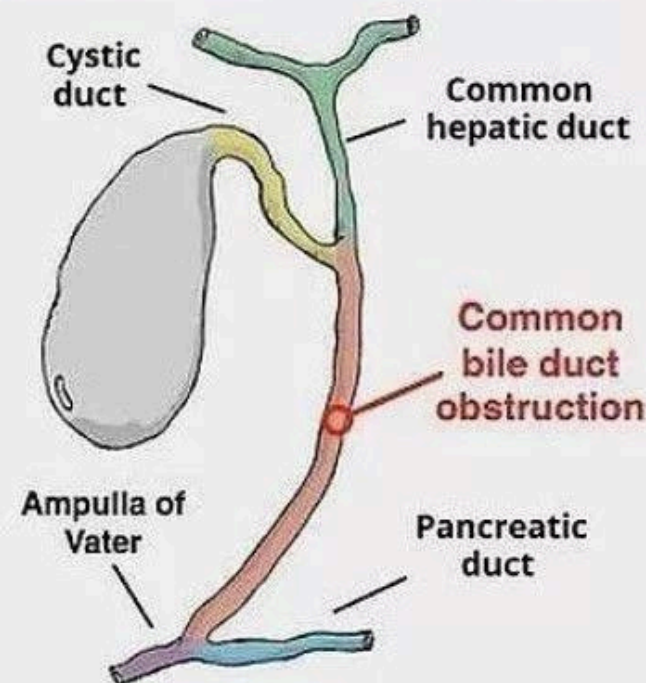
charcot's triad



Acute (Ascending) Cholangitis

A clinical syndrome characterized by **fever**, **jaundice**, and **abdominal pain** that develops as a result of **stasis** and **infection** in the **biliary tract**.

Bacterial infection in a patient with biliary obstruction



▲ Charcot's Triad
Fever
Abdominal pain
Jaundice

◆ Reynolds pentad
Fever
Abdominal pain
Jaundice
+
Confusion
Hypotension

Management

- Broad-spectrum antibiotics (e.g. Ampicillin-sulbactam)
- Biliary drainage (e.g. ERCP)



Do you have pain ?

painless.  **Hepatocellular jaundice**

Dull ache or "heavy sensation" in RUQ  **acute hepatitis**

Painful obstructive jaundice  **presence of gall stones (biliary obstruction)**

Painless obstructive jaundice  **indicates malignant biliary obstruction**

Fever + pain + jaundice = charcot's triad  **indicates ascending cholangitis**



past medical and surgical history:

Gallbladder stones

Pancreatitis

Biliary surgeries (stricture)

Autoimmune diseases (ibd)

hemoglobinopathy

Missed menses (pregnancy):HELLP syndrome

Drug History:

(paracetamol, sulpha drugs, OCPs)

Social History:

Alcohol drinking?

Travel? (hep A, or fasciolosis (parasitic) (poor areas))

Immunization? (hep B)

Blood transfusion?

Skin tattooing? IV drug use? Addiction?

Sexual and contact history? (hep B or C)

Family History:

recurrent mild jaundice in family, hereditary liver disorders,
Autoimmune, metabolic syndromes

PHYSICAL EXAM

By saleh ALhiyari

physical examination of the abdomen

preparation

The environment

1.Comfortable.

2.Private.

3.Good light, **moreover daylight helps you identify jaundice much easier**

physical examination of the abdomen

preparation

The examination couch or bed

hard couch with a backrest that can be raised by **15–20°**.

To maintain patient dignity cover the lower abdomen with a sheet or blanket while the abdomen is being palpated.

relax the patient.

physical examination of the abdomen

Examination

In general

Patient may have abdominal pain, fever or rigors and jaundice. This is called charcot triad .

Patient may have pruritus occurs in cases of obstructive jaundice so he may have **itch marks** .

Patient may be confused, hypotensive and tachycardia this may occur in raynold pentad .

**raynold also
includes charcot**

confusion means :

- Decline in cognitive ability
- Difficulty focusing
- Feeling disoriented

physical examination of the abdomen

Examination

In general

anaemic pallor combined with jaundice may produce a pale lemon complexion occurs in haemolytic disorders in **pre-hepatic jaundice**.

may have body tattoo .

bruises .

may have spider nevi .

physical examination of the abdomen

Examination

In general

Jaundice is initially visible against the white background of the sclerae, and then becomes more obvious as the skin turns yellow with elevation of the serum levels.

