LIVER DISEASES

HEPATITIS

- An infectious disease characterised by inflammation and degeneration of liver cells.

❑**Two types**

- Viral hepatitis
- Drug induced hepatitis

- Viral hepatitis is more common and may be either infective (type A) or serum hepatitis (type B)

HEPATITIS A

- VIRAL HEPATITIS
- Caused by HAV.
- Most common cause of acute viral hepatitis(20-25%) and extremely contagious.
- Incubation period- 2-6 weeks.
- Generally transmitted via faecal oral route.

SIGNS AND SYMPTOMS

- Signs and symptoms typically don't appear until you've had the virus for a few weeks but not everyone with Hepatitis A develop them.
- They may be relatively mild and go away in a few weeks and sometimes they may result into severe illness that lasts several months.

- These include:
  - Fever
  - Fatigue
  - Sudden nausea and vomiting
  - Loss of appetite
  - Dark urine
  - Abdominal pain

CAUSES

- Drinking contaminated water
- Eating raw shellfish from water polluted with sewage
- Eating food handled by someone with virus or one who doesn’t thoroughly wash his/her hands after using the toilet.
- Consumption of contaminated food.

DIAGNOSIS

- Specific diagnosis is made by the detection of HAV-specific Immunoglobulin G (IgM) antibody in blood.
- Additional tests include revere transcriptase polymerase chain reaction(RT-PCR) to detect Hepatitis A virus RNA and may require specialized lab facilities.

HEPATITIS B

- Caused by HBV.
- Incubation period- 4- 26 weeks.
- Parenteral transmission through blood transfusion, reuse of contaminated syringe and mother to child, sexual contact.
- It can also be transferred from infected mother to child during the time of birth.

SIGNS AND SYMPTOMS

- Fever
- Fatigue
- Sudden nausea and vomiting
- Loss of appetite
- Dark urine
- Abdominal pain
- Itching
- Jaundice

- A small subset of persons with acute hepatitis can develop acute liver failure, which can lead to death.
- In some people with HBV , it can also cause chronic liver infection that can later develop into cirrhosis or hepatocellular carcinoma. .

DIAGNOSIS

- It is not possible, on ground basis ,to differentiate Hepatitis B from hepatitis caused by other viral agents, hence, laboratory confirmation of diagnosis is essential.
- A number of blood tests are available to diagnose and monitor people with hepatitis B. They can be used to distinguish acute and chronic infection.
- Lab diagnosis of hepatitis B focuses on detection of hepatitis B surface antigen HBsAg.

1.) Acute HBV infection is characterized by presence of HBsAg and immunoglobulin M(IgM) antibody to the core antigen ,HBcAg.

- *During initial phase of infection, patients are also seropositive for Hepatitis B e antigen(HBeAg).* It is usually a marker of high levels of replication of virus . Presence of HBeAg indicates that the blood and body fluids of individuals are highly infectious.

2.) Chronic infection is characterized by the persistence of HBsAg for atleast 6 months.

- Persistence of HBsAg is the principle marker of risk for developing chronic liver disease and hepatocellular carcinoma later in life.

MNT OF VIRAL HEPATITIS

- **OBJECTIVES** of nutritional management of hepatitis:

1) To relieve symptoms
2) To aid the regeneration of liver tissues
3) To prevent further liver damage

DIETARY MODIFICATIONS

1) ENERGY:

- High energy is needed to promote weight gain and ensure maximum protein utilization.
- Initially the patient may not be able to eat such quantities due to anorexia and only 1500-2000kcal may be acceptable.
- Gradually, energy intake may be increased to 20-30% more than normal or 2000-2500kcal daily.

2.) PROTEIN:

- Protein requirements are increased to overcome negative N-balance, to promote regeneration of liver cells, prevent fatty infiltration of liver.
- However damaged liver may not be able to tolerate a high protein load because conversion of ammonia to urea gets affected and there's a danger of impending hepatic coma, therefore, protein is adjusted accordingly.
- In mild-moderate cases, daily intake of 1.5-2g/kg/IBW/day or 80-100g protein is advised.
- Provide protein of high BV or supplements with proteins of vegetable origin.

