Jaundice

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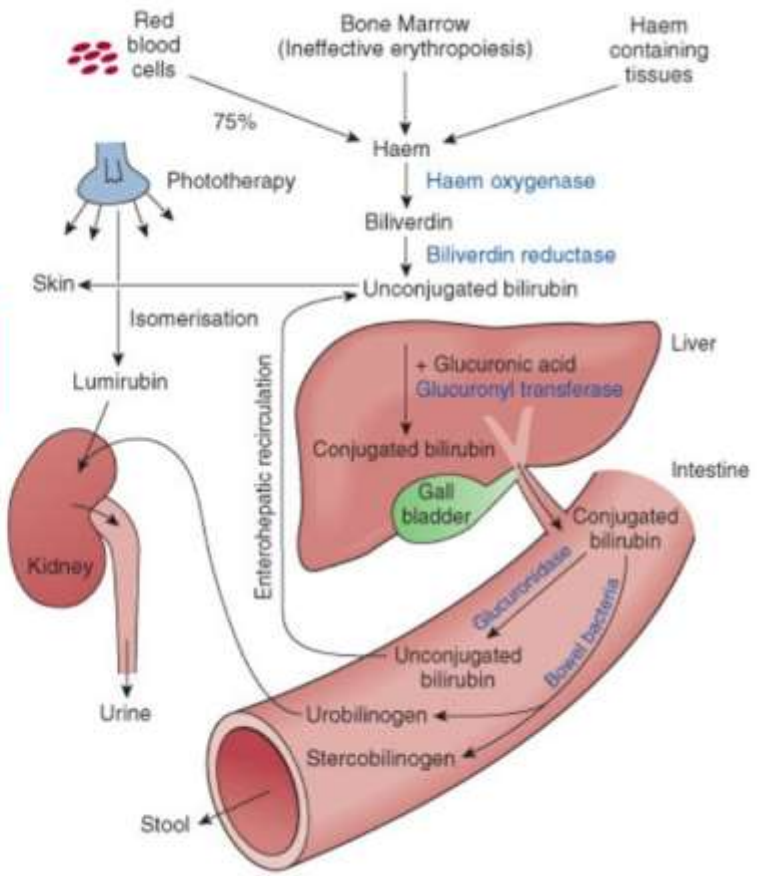
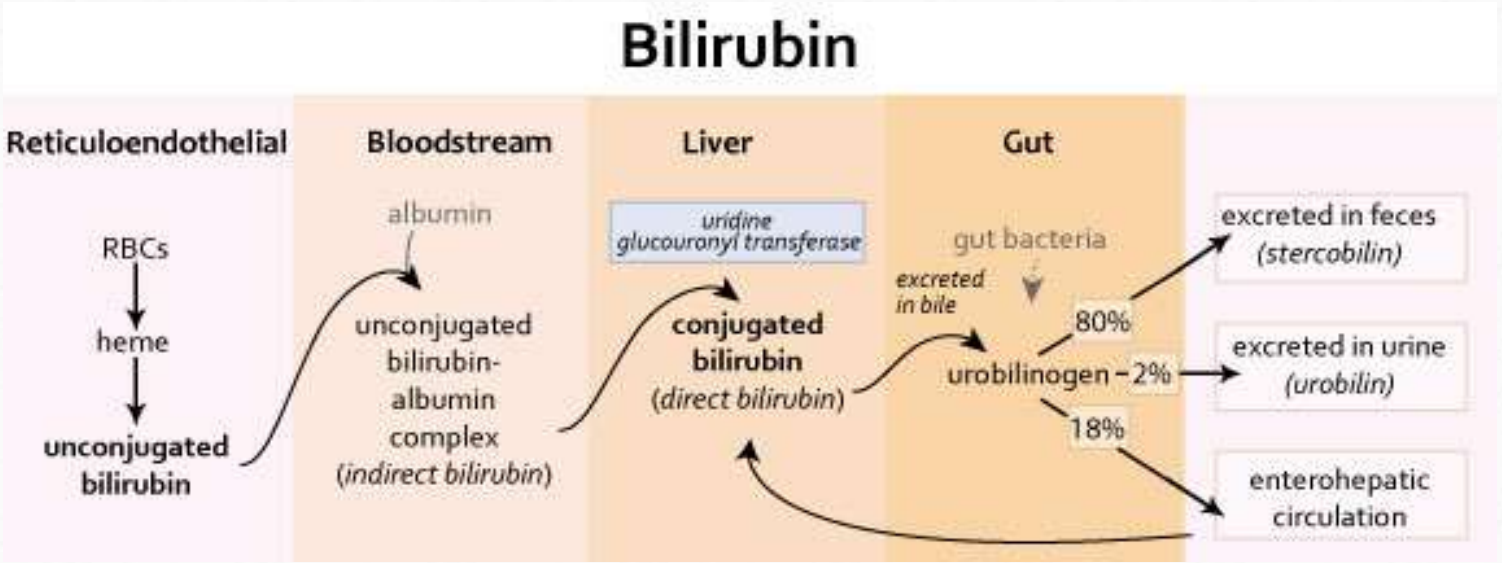
Introduction