Edema (swelling with fluid in your ankles, feet, hands or face).

physical examination of the abdomen

Examination

Inspection

From the **foot of the bed** :

jaundice is visible.

may see caput medusa

distension of abdomen

may see dilated veins

right hypochondral localised swelling due to
liver enlargement

in general look for scars ,sinuses, fistulas, trauma,
hernias

physical examination of the abdomen

Examination

Inspection

From the **right side of the patient:**

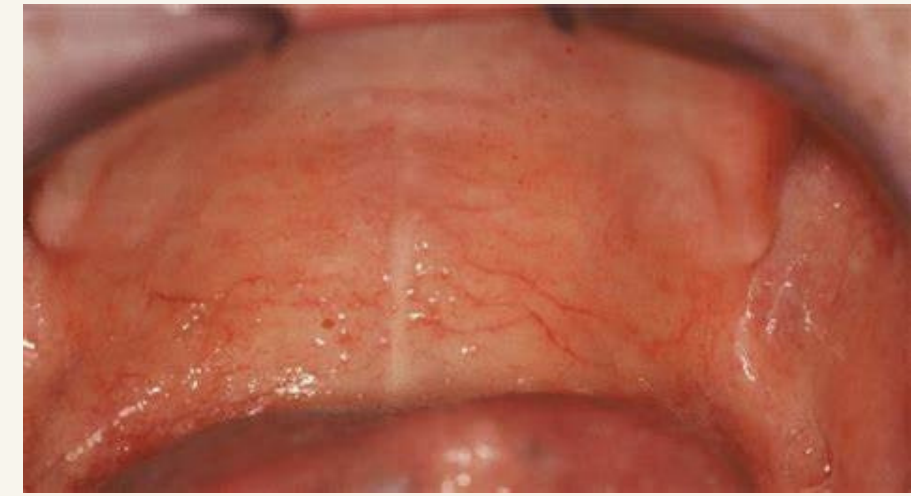
For jaundice look for **yellow sclera, yellow face** and **mucous membranes of mouth**.

fetor hepaticus, or may smell alcohol odour

look for palmar erythema .

look for leuconychia .

look for clubbing found in liver cirrhosis .



physical examination of the abdomen

Examination

Inspection

From the **right side of the patient**:

Dupuytren's contracture of palmar fascia .

Asterixis, a coarse flapping tremor

Look for signs of drug abuse.



physical examination of the abdomen

Examination

Palpation

Examination sequence for **hepatomegaly**:

If you feel a liver edge, describe:

size.

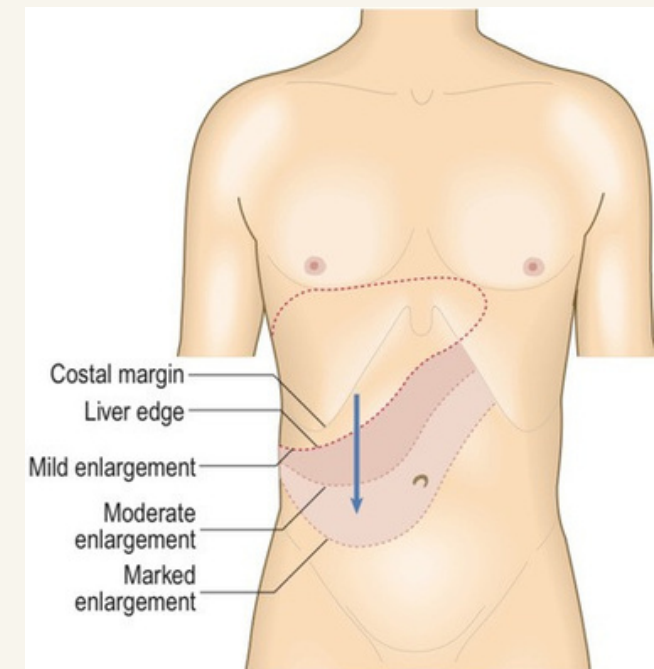
surface.

edge.

consistency.