3.) CARBOHYDRATES:

- A daily intake of 300-400g is recommended.
- High carbohydrate diet is advised to provide bulk to energy and build up glycogen stores in liver as a protection against fatty infiltration and protein sparing action.
- Simple carbohydrate such as glucose, sugar, honey, fruits and fruit juices and starches like cereals and root vegetables.

4.) FATS:

- Since bile secretion is impaired and digestion and absorption of fat is impaired, therefore, fat intake is decreased , though not severely.
- **In mild-moderate** cases, 40-50g total fat per day may be given and **in severe case** 20-30g/day is advisable.
- More than quantity, quality of fat is must.
- Emulsified fats (such as fat from milk and egg) and MCTs are recommended.

5.) VITAMINS:

- Since fat digestion is impaired, therefore, fat soluble vitamins must be supplemented.
- B-group vitamins are increased due to increased energy metabolism.
- Vitamin-C is needed for tissue healing.

6.) MINERALS:

- Diet must provide all minerals , particularly calcium and iron in adequate amount in view of increased tissue catabolism.

DIET AND FEEDING PATTERNS

- Patient must be encouraged to eat since the patient feels anorexic in this state.
- Food must be well-cooked, attractive and appetizing.
- Patient with mild hepatitis may be able to eat a normal consistency diet while in acute case, liquid diet is preferred and in severe cases tube-feeding may have to be done.

CIRRHOSIS OF LIVER

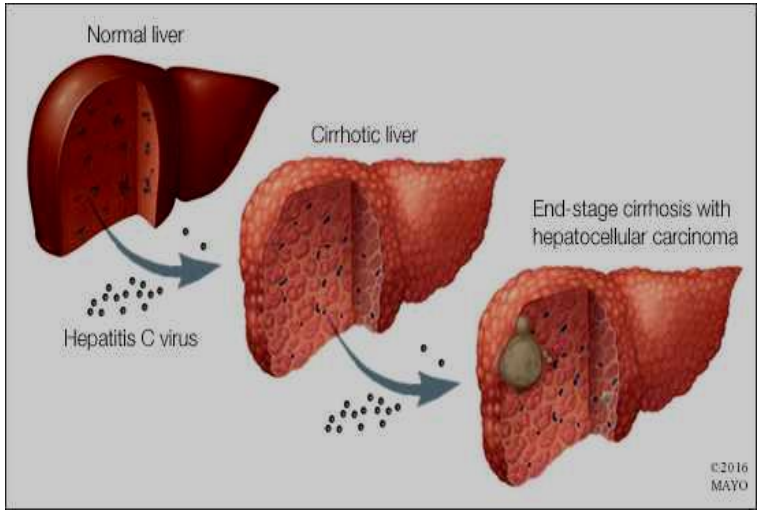
- Hepatic cirrhosis is a common disease characterised by diffuse hepatic fibrosis and nodule formation.
- It is a last and final stage of alcoholic liver disease.
- **Classification of cirrhosis**

1. **Micronodular cirrhosis(Laennec's cirrhosis)**

- It is characterised by small nodules about 1mm in diameter and seen in alcholic cirrhosis

2. **Macronodular cirrhosis**

- It is characterised by larger nodules of various sizes, irregular growing upto several centimeters



CAUSES

- Alcoholic liver disease—damage to the liver and its function due to alcohol abuse
- Nonalcoholic fatty liver disease
- Chronic hepatitis C
- Chronic hepatitis B

- **Some of the less common causes of cirrhosis include**
- Autoimmune hepatitis
- Diseases that damage, destroy, or block bile ducts, such as primary biliary cholangitis and primary sclerosing cholangitis
- Long-term use of certain medicines
- Chronic heart failure with liver congestion, a condition in which blood flow out of the liver is slowed