Bilirubin

- Bilirubin is yellow product occur in normal Circumstance during heme catabolism
- The major of bilirubin produced from break down RBCs (after120 das)
- The minor of bilirubin produced from heme containg protein from other tissue site
- Normal range in adult 1.2 milligrams per deciliter (mg/dL)

BILIRUBIN METABOLISM

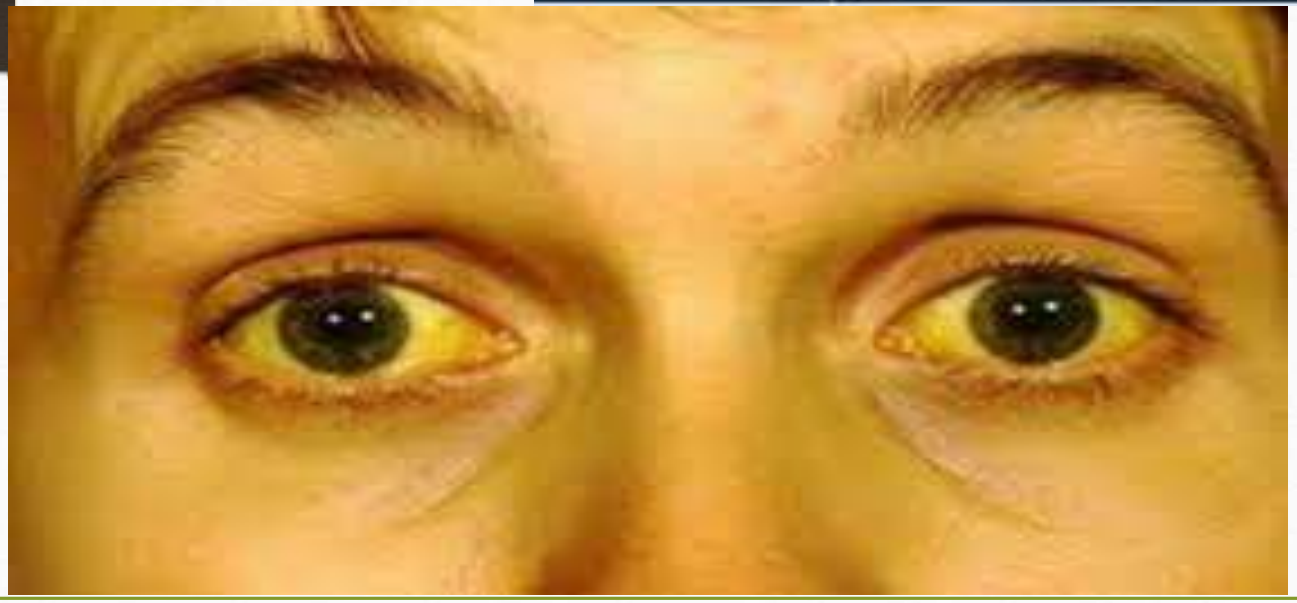



Bilirubin
Reticuloendothelial
Bloodstream
Liver
Gut
RBCs
heme
unconjugated bilirubin
albumin
unconjugated bilirubin-albumin complex (indirect bilirubin)
uridine glucouranyl transferase
conjugated bilirubin (direct bilirubin)
gut bacteria
excreted in bile
urobilinogen
80%
2%
18%
excreted in feces (stercobilin)
excreted in urine (urobilin)
enterohepatic circulation

