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 canalliculi. 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 "chocolate".

Biological effects on 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.

Consequences of inadequate enterohepatic circulation

- Impairment of blood clotting is not so great as to cause spontaneous haemorrhage or bruising.
- Impaired wound healing may occur.
- The two combine to give the stool a characteristic "bucky" colour.

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.
  - If the plasma bilirubin level exceeds about 30 μmol/L, jaundice should be clinically detectable. Above 60 μmol/L, jaundice is obvious.
  - Unconjugated 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.

Occasional jaundice:

- Iatrogenic:
  - Gall stone disease
  - Oral contraceptive pill episodes of pain typical of gallstone disease.
  - Previous episodes of obstructive jaundice which resolved spontaneously.
  - Thus the patient's coagulation profile must be checked before any invasive procedure.

Distinguishing intrahepatic from extrahepatic cholestasis

- The transaminases, derived from hepatocytes, are usually only mildly elevated.
- There is marked elevation of plasma bilirubin.
- The failure of the prothrombin time to correct with parenteral administration of vitamin K indicates severe hepatocellular dysfunction.
- The distal common bile duct is a particularly difficult area to visualize by ultrasound because of overlying bowel gas.
- CT, MRCP, ERC & ERCP.
- A consequence of poor dietary fat absorption is bile acid malabsorption.
- Extra-hepatic cholestasis.

Common causes of obstructive jaundice

- Previous biliary tract surgery.
- Liver secondaries.
- Biliary obstruction also dams back bile acids, which raises their blood concentration leading to coagulopathy.
- Impairment of blood clotting is not so great as to cause spontaneous haemorrhage or bruising.

Clinical examination:

- The skin,巩膜, and oral mucosa may turn yellow.
- The urine may appear dark amber.
- The stool may be very pale, or even white due to the absence of bile pigments in the gut.
- The two combine to give the stool a characteristic "bucky" colour.

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:
   - To differentiate between: a hepatocellular process & cholestasis process
   - Patients with a hepatocellular process have a disproportionate rise in the aminotransferases compared to the ALP.
   - Patients with a cholestasis process have a disproportionate rise in the ALP compared to the aminotransferases.
   - The two processes are usually elevated in both hepatocellular & cholestasis conditions therefore, is not necessarily in differentiating.

B) Assessment of liver function:

Jaundiced patients should have additional blood tests, to assess liver function:

1. Albumin level: Normal albumin level is suggestive of a more acute process such as viral hepatitis or cholecystolithiasis

2. Prothrombin time:
   - An elevated prothrombin time indicates either 1- Vitamin K deficiency, due to prolonged jaundice & malabsorption of vitamin K ⇒ 2- Significant hepatic dysfunction.

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

   - When pattern of liver tests suggests a cholestasis disorder, the next step is to determine whether it's:
     - Intra or extra heptic cholestasis.

Imaging:

A) Hepatobiliary ultrasound:

- It can delineate the size of hepato-biliary anatomy.
- Contribute to determine the site of the obstruction.
- Presence of bile duct dilatation suggests obstructive jaundice.

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 cholecystolithiasis, particularly when the ducts are not dilated b- small cancerna.

Endoscopy-diagnostic & therapeutic:

- If ultrasound demonstrates dilated ducts ⇒ ERCP is frequently next investigation (gold standard for identifying & treating cholecystolithiasis).

Liver biopsy:

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

Occasionally, a firm diagnosis cannot be made before operation & abdomen exploration or from section histology: opportunity for treatment at the same time.