SYMPTOMS

- Feeling tired or weak
- Poor appetite
- Losing weight
- Nausea and vomiting
- Mild pain or discomfort in the upper right side of your abdomen
- **As liver function gets worse, other symptoms, including**
- Bruising and bleeding easily
- Confusion, difficulties thinking, memory loss, personality changes, or sleep disorders
- Swelling in lower legs, ankles, or feet, called edema
- Bloating from buildup of fluid in your abdomen, called ascites
- Severe itchy skin
- Darkening of the color of urine
- Yellowish tint to the whites of eyes and skin, called jaundice

CLINICAL FINDINGS

- **Jaundice**
- **Portal hypertension**
- **Hemorrhage**
- **Varicose veins** in oesophagus and upper part of stomach develops as a complication of portal hypertension.
- **Ascites –** develops as a consequence of portal hypertension, obstruction of hepatic vein, a fall in plasma colloid osmotic pressure due to impaired albumin synthesis, increased sodium retention or impaired water excretion.
- **Hepatic encephalopathy.**

MNT OF LIVER CIRRHOSIS

- **Objectives** of nutritional therapy are:

1.) To correct the fluid and electrolyte balance

2.) To promote regeneration of liver cells

3.) To correct nutritional deficiencies , if any

DIETARY MODIFICATIONS

1.)ENERGY:

- Energy needs are increased to correct malnutrition and promote regeneration of liver cells.
- In patients with **compensated cirrhosis**, 25-35kcal/kg BW/day is recommended , while in **malnourished cirrhosis patients** the intake should be 35-40kcal/kg BW/day is advised.

2.) PROTEIN:

- Sufficient protein is given to maintain a normal N-Balance. Protein requirement will vary with the state of the disease and the patients condition.
- **In absence of hepatic coma** or encephalopathy, protein intake should be 0.8-1.2g/kg actual body weight/day.
- **If hepatic coma persists**, protein is decreased to 0.5-0.8g/kg body weight/day.
- Emphasis should be mainly on vegetable proteins.

3.) CARBOHYDRATES:

- High carbohydrates diet is recommended to provide energy and protect liver cells from further damage.
- Daily intake of 300g CHO in form of simple carbs are advised.
- Irritating fibers should be eliminated due to presence of esophageal varices. Thus, dehusked pulses, refined cereals and low fiber vegetables and fruits must be selected.

4.) FATS:

- **During the acute stage** , most patients are able to tolerate only 15-20% of energy coming from fat.
- As the condition improves, the amount of fat can be gradually increased.
- Inclusion of moderate amount of fat increases the palatability of diet and promotes recovery.
- Emulsified fats and MCTs are better tolerated.

5.) VITAMINS:

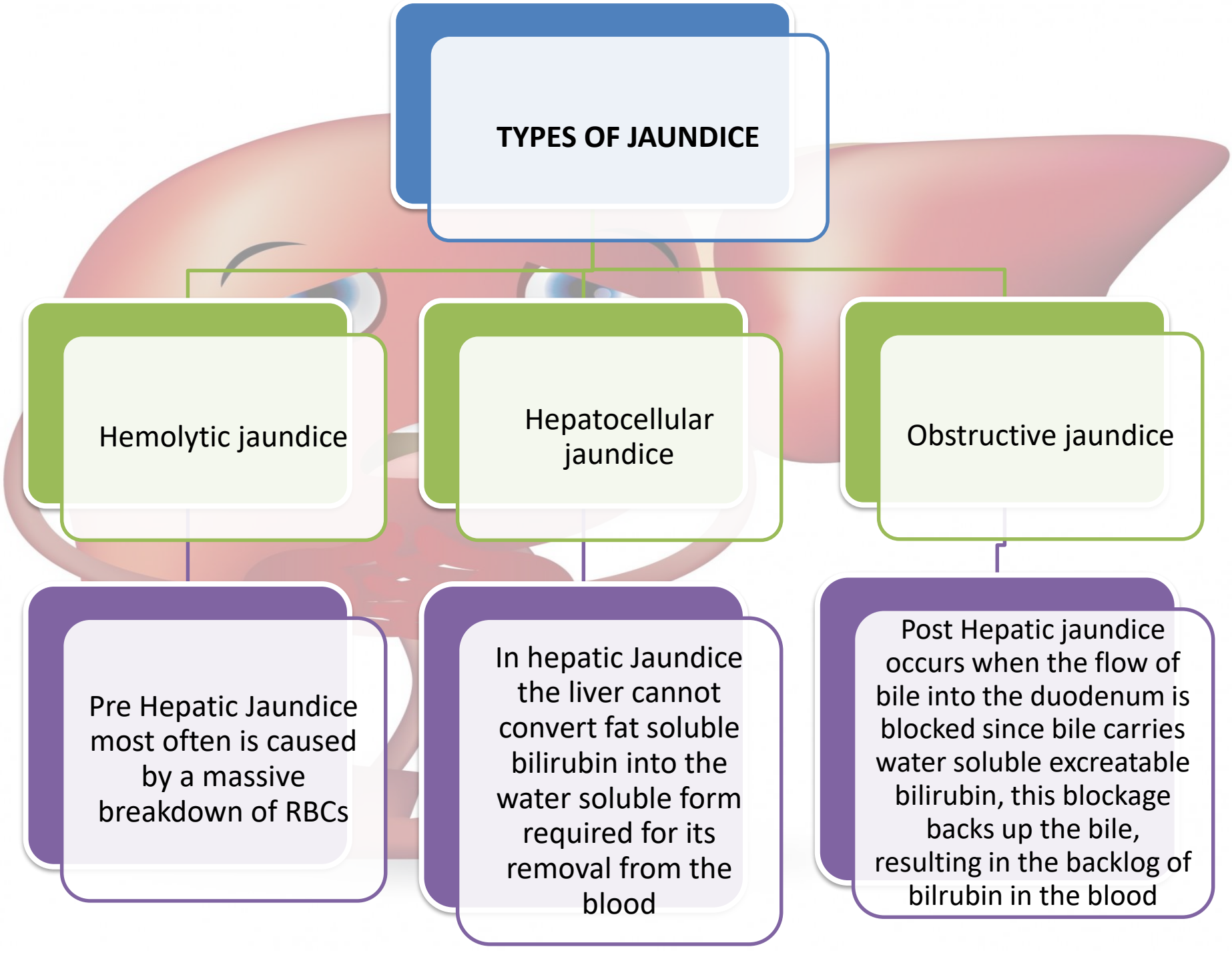
- Availability of fat soluble vitamins like Vitamin A and Vitamin D is affected due to decreased intake and impaired absorption of fat. Thus, diet should be rich in β-carotene rich foods.
- Supplements of Vitamin A and Vitamin D may be recommended in acute cases.
- Supplements of B-group vitamins may have to be provided to replenish liver stores and repair tissue damage.

6.) MINERALS:

- Supplementation with calcium(1000mg/day) and Vitamin D(800IU/day) is considered necessary.
- Zn deficiency is common and supplementation with 600mg zinc daily for 3 months has shown to be beneficial.

Jaundice

- Jaundice is just a symptom, it is not a disease.
- Jaundice also known as icterus, is a term used to describe a yellowish tinge of the skin and sclerae (the white part of the eye) that is caused by hyperbilirubinemia (an excess of bilirubin in the blood)
- When liver is not metabolizing bilirubin , then jaundice occurs.
- Normal Bilirubin ranges 0.5 to 1.0 mg/dl. Levels >3.0 mg/dl to be clinically jaundiced.


TYPES OF JAUNDICE
Hemolytic jaundice
Hepatocellular jaundice
Obstructive jaundice
Pre Hepatic Jaundice most often is caused by a massive breakdown of RBCs
In hepatic Jaundice the liver cannot convert fat soluble bilirubin into the water soluble form required for its removal from the blood
Post Hepatic jaundice occurs when the flow of bile into the duodenum is blocked since bile carries water soluble excreatable bilirubin, this blockage backs up the bile, resulting in the backlog of bilrubin in the blood

Symptoms

- A yellow tinge of the skin and in the white of the eyes
- Dark urine
- Fatigue
- Abdominal pain
- Weight loss
- Vomiting
- Fever
- Swelling of the abdomen due to accumulation of fluid
- Loss of appetite