JAUNDICE

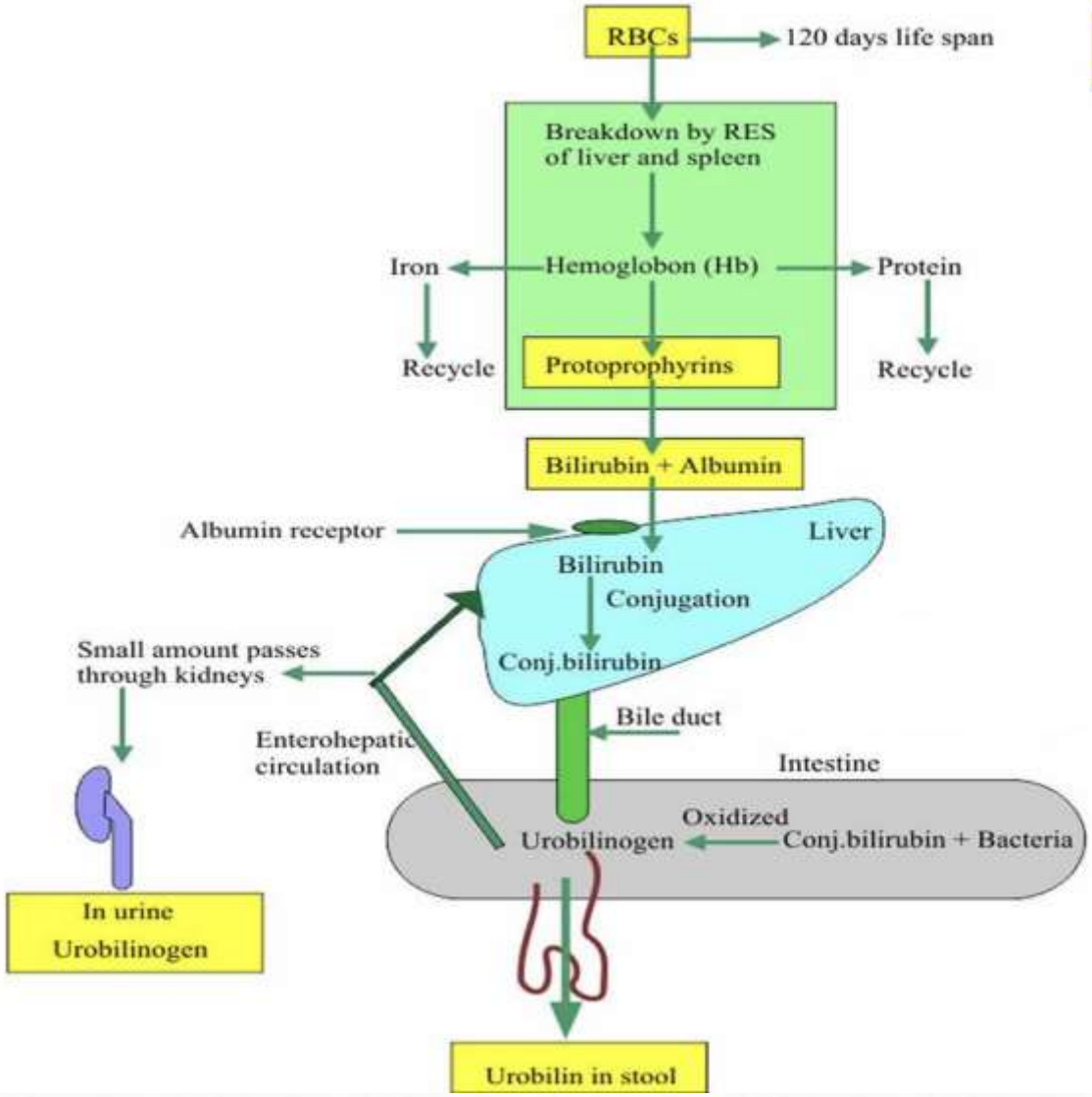
- It refers to yellowish discolouration of skin or scelera due to excess deposition of bilirubin in tissue
- Classify to two categories :
- _ Plasma elevation of predominantly unconjugated (indirect) bilirubin
- _ Plasma elevation of both unconjugated and conjugated (direct) bilirubin
- **_normal level** is: Direct (also called conjugated) **bilirubin**: less than 0.3 mg/dL (less than 5.1 μmol/L) Total **bilirubin**: 0.1 to 1.2 mg/dL (1.71 to 20.5 μmol/L)


JAUNDICE IN ADULTS
#clinic100hrs - Unlimited Chat With a Doctor for the next 100 Hours



Pathophysiology

Bilirubin metabolism



Conjugated hyperbilirubinemia is defined by:

- A serum conjugated bilirubin concentration greater than 1mg/dL if the total bilirubin is <5 mg/dL
- Or more than 20% of the total bilirubin if the total bilirubin is >2 mg/dL
- Or if the direct bilirubin is >2mg/dL

