

LIVER FUNCTION TESTS (LFT)

Functions:

Liver is the largest Organ of the body weighing about 1.5kg.

Liver is called kitchen of our body.

Carbohydrate Metabolism

In **fed state glycogen** synthesis and excess glucose is converted to fatty acid and then TAGS which get incorporated to VLDL and transported to adipose tissue.

In **Fasting state** glucose concentration is maintained by glycogenolysis and gluconeogenesis

Protein Metabolism:

1. Synthesis of albumin and various plasma proteins except immunoglobulins.

Most of the coagulation factors like fibrinogen, Prothrombin(II), V, VII, IX , X , XI, XII, XIII.

Out of these II , VII ,IX, X cannot be synthesized without vitamin –K.

Transport proteins – eg: Transferrin

Amino Acid Metabolism & Urea Formation:

Lipid Metabolism:

Synthesis of lipoproteins, Phospholipids , Cholesterol.

Fatty acid Metabolism – β Oxidation , Ketone body formation,

Bileacid synthesis.

Excretion and Detoxification:

Conjugation and Excretion of bilirubin

Cholesterol is excreted in the bile as bile acids and cholesterol.

Steroid hormones are metabolized and inactivated by conjugation with glucuronic acid and sulphate and are excreted in Urine.

Drugs are metabolised and inactivated by CYT P450 of endoplasmic reticulum and excreted through bile / urine

▪

Miscellaneous function:

Iron storage, vitamins ADE storage, B₁₂ storage.

Note: Liver has very large functional reserve.

Deficiencies of Synthetic functions can only be detected if liver disease is very extensive.

LFT :

- Total Bilirubin 0.2 to 0.8 mg/dl
- Conjugated bilirubin 0 to 0.2 mg/dl
- Total protein 6 – 8 gm/dl
- Albumin 3.5 – 5 gm/dl
- Coagulation Factors – PT- 11 to 12 seconds

Enzymes:

- ✓ ALT(SGPT) – Marker enzyme for liver diseases
- ✓ AST(SGOT)
- ✓ Alkaline phosphatase (ALP)
- ✓ Gama glutamyl transferase (GGT)
- ✓ 5' – Nucleotidase

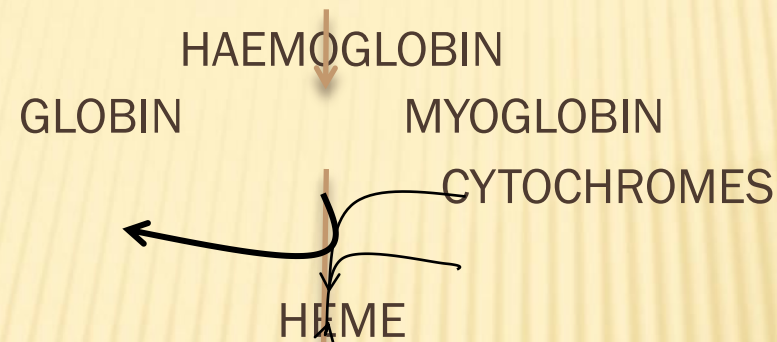
Special tests:

- ✓ Bile acid levels
- ✓ Blood ammonia
- ✓ α 1- antitrypsin
- ✓ α 1-Fetoprotein
- ✓ Hepatitis markers
- ✓ Immunoglobulins
- ✓ Ceruloplasmin
- ✓ Ferritin

Liver Function Tests :