Portal hypertension

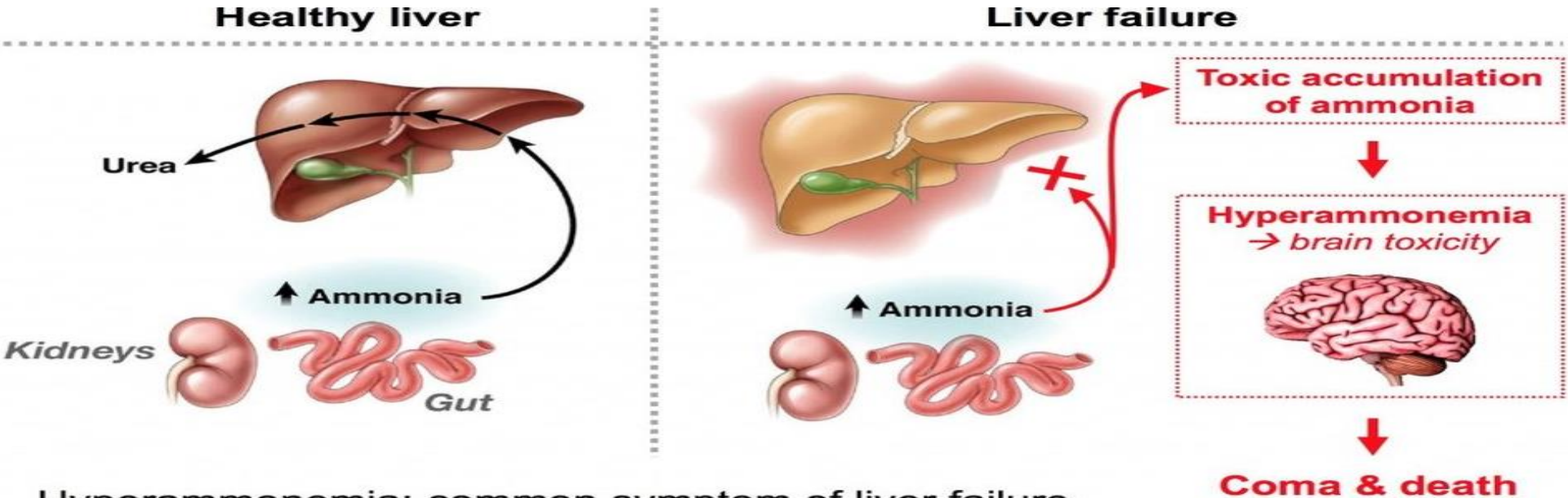
- Increase in the blood pressure within a system of veins called the PORTAL VENOUS SYSTEM in which the portal pressure gradient (PPG) is increased above the upper normal limit of 5mmHg.
- If the vessels in the liver are blocked due to damage, blood cannot flow through the liver and as a result high pressure in the portal system develops.

❑ Classification:

- Prehepatic
- Intrahepatic
- Posthepatic

HEPATIC ENCEPHALOPATHY

- It is a neuropsychiatric syndrome caused by chronic liver disease.
- The toxic products may be ammonia and other nitrogenous substances from intestinal bacteria which reach the systemic circulation without detoxification in the damaged liver and thus damage the brain.
- It is acute, reversible or chronic and progressive.



PRECIPITATING FACTORS

- Gastrointestinal bleeding, fluid and electrolyte abnormalities, uraemia, infections, use of sedatives, hyper/hypo-glycemia, alcohol withdrawal, constipation, dehydration, azotemia, portosystemic shunts and acidosis can precipitate hepatic encephalopathy.

Stages of Hepatic Encephalopathy

Stage	Symptoms
I	Mild confusion, irritability, agitation, sleep disturbance, decreased attention
II	Lethargy, disorientation, inappropriate behaviour, drowsiness
III	Somnolent but arousal, incomprehensible speech, confused, aggressive behaviour when awake
IV	Coma

SYMPTOMS

- Confusion
- Restlessness
- Irritability
- Inappropriate behaviour
- Delirium and drowsiness
- Inco-ordination and a flapping tremor of arms and legs when extended.