There are Three types of Jaundice

- Prehepatic Jaundice
- Hepatocellular Jaundice
- Obstructive (cholestatic) Jaundice

Prehepatic Jaundice

- It is caused either by hemolysis or by congenital hyperbilirubinemia
- In Hemolysis, destruction of RBCs or their marrow precursors causes increase bilirubin production
- It is usually mild, since a healthy liver can excrete a bilirubin load six times greater than normal.
- Other prehepatic jaundice occurs due to congenital issues, such as Gilbert's syndrome or Dubin Johnson syndrome

Prehepatic Jaundice

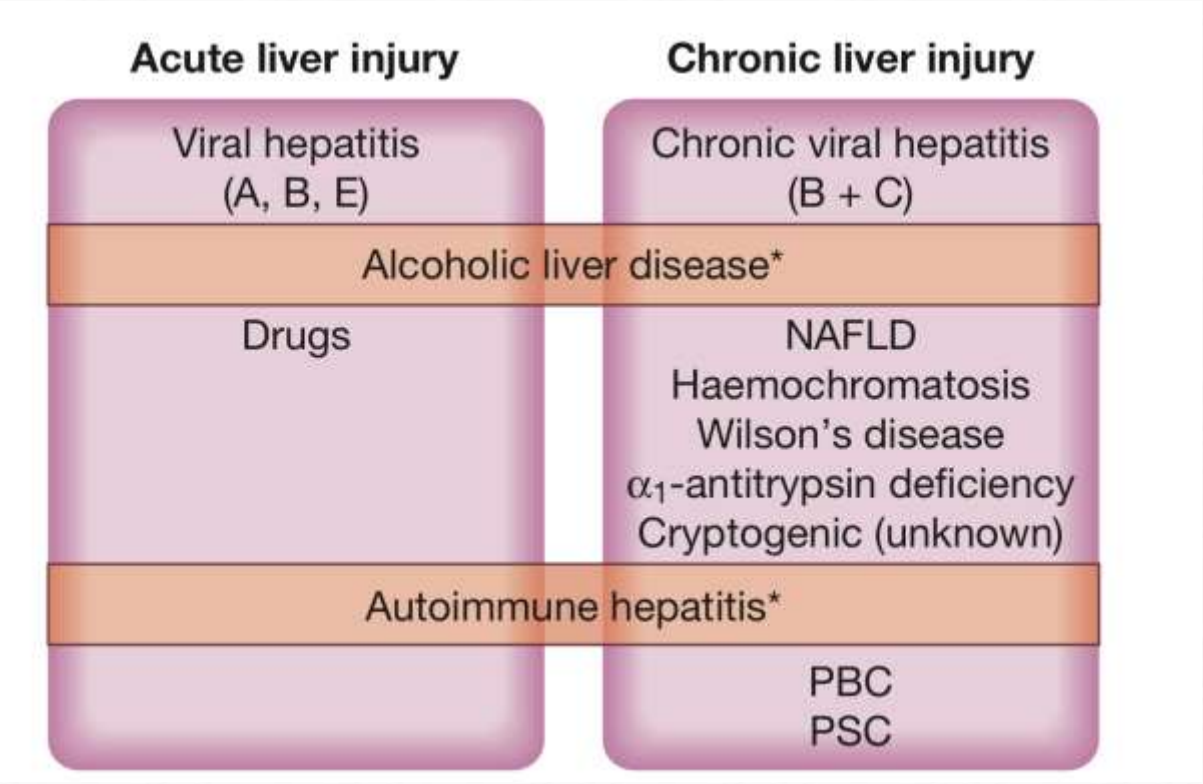
23.17 Congenital non-haemolytic hyperbilirubinaemia

Syndrome	Inheritance	Abnormality	Clinical features	Treatment
Unconjugated hyperbilirubinaemia				
Gilbert's	Autosomal dominant	↓ Glucuronyl transferase ↓ Bilirubin uptake	Mild jaundice, especially with fasting	No treatment necessary
Crigler–Najjar				
Type I	Autosomal recessive	Absent glucuronyl transferase	Rapid death in neonate (kernicterus)	
Type II	Autosomal dominant	↓↓ Glucuronyl transferase	Presents in neonate	Phenobarbital, ultraviolet light or liver transplant
Conjugated hyperbilirubinaemia				
Dubin–Johnson	Autosomal recessive	↓ Canalicular excretion of organic anions, including bilirubin	Mild	No treatment necessary
Rotor's	Autosomal recessive	↓ Bilirubin uptake ↓ Intrahepatic binding	Mild	No treatment necessary