1. Serum Bilirubin :

OLD R.B.C / IMMATURE CELLS



RE
system
i.e.
Spleen
Bone
Marrow

Bilirubin - Albumin PLASMA

GILBERT'S DISEASE x Uptake defect

LIVER

Conjugation defect in

1. Neonatal jaundice
2. Toxic jaundice
3. Crigler najjar syndrome
4. Gilberts disease

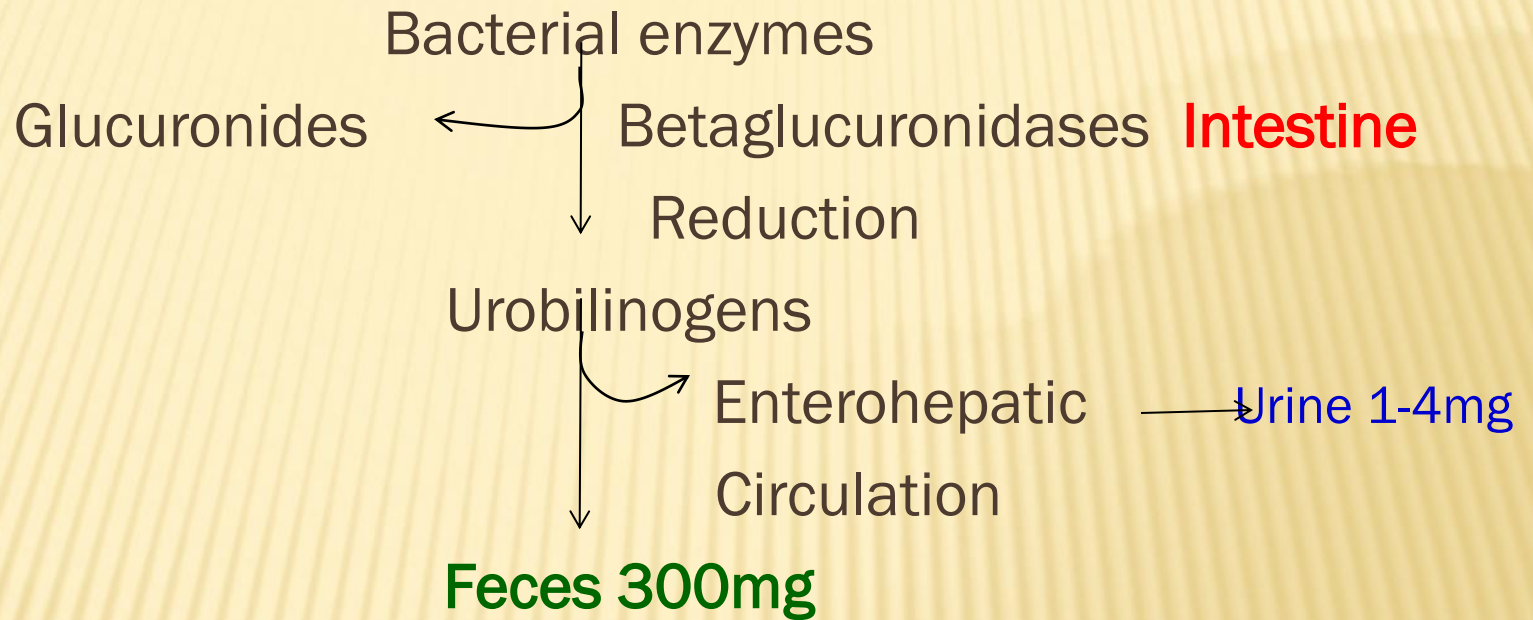


BDG

Secretion Defect

Dubin Johnson syndrome

Bilirubin diglucuronide (BDG)



Total Serum bilirubin 0.2 to 0.8 mg/dl

Conjugated bilirubin <0.2mg/dl

Unconjugated bilirubin 0.2 to 0.6 mg/dl

Van den bergh reaction:

Normal serum gives a negative van den bergh reaction.

Principle of the reaction:

The reagent is a mixture of equal volumes of sulfanilic acid in dilute HCl and sodium nitrite.

That diazotised sulfanilic acid (the above mixture) reacts with bilirubin to form a purple coloured azobilirubin.

Direct Positive:

conjugated bilirubin gives a purple color immediately on addition of the reagent.

Indirect Positive:

Purple color develops only when the reagent and methanol are added.

Unconjugated bilirubin gives color only when methanol is added.

BiPhasic:

Purple color develops on addition of reagent.

Addition of methanol intensifies the color.

Elevation of both unconjugated and conjugated bilirubin

Indirect Positive

Hemolytic jaundice

Direct Positive

Obstructive jaundice

Biphasic

Hepatic jaundice

Bilirubin in Urine:

Normally bilirubin is absent in urine.

Conjugated bilirubin being water soluble is excreted in urine in obstructive jaundice.

This can be detected by Fouchet's test

Urine urobilinogen - normally trace amounts is present.

In obstructive jaundice no urobilinogen is present in urine.

because bilirubin cannot enter intestine.

Note: Presence of bilirubin in urine and absence of urobilinogen in urine is seen in obstructive jaundice.

In hemolytic jaundice increased production of bilirubin causes increased formation of urobilinogen which appears in urine.

Note: Increased urobilinogen in urine and absence of bilirubin in urine is seen in hemolytic jaundice.

Fecal urobilinogen - Normal about 300mg.

Increased in Hemolytic jaundice in which color of feces is dark.

In Obstructive jaundice urobilinogen is not excreted through feces and the color of the feces is pale.

Jaundice

Clinical jaundice appears when bilirubin concentration is more than 3 mg/dl.

Levels between 1 and 3 mg/dl is sub-clinical jaundice.

Classification of Jaundice:

Prehepatic

or

Hemolytic jaundice

or

Unconjugated hyperbilirubinemia

Causes :

Increased production of unconjugated bilirubin from hemolysis - **sickle cell anemia**

Rapid turnover of RBC - Neonate

Physiological jaundice (Bilirubin 5mg/dl).

Kernicterus Bilirubin >20mg/dl.

Brain damage due to entry of bilirubin.

No blood brain barrier.

Decreased uptake of bilirubin by hepatocyte - Gilbert syndrome.

Decreased conjugation - Neonatal Jaundice, drug inhibition, crigler – najjar syndrome, Hepatocellular dysfunction.

Obstructive jaundice:

or

Post hepatic jaundice

or

Conjugated hyperbilirubinemia

Decreased secretion of conjugated bilirubin into canaliculi - Hepatocellular disease, hepatitis.

Decreased drainage - Intrahepatic obstruction by drugs , cirrhosis.

Extra hepatic obstruction - stones , Carcinoma.

Hepatocellular jaundice

Acute hepatitis is usually caused by viral infections
Hepatitis A, C, D, E. (or) by toxins
eg: paracetamol, Carbontetrachloride etc.