tenderness.

pulsatility.



physical examination of the abdomen

Examination

Palpation

Examination sequence for **obstructive jaundice** :

Palpable **painless** distension of the gallbladder (Courvoisier's sign) is

At the intersection of the right lateral vertical plane (mid clavicular line) and the costal margin, i.e. tip of the ninth costal cartilage.

physical examination of the abdomen

Examination

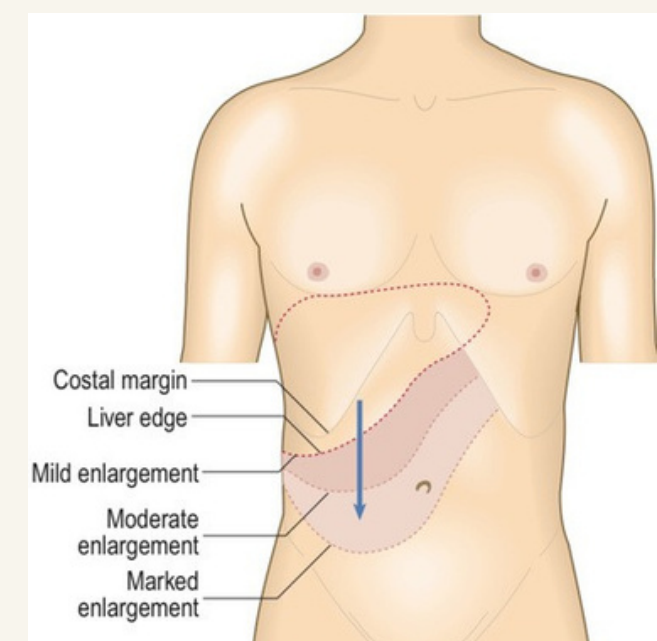
Percussion

Examination sequence for **hepatomegaly**:

Ask the patient to hold their breath in full expiration.

Percuss downwards from the right fifth intercostal space in the mid-clavicular line.

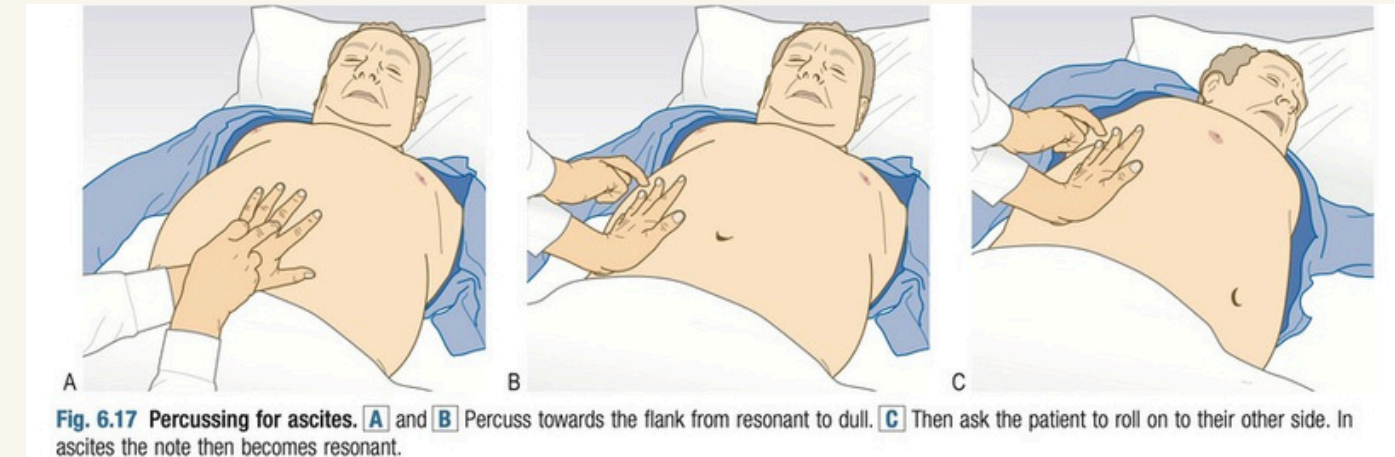
Measure the distance below the costal margin in the mid-clavicular line



physical examination of the abdomen

Percussion

Examination for ascites : Shifting dullness



Examination for ascites : Eliciting a fluid thrill.

