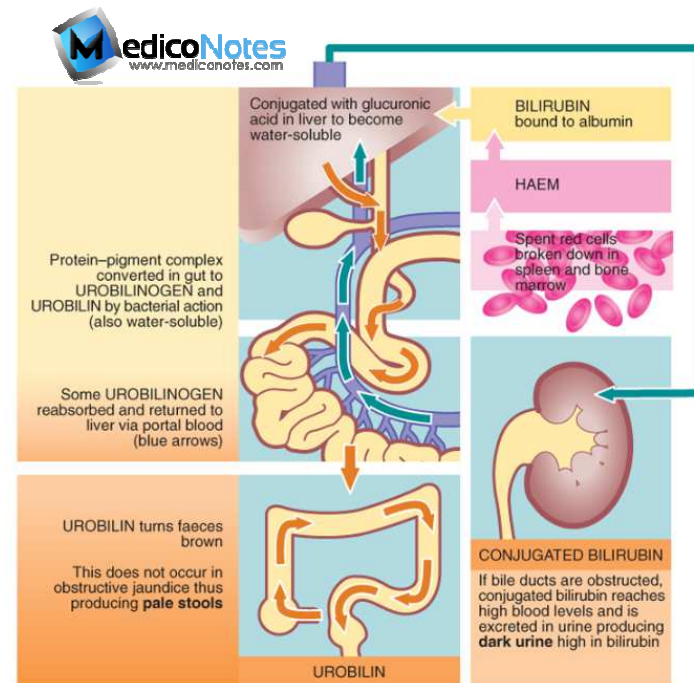


Approach to a jaundiced patient

THE NORMAL ENTEROHEPATIC CIRCULATION

- The **haem** component of spent red cells is normally broken down to bilirubin (mainly in the spleen and bone marrow), bound to albumin and transported to the liver. *This relatively stable protein-pigment complex is insoluble in water and is not excreted in the urine.*
 - In the liver, the complex is split and the bilirubin conjugated with glucuronic acid which makes it water-soluble, before it is excreted into the bile canaliculi. *The normal concentration of both conjugated and unconjugated bilirubin in the blood is very low.*
 - Bacterial action in the bowel converts conjugated bilirubin into colourless **urobilinogen** & pigmented **urobilin** which gives the brown colour to normal faeces.
 - Some urobilinogen is reabsorbed, passing to the liver in the portal blood, and is then re-excreted in the bile. The entire process is called an **enterohepatic circulation**. A small amount of urobilinogen escapes into the systemic circulation and is excreted in the urine, *colouring it yellow.*
- ➔ **Bile acids (salts)** are synthesised in the liver from cholesterol-based precursors. These are excreted in bile to the duodenum and facilitate lipid digestion and absorption in the small intestine. About 95% of the bile acids are reabsorbed in the distal ileum and returned to the liver via the portal vein, only to be re-excreted in the bile ➔ Thus both bilirubin and bile acids are involved in enterohepatic circulations.



PATHOPHYSIOLOGY OF OBSTRUCTIVE JAUNDICE

If biliary outflow becomes obstructed :

- Conjugated bilirubin is dammed back in liver from where it enters bloodstream and causes a gradual rise in plasma bilirubin.
- Once the plasma bilirubin level exceeds about 30 $\mu\text{mol/L}$, jaundice should be clinically detectable. Above 60 $\mu\text{mol/L}$, jaundice is obvious.
- Urine:** Conjugated bilirubin, being water-soluble, is excreted in the urine, turning it **dark urine**.
- Feces:** Diminished or absent excretion of bile into the bowel ➔ less urobilin ➔ causing **pale faeces**.
Diminished bile acids ➔ **defective fat absorption**
The two combine to give the stool a characteristic 'putty' colour.
- Skin:** Biliary obstruction also dams back bile acids, which raises their blood concentration leading to ➔ deposition in skin – causing **intense itching**.
➔ A consequence of poor dietary fat absorption is **malabsorption of vitamin K** ➔ leading to decreased hepatic synthesis of clotting factors (prothrombin) .
• Impairment of blood clotting is not so great as to cause spontaneous haemorrhage or bruising but there is a significant risk of haemorrhage during surgery.
• Thus the patient's coagulation profile must be checked before any invasive procedure.
• The coagulopathy is corrected with parenteral vitamin K or, in the case of an urgent procedure, fresh frozen plasma.

Approach to a jaundiced patient

History-taking

➔ Change in colour of urine and stools, i.e. **dark urine & pale stools** .