Hepatocellular Jaundice

- Results from an inability of the liver to transport bilirubin into the bile
- Transport of bilirubin maybe impaired at any point between uptake of unconjugated bilirubin into cells and transport of bilirubin into canaliculi
- Both Conjugated and unconjugated bilirubin in blood increase.
- Hepatocellular Jaundice can be due to acute or chronic injury

Causes of Hepatoceullar Jaundice



Obstructive (Cholestatic) Jaundice

- Maybe caused by:

1. Failure of hepatocytes to initiate bile flow
2. Obstruction of bile ducts or portal tracts
3. Obstruction of bile flow in the extrahepatic bile ducts between the ports hepatis and the papilla of valer

- Cholestatic jaundice tends to become more severe progressively because conjugated bilirubin is unable to enter the bile canaliculi and back to the blood

Obstructive (Cholestatic) Jaundice

23.15 Causes of cholestatic jaundice

Intrahepatic

- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Alcohol
- Drugs
- Hepatic infiltrations (lymphoma, granuloma, amyloid, metastases)
- Cystic fibrosis
- Severe bacterial infections
- Pregnancy (p. 977)
- Inherited cholestatic liver disease, e.g. benign recurrent intrahepatic cholestasis
- Chronic right heart failure

Extrahepatic

- Carcinoma
  - Ampullary
  - Pancreatic
  - Bile duct (cholangiocarcinoma)
  - Liver metastases
- Choledocholithiasis
- Parasitic infection
- Traumatic biliary strictures
- Chronic pancreatitis

History

As any history taking we start with the patient profile:

Name: Age: Marital status:

Occupation: important healthcare worker

Living place:

Then we should ask if it happened before.

- As it's an indication of recurrent jaundice:
- Haemolytic.
- Wilson's disease .
- Cholestasis in pregnancy (common).
- Gall stones
- We should keep it in mind while taking history.

Then we ask if there is pain or not

- **As painful jaundice is most likely associated with**
- Gall stones
- Hepatitis
- Cancer head of pancreas
- Hepatoma
- Then we ask about Socrates
- **Where as painless jaundice jaundice we think and ask more about familial jaundice(inherited):**
- Gilbert. Grigler Najjar. Inherited hemolytic anemia. Hemochromatosis. Cholestasis of pragnancy
- So we ask about family history and the consequences

Now we will talk about clues in history that will indicates if it's prehepatic or intra or obstructive.

- SOB , fatigue , dizziness ,cold extremes
- +family history or any blood disorders it's most likely extra –hepatic.

- Fever ,headache ,pharyngitis , right upper abdominal pain , we should ask about food +if there is others with same symptoms + blood transfusion + alcohol as it's highly indicates intra-hepatic
- As hepatitis (work +contaminated food) , alcoholic hepatitis , autoimmune hepatitis +hepatotoxicity(paracetamol,statins,rifampicin
- Of course don't forget to ask about HTN plus D.M (liver cirrhosis)

Now about post-hepatic clues in history

- The most important one ,which it's medical emergency
- The triad of :
- FEVER. URQ Pain(intolerable). Jaundice. It's indication of **acute cholangitis**
- Gall stones :ask about pain in RUQ , discomfort after fatty food(no bile) , past history of gall stones or surgery or pancreatic cancer
- Nausea and vomiting
- Then complete normal history sequence

Physical examination

Vital sign

- Fever
- Tachycardia
- Hypotension
- Tachypnea
- It most occur with viral hepatitis

Hand examination

- Palmar erythema.
- Dupuytrens contracture.
- Finger clubbing & leuconychia
- Astrexis in hepatoencephalopathy.