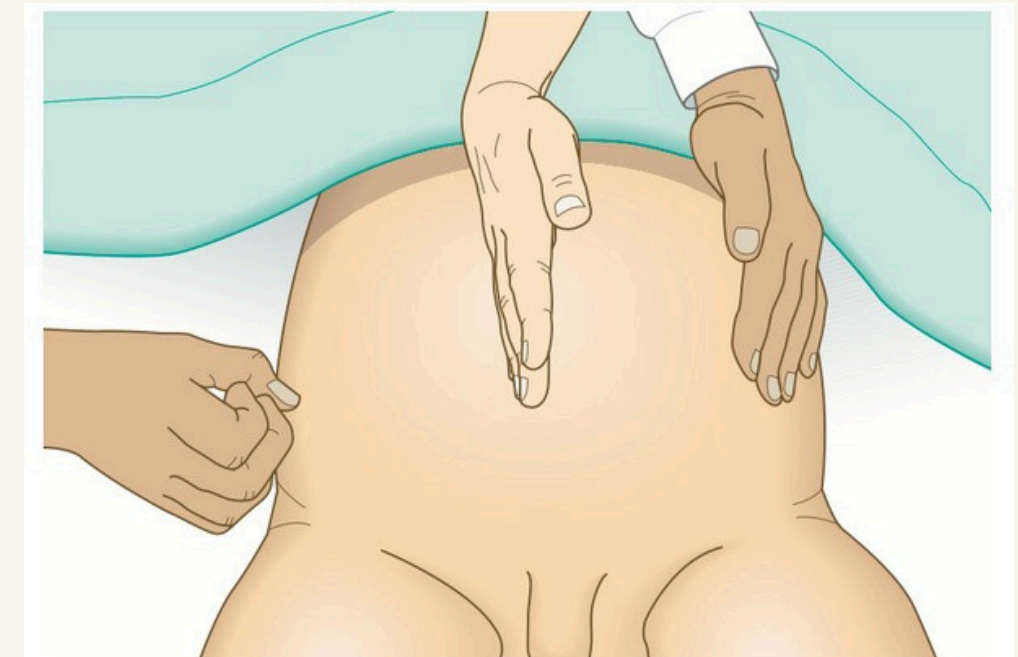


Fig. 6.18 Eliciting a fluid thrill.

physical examination of the abdomen

Auscultation

A bruit over the liver may be heard in acute alcoholic hepatitis, hepatocellular cancer