Medical Nutrition Therapy

- Treatment is emperical and mainly based on preventing the formation and absorption of gut derived toxins prinicipally ammonia.
- Practice of protein restriction in patients with low-grade hepatic encephalopathy is based on the premise that protein intolerance causes hepatic encephalopathy.
- True dietary protein intolerance is rare except in fulminant hepatic failure, or in a rare patient with chronic endogenous hepatic encephalopathy and they require a transient restriction of protein intake to 0.5g/kg.
- Unnecessary protein restriction may worsen body protein losses and must be avoided.
- More than 95% of patients with cirrhosis can tolerate mixed-protein diets up to 1.5 g/kg of body weight.

- The high-fiber content of a vegetable-protein diet also may play a role in the excretion of nitrogenous compounds.
- Finally, it has been proposed that probiotics and synbiotics (sources of gut-friendly bacteria and fermentable fibers) can be used to treat hepatic encephalopathy.
- Probiotics may improve hepatic encephalopathy by reducing ammonia (Pereg et al, 2011) or by preventing production or uptake of lipopolysaccharides in the gut (Gratz et al, 2010).
- Thus they decrease inflammation and oxidative stress in the hepatocyte (thus increasing hepatic clearance of toxins including ammonia), and minimizing uptake of other toxins.

Diagnostic Markers

Laboratory Tests	Comment
Hepatic Excretion	
Total serum bilirubin	Increased levels may indicate overproduction or impaired hepatic uptake, conjugation or excretion.
Indirect serum bilirubin (unconjugated bilirubin)	Increased with hemolysis, immaturity of enzyme systems, inherited defects, drug effects.
Direct serum bilirubin (Conjugated bilirubin)	Increases with depressed bilirubin excretion, hepatobiliary disease, intrahepatic or benign postoperative jaundice and sepsis and congenital conjugated hyperbilirubinemia.
Cholestasis	
Serum alkaline phosphatase	Increased levels suggest cholestasis but increase can also be seen with bone disorders, pregnancy, normal growth and some malignancies.
D-Glutamyl transpeptidase (GGT)	Levels increase during liver disease, but also after myocardial infarction, in neuromuscular disease, pancreatic disease, pulmonary disease, diabetes mellitus and during alcohol ingestion.

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Laboratory Tests	NORMAL VALUES	Comments
Hepatic serum enzyme		
Alanine aminotransferase (ALT, SGPT)	4-41U/L (M) Upto 33U/L (F)	Increased when liver cells are damaged.
Aspartate aminotransferase (AST, SGOT)	4-40U/L (M) 4-32U/L (F)	Increased when liver cells are damaged.
Serum lactic dehydrogenase	135-214U/L F 135-225U/L M	Levels increase with livr disease but lack sensitivity and specificity because it is found in most other body tissues.
Serum proteins		
Prothrombin time (PT)	11-15 sec.	Vitamin K deficiency and decresased synthesis of blood clotting factors icrease PT indicating liver dysfunction
International normalised ratio (INR)	1.0	Standardised way to report PT levels so that

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Laboratory Tests	NORMAL VALUES	Comments
Serum albumin	3.4-5.2g/dl	Hypoalbumenia can result from expanded plasma volume or reduced synthesis as well as increase losses as occurs with protein losing interopathy nephrotic syndrome, burns, GI bleeding.
Serum globulin	2-3.5g/dL	Levels are high in chronic liver disease
Miscellaneous		
Ammonia	18.7-86.9 µg/dL (Females) 27.2-102 µg/dL (Males)	Increased level may indicate hepatic failure and portal systemic shunts.