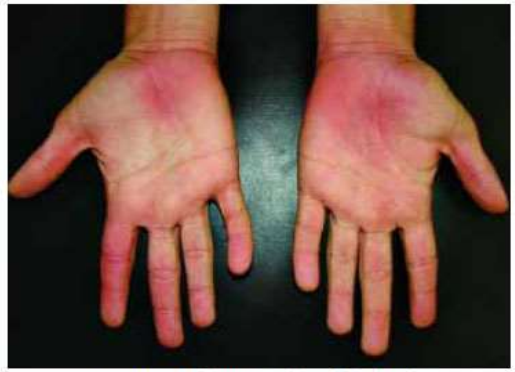
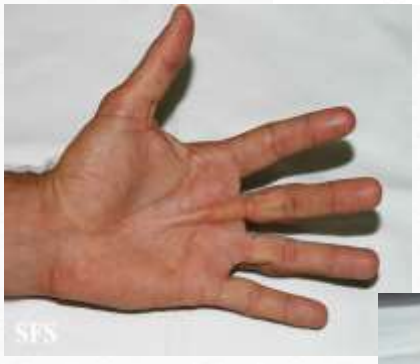
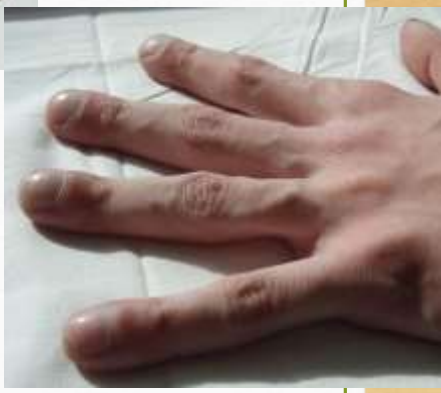


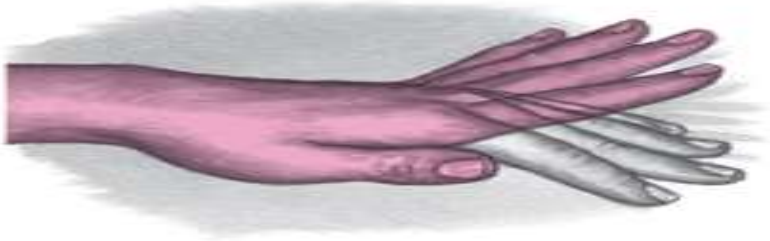
FIGURE 3: Erythema on palms of the hands



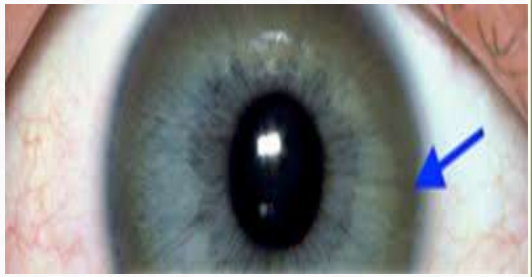


Recognizing asterixis

In asterixis, the patient's wrists and fingers are observed to "flap" because of a brief, rapid relaxation of wrist dorsiflexion.



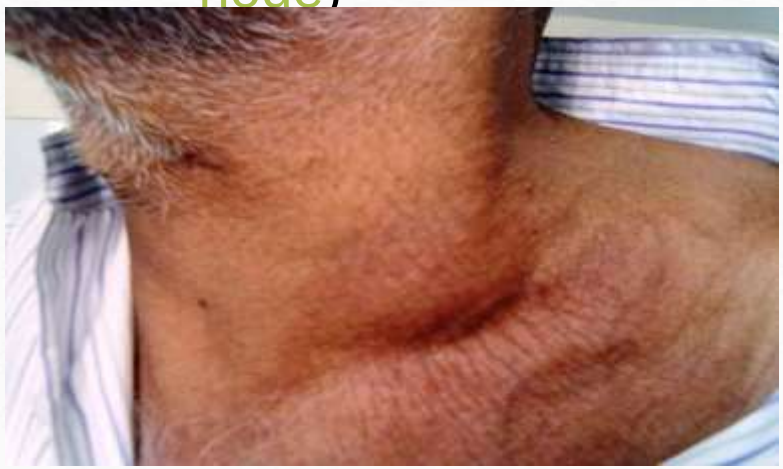
Face examination



- signs of jaundice (skin, sclera , mucos membranes).
- Pale conjunctiva in hemolysis .
- Kayser-Fleischer rings in wilson disease.
- Parotoid gland enlargment in hepatic disease especially fatty liver (e.g alcoholic)
- sweet smell on breath of Fetor hepaticus (release of dimethyl sulfide)