- Gall stone disease**, Enquiry about :
 - Episodes of pain typical of gallstone disease,
 - Previous episodes of obstructive jaundice which resolved spontaneously,
 - Previous attacks of acute pancreatitis also suggest gallstone disease.



- Iatrogenic:** Previous biliary tract surgery.
- Drug history:** e.g : oral contraceptive pill-potential for intrahepatic cholestasis
- Risk factors for viral hepatitis:** blood product transfusion, intravenous drug abuse, tattoos, shellfish ingestion, sexual exposure.
- Alcohol intake:** if excessive, predisposes to pancreatitis & cirrhosis
- Symptoms suggestive of malignancy:** anorexia, weight loss & non-specific upper GIT disturbance is common in carcinoma of the pancreas
- History of inflammatory bowel disease:** predisposes to **sclerosing cholangitis** (rare).

Examination

General examination:

- Jaundice** : is first detectable in the sclera of the eye.
- Scratch marks** : In some cases of obstructive jaundice, the patient develops generalised itching (pruritus) and scratch marks
- Stigmata of liver disease:** such as : spider naevi and liver 'flap', are only found when jaundice is caused by primary liver disease
- Enlarged left supraclavicular node (Virchow's node) or periumbilical nodule (Sister Mary Joseph's nodule) suggests an : **abdominal malignancy**.
- Jugular venous distention, a sign of right-sided heart failure, suggests : **hepatic congestion** .

Local Abdominal Examination:

- Abdomen should be examined for :
- Ascites:** Ascites in the presence of jaundice suggests either : 1- cirrhosis or 2- malignancy with peritoneal spread.
 - Enlarged liver or spleen :**
 - An enlarged nodular liver may be caused by primary or secondary malignancy.
 - Splenomegaly & hepatomegaly is an important sign of chronic parenchymal liver disease (usually cirrhosis) & indicates portal hypertension
 - Abnormal masses :** Obvious abdominal mass suggests malignancy.
 - Palpable gall bladder :**
 - Courvoisier's 'law'** : states that obstructive jaundice in the presence of a palpable gall bladder is not due to stone (and is therefore likely to be caused by tumour) :
 - In gall stone: - Gallstones cause chronic inflammation leading to: fibrosis of the gall bladder, which **prevents its distension**.
 - Intermittent stone obstruction leads to : thickening of the gall bladder wall, which **prevents its distension**.
 - In malignancy:
 - Progressive obstruction occurs over a short period and the gall bladder distends easily.
 - Rectal Examination :** Pale stool is characteristic of obstructive jaundice.
➔ The urine should be inspected : - dark yellow or orange from the presence of conjugated bilirubin, and - froths when shaken due to the detergent effect of bile acids.

Approach to investigation of jaundice , step by step as follows:

Laboratory :

1- **Urine tests :** Presence of substantial quantities of bilirubin in the urine which is established by: 1- clinic al or 2- bedside dipstick urine tests

2- **Blood tests :** ➔

A) Enzyme tests : To differentiate between : a **hepatocellular process** & **cholestatic process**

- Patients with a hepatocellular process :** have a disproportionate rise in the aminotransferases compared to the ALP.
- Patients with a cholestatic process:** have a disproportionate rise in the ALP compared to the aminotransferases.
- The bilirubin can be prominently elevated in both hepatocellular & cholestatic conditions ➔, therefore, is not necessarily in differentiating.*

B) Assessment of liver function : All jaundiced patients should have additional blood tests, to assess liver function :

- Albumin level:** - Low albumin level suggests a chronic process such as cirrhosis or cancer.
- Normal albumin level is suggestive of a more acute process such as viral hepatitis or choledocholithiasis
- Prothrombin time:**
 - An elevated prothrombin time indicates either 1- Vitamin K deficiency ,due to prolonged jaundice & malabsorption of vitamin K or 2- Significant hepatocellular dysfunction.

The failure of the prothrombin time to correct with parenteral administration of vitamin K indicates severe hepatocellular injury.

➔ **By liver function tests, Obstructive jaundice is characterised by :**

- Elevated level of plasma bilirubin, predominantly in the conjugated form.
- There is marked elevation of plasma ALP , which is derived from bile canaliculi.
- The transaminases, derived from hepatocytes, are usually only mildly elevated

- When pattern of the liver tests suggests a **cholestatic disorder**, the next step is to determine whether it's :
 - Intra- hepatic cholestasis or
 - Extra-hepatic cholestasis.
- Distinguishing intrahepatic from extrahepatic cholestasis may be difficult. History, physical examination, and laboratory tests are often not helpful.
- The next appropriate test is an **ultrasound**.