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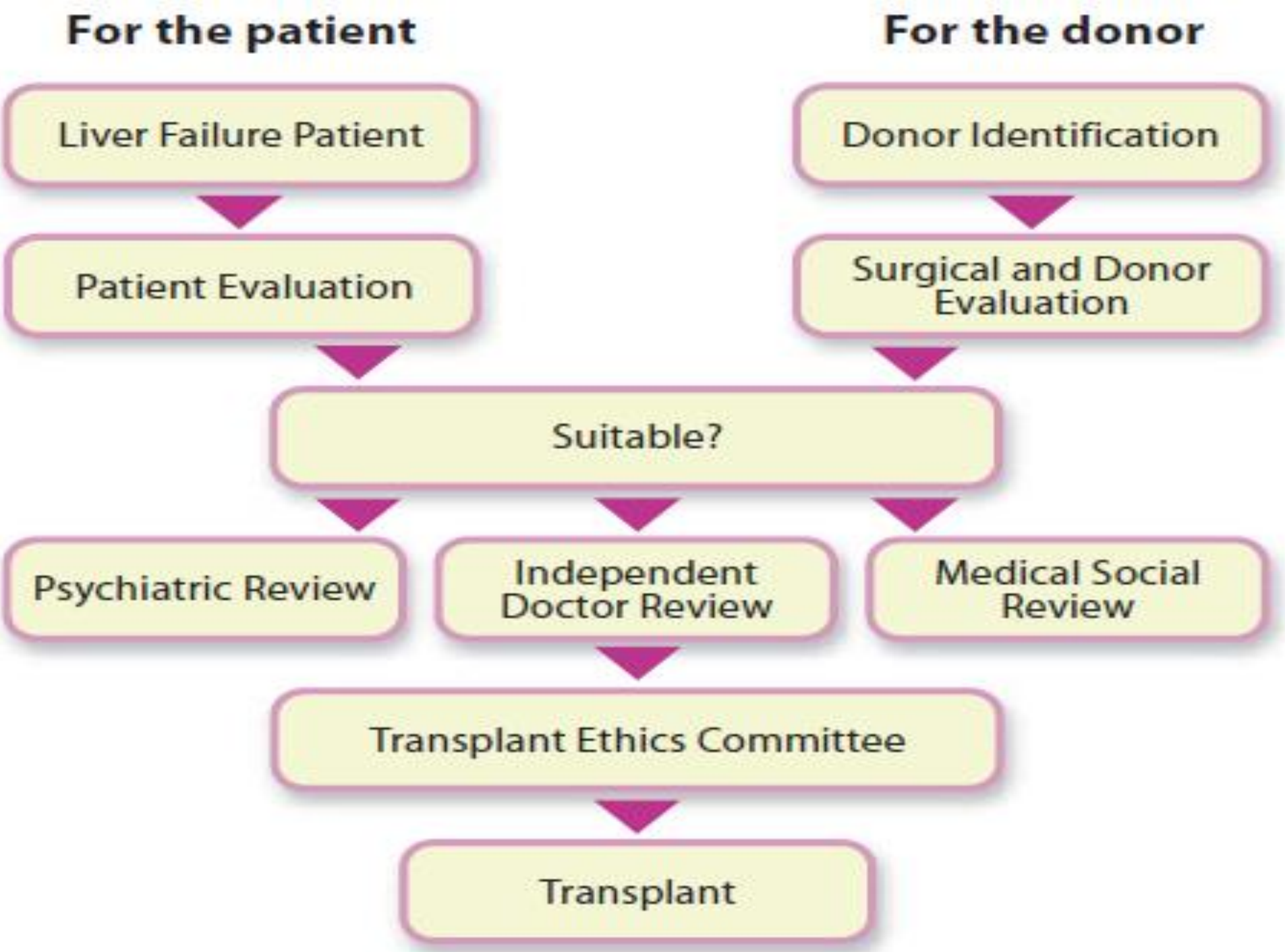
Laboratory tests	Comments
Markers for viral hepatitis	
Anti-HAV IgM	Marker for hepatiris A indicate current or recent infections
HBSsAg	Marker for hepatitis B positive in most cases of acute or chronic infection
Anti HCV	Positive 5-6 weeks after onset of hepatitis C virus , reflex infectious state and is detectable during and after treatment

TREATMENT, PREVENTION AND MANAGEMENT OF LIVER DISEASES

SURGICAL APPROACH
Liver transplant

- Treatment for most end-stage liver disease, confined