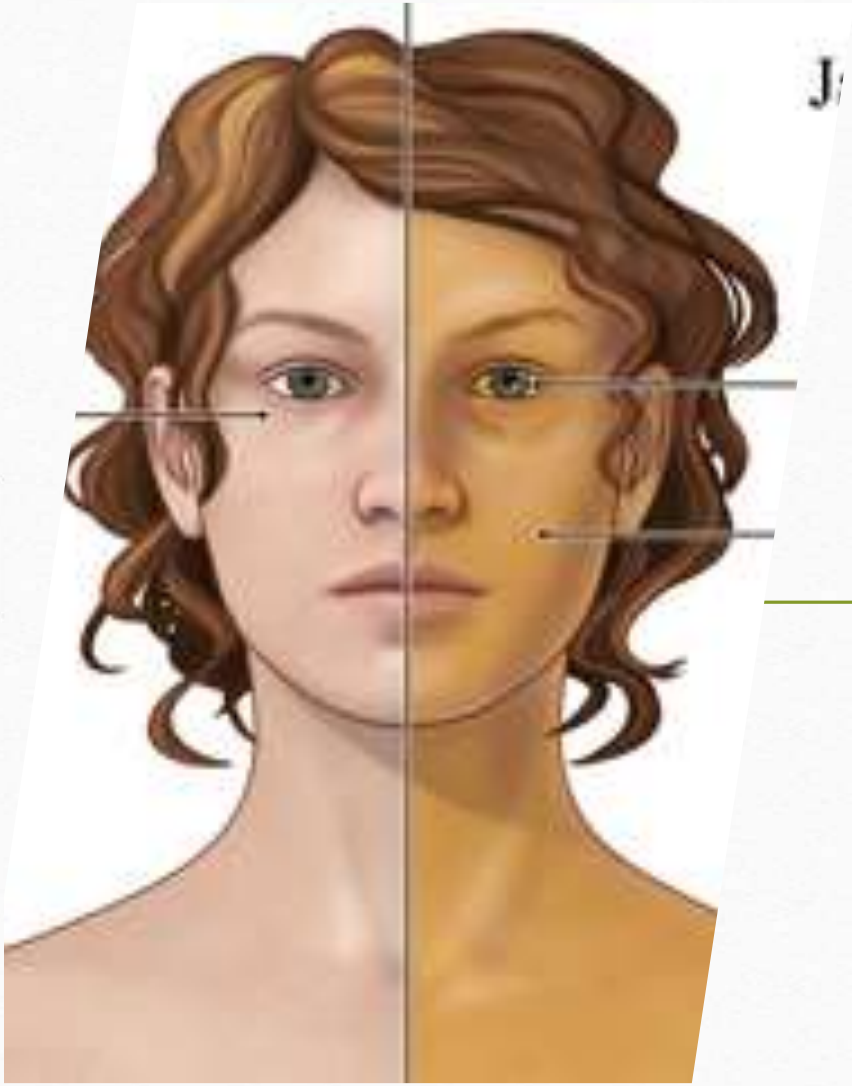
Neck examination

- viral hepatitis can cause Cervical lymphadenopathy
- metastatic pancreatic cancer :
  - Left supraclavicular lymphadenopathy (Virchow's node)



- Cirrhosis : patient apperar weak

- Viral hepatitis : depend on age
- Young tend to be asymptomatic while older tend to symptomatic
- Panceriatic cancer : tend to be in 6^{th} decades appear cachectis pateient



Investigations of jaundice

Investigations of jaundice include
-laboratory test
-imaging
-instrumental

Laboratory investigations

- 1-liver function test

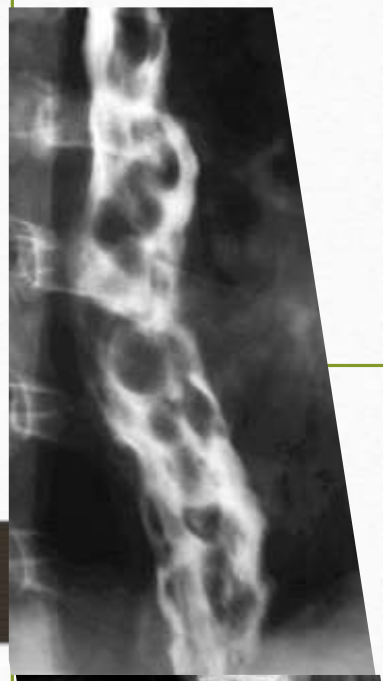
	Unconjugated bilirubin	Conjugated bilirubin	SGOT & SGPT	Alk. Phosph.	Stercobilinogen	urobilinogen
Hemolytic	↑↑	normal	normal	normal	↑	↑
Hepatocellular	↑	↑↑	↑↑↑	↑	↓	↑
Obstructive	normal	↑↑	Normal or ↑	↑↑↑	↓	↓