physical examination of the abdomen

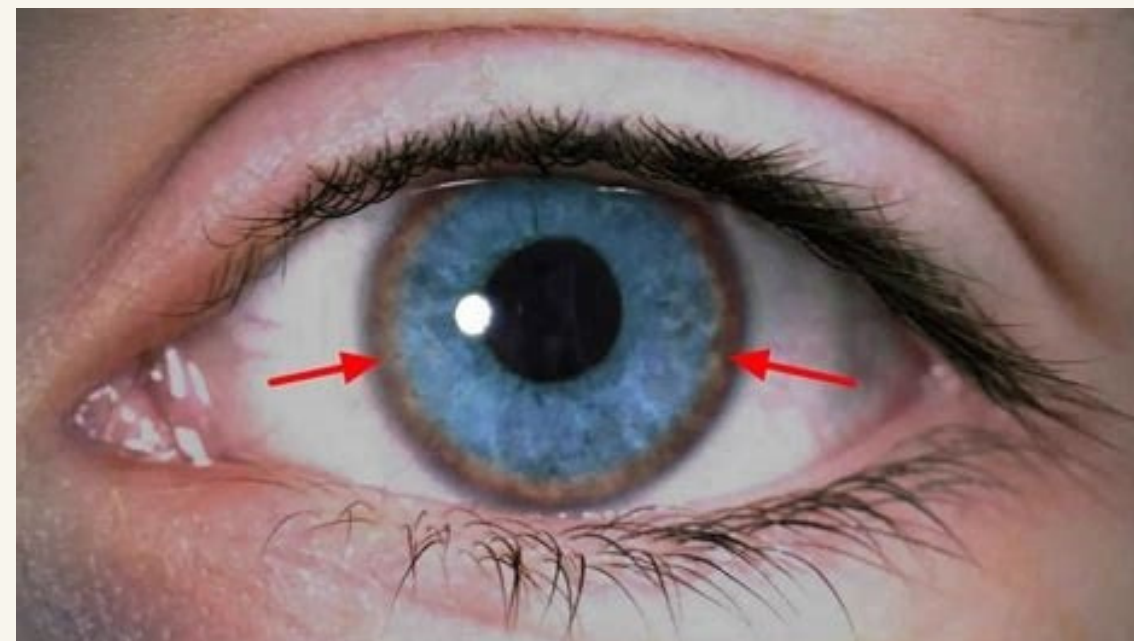
Examination

Special cases

Hepatic encephalopathy may occur in liver diseases so must conduct a neurological exam.

testicular atrophy and gynecomastia may occur in males with liver diseases due to accumulation of estrogen.

Wilson disease.



6.11 Grading of hepatic encephalopathy (West Haven)

Stage	State of consciousness
0	No change in personality or behaviour No asterixis (flapping tremor)
1	Impaired concentration and attention span Sleep disturbance, slurred speech Euphoria or depression Asterixis present
2	Lethargy, drowsiness, apathy or aggression Disorientation, inappropriate behaviour, slurred speech
3	Confusion and disorientation, bizarre behaviour Drowsiness or stupor Asterixis usually absent
4	Comatose with no response to voice commands Minimal or absent response to painful stimuli

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MANAGEMENT & INVESTIGATION

By Aseal ALghwadreh

Differential Diagnosis of Jaundice

Condition		Comments
<i>Prehepatic (hemolytic)</i>	Hemolytic anemia	Pallor, fatigue, shortness of breath
	Gilbert's syndrome	Benign condition triggered by overexertion, dehydration, menstruation, concurrent illness, surgery; decreased activity of the enzyme UDP-glucuronyl transferase
<i>Hepatic (hepatocellular)</i>	Ischemic liver injury	May result from transient hypotension and hypoxia or trauma or in transplanted donor liver; marked rise in ALT and AST
	Viral	Viral infection of hepatocytes; anorexia, nausea, vomiting; aminotransferases >25× upper limit of normal; may progress to acute liver failure, become a chronic infection, or completely resolve
	Toxic ingestion	Acetaminophen, alcohol (AST:ALT ratio > 2), iron overload; can cause acute liver failure
	Nonalcoholic fatty liver disease	Often asymptomatic; hepatic steatosis, metabolic syndrome, AST:ALT ratio < 1
	Primary biliary cirrhosis	Autoimmune process, skin xanthomas, associated with rheumatoid arthritis or Sjogren's syndrome; antimitochondrial antibody
	Primary sclerosing cholangitis	Associated with ulcerative colitis, fatigue, pruritus, p-ANCA
	Hepatolenticular degeneration (Wilson's disease)	Autosomal recessive, retention of copper, Kayser-Fleischer ring (brown ring on the edge of the iris), cirrhosis, and neurologic manifestations including parkinsonism
	Cirrhosis	Hyperestrinism (gynecomastia, testicular atrophy, decreased body hair, spider angiomas, palmar erythema), portal hypertension (esophageal varices, splenomegaly, ascites, caput medusae, anorectal varices), hepatic synthetic dysfunction (ecchymoses, edema, hydrothorax)
<i>Posthepatic</i>	Choledocholithiasis	Gallstone (from the gallbladder) enters the common bile duct (CBD) causing obstruction, RUQ pain, nausea
	Acute cholangitis	Ascending infection of the CBD often caused by gallstone obstruction; fever, jaundice, RUQ pain, ± hypotension, ± altered mental status
	Chronic pancreatitis	May cause biliary stricture, recurrent epigastric pain, malabsorption
	Mirizzi syndrome	RUQ pain due to external compression of the common hepatic duct by large gallstone in gallbladder or cystic duct
	Pancreatic carcinoma	Painless jaundice is more common in carcinomas arising from the head of the pancreas; new-onset diabetes may precede diagnosis of cancer; poor prognosis
	Ampullary carcinoma	Malignancy arising from the ampulla of Vater; most common presentation is painless obstructive jaundice
	Cholangiocarcinoma	Associated with sclerosing cholangitis, chronic parasitic infections, malignancy of the bile ducts usually present at advanced stage, highly lethal

Investigation

Prehepatic jaundice

- This is caused either by haemolysis or by congenital hyperbilirubinaemia, and is characterised by an **isolated raised bilirubin level.**
↑Reticulocytes, ↓Haptoglobin

In haemolysis, destruction of red blood cells or their marrow precursors causes increased bilirubin production.

The most common form of non-haemolytic hyperbilirubinaemia is Gilbert syndrome, an inherited disorder of bilirubin metabolism.



25.36 Investigation results indicating active haemolysis

Hallmarks of haemolysis

- ↓ Haemoglobin
- ↑ Unconjugated bilirubin
- ↑ Lactate dehydrogenase
- ↑ Reticulocytes
- ↑ Urinary urobilinogen

Additional features of intravascular haemolysis

- ↓ Haptoglobin
- ↑ Methaemalbumin
- Positive urinary haemosiderin
- Haemoglobinuria

Lab test

Both hepatic and post-hepatic causes of jaundice will have some degree of elevation in total bilirubin, AST, ALT, GGT, ALP.

Disproportionate rise in the transaminases (AST, ALT) is indicative of hepatocellular damage.

A marked rise in AP, out of proportion to the AST and ALT, is therefore more indicative of posthepatic (biliary obstruction) pathology. Since AP levels increase with many other diseases (such as bone pathology), a concomitant and proportionate rise in GGT is helpful, as it is more specific for liver disease.

Imaging

for Painful Jaundice: The first-line diagnostic test is RUQ ultrasound (US).

RUQ US is excellent in terms of visualizing gallstones within the gallbladder and in demonstrating dilation of the CBD due to obstruction.

for Painless Jaundice:

since the suspicion for malignancy is high, the next study of choice is a “triple-phase” abdominal CT scan as ultrasound cannot rule out pancreatic lesions.

Triple-phase CT

captures images during three phases of contrast: (1) arterial phase, (2) early venous phase, and (3) late venous phase.

Lab test

i

24.2 'Hepatic' and 'cholestatic' liver function tests

Pattern	AST/ALT	GGT	ALP
Cholestasis	↑	↑↑	↑↑↑
Hepatitis	↑↑↑	↑	↑
Alcohol/enzyme-inducing drugs	N/↑	↑↑	N

N = normal; ↑ mild elevation (< twice normal); ↑↑ moderate elevation (2–5 times normal); ↑↑↑ marked elevation (> 5 times normal).

(ALT = alanine aminotransferase; ALP = alkaline phosphatase; AST = aspartate aminotransferase; GGT = gamma-glutamyltransferase)

CA 19-9 (in about 75%) and CEA levels (in about 45%) can be elevated in pancreatic cancer.

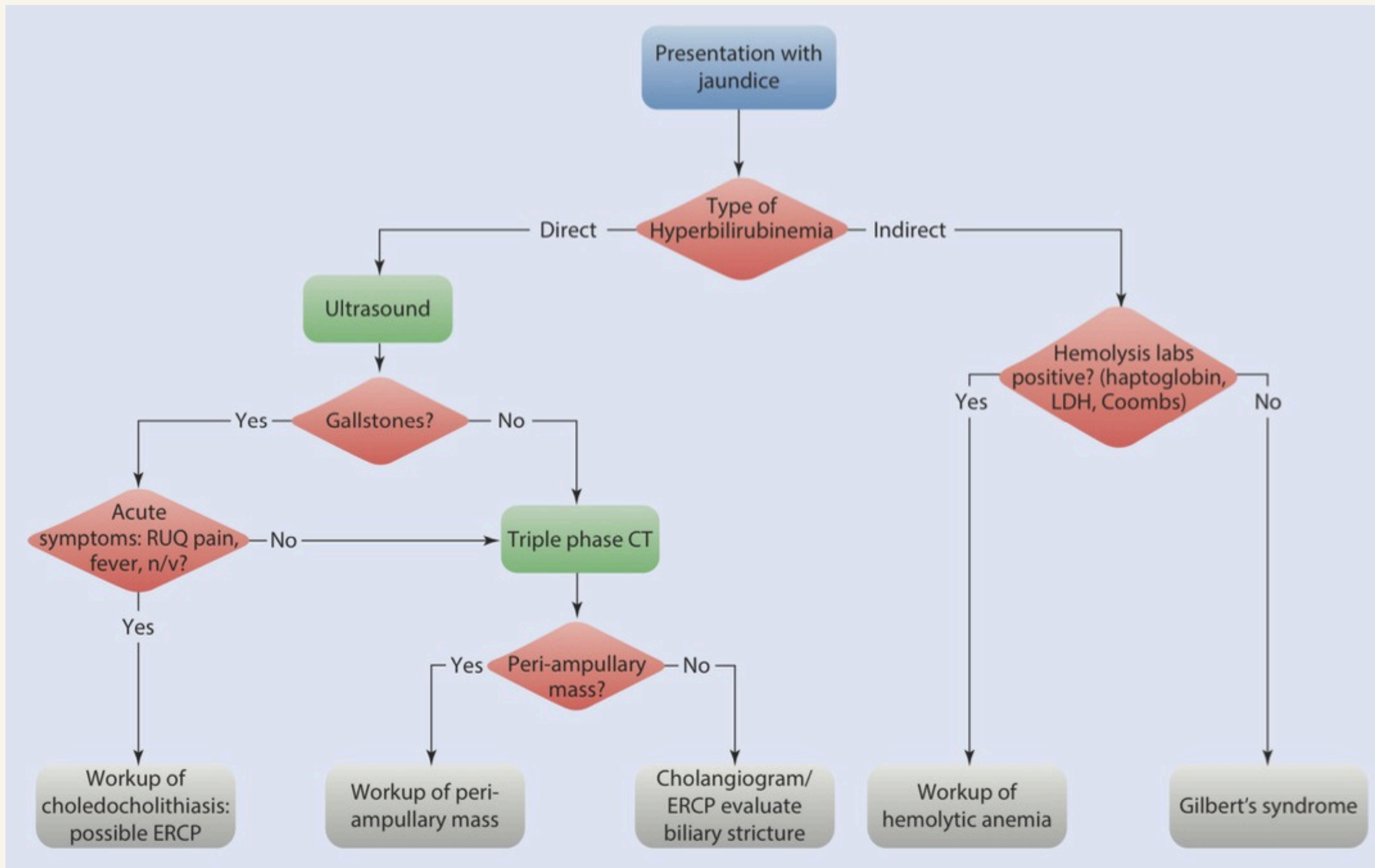
Imaging

for Painless Jaundice:

EUS helps to better delineate the mass, detect smaller masses, detect vascular invasion, and identify enlarged lymph nodes. It can also identify other causes of biliary obstruction, such as a biliary stricture.

If CT/EUS does show a pancreatic mass suspicious for malignancy, biopsy is not necessary if patient is a surgical candidate. MRCP and ERCP are not routinely needed. They are used to further delineate the biliary tree especially if no mass is seen.

Management (stones)



Management (stones)

Once a Patient Develops Fever and Leukocytosis

First, the patient should receive targeted aggressive intravenous fluid resuscitation with normal saline or lactated Ringer's. Indwelling foley catheter should be considered in order to guide resuscitation.

In the case of suspected cholangitis, antibiotics should cover enteric organisms (Gram-negative rods, enterococcus, and anaerobes).

Management (stones)

Next step

The next step in treatment is to drain the infected bile, termed biliary decompression. This is best accomplished by ERCP.

If ERCP is unsuccessful, percutaneous transhepatic drainage (PTC) is the next choice. In this procedure, the bile is drained via a catheter inserted directly into the liver.

If both of these options fail, the bile may need to be decompressed operatively by placing a drain (called a T-tube) directly into the common bile duct.

During ERCP, a scope is inserted through the mouth to the ampulla of Vater. The ampulla is cannulated, the stone can be extracted, the sphincter of Oddi is cut (sphincterotomy) to allow drainage of the bile into the duodenum, and a stent is often placed.

Management (stones)

**Following Successful ERCP
they should undergo laparoscopic
cholecystectomy (LC) to prevent
future episodes.**



the end

PDF done by Saleh ALhiyari



THANK YOU