Imaging :

A) Hepatobiliary ultrasonography, shows :

- U/S can detect dilation of the intra- and extrahepatic biliary tree :
Absence of biliary dilatation suggests: **intrahepatic cholestasis** / while Presence of biliary dilatation indicates: **extrahepatic cholestasis**.
- Liver secondaries
- Gall bladder abnormalities including stones.
➔ Although ultrasonography may indicate extrahepatic cholestasis, **it rarely identifies the site or cause of obstruction**.
➔ The distal common bile duct is a particularly difficult area to visualize by ultrasound because of overlying bowel gas.

Appropriate next tests include: CT, & ERCP.

B) CT scanning : CT scanning & MRCP are better than ultrasonography for :

- Assessing the head of the pancreas and
- Assessment distal common bile duct for a- choledocholithiasis , particularly when the ducts are not dilated b- small carcinoma .

C) Endoscopy-diagnostic & therapeutic

- If **ultrasound demonstrates dilated ducts** ➔ **ERCP** is frequently next investigation (gold standard for identifying & ttt choledocholithiasis).
- If ERCP failed ➔ Percutaneous Transhepatic Cholangiopancreatography (PTC).

D) Liver biopsy :

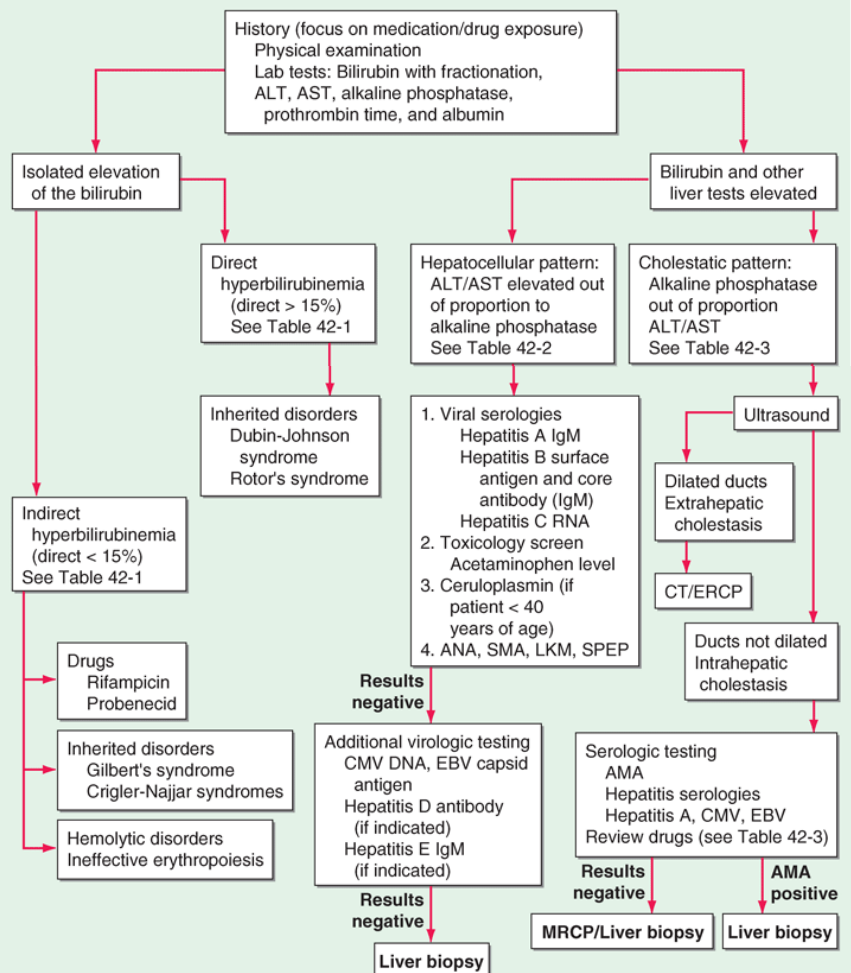
- If **bile ducts are not dilated** (intrahepatic cholestasis) ➔ 1- **Serologic testing in combination with** 2- **Percutaneous liver biopsy**

E) Laparoscopy, indications :

- In patients unsuitable for percutaneous biopsy, or
- Those who require visualisation of other organs,
• Laparoscopy may be used to visualise the liver directly and to obtain biopsy specimens from suspicious areas.

Occasionally, a firm diagnosis cannot be made before operation ➔ abdominal exploration and frozen section histology + opportunity for treatment at the same time.

ALGORITHM FOR PATIENT WITH JAUNDICE



Courvoisier's law :



Conditions causing obstructive jaundice :