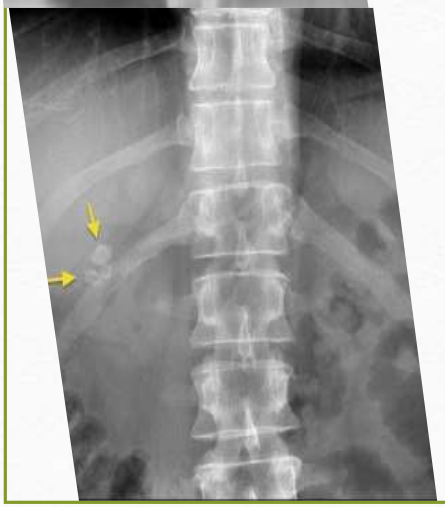
Note that :

- An elevation in the serum alkaline phosphatase concentration can be also derived from extrahepatic tissues , particularly bone so you can measure GGT to confirm the hepatic origin of alkaline phosphatase
- Note that alkaline phosphatase is increased in obstructive jaundice or space occupying lesion (Tumor in the liver)

- 2-blood Picture : features of hemolytic anemia
- 3-ESR: marked increase in malignancy
- 4-serological Test : Hepatitis markers

- 5-Therapeutic Test :
- Vit K : to differentiate between obstructive and hemolyticjaundice (vit K corrects prothrombin time in obstructive jaundice not)
- Cortisone Test : to differentiate between Intrahepatic and extrahepatic





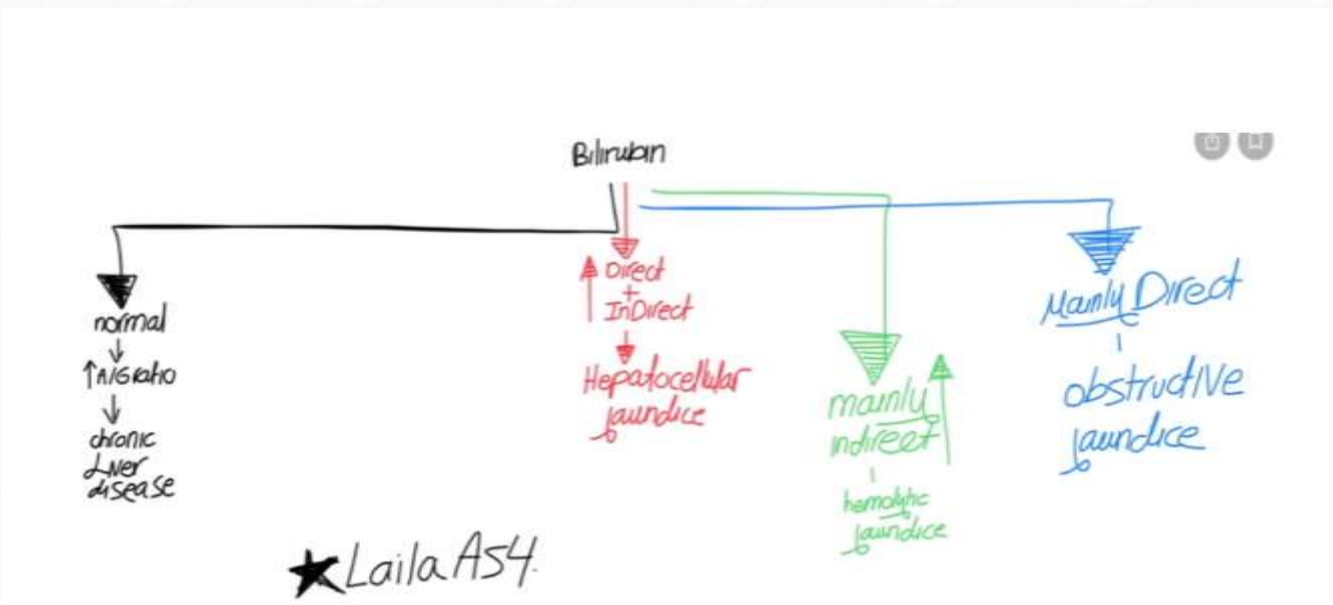
IMAGING

- X-ray : gall-stones
- Barium : swallow :esophageal varies
- Meal : wide C duodenum in cancer of head of pancreas
- Ultrasound
- CT

INSTRUMENT

- 1-EPCP (endoscopic retrograde cholangio – pancreatography) : to investigate obstructive jaundice
- 2-PTC (percutanous Transhepatic cholangiogram)
- 3- liver biopsy (after correction of bleeding tendency)

Diagnostic pathway for jaundice



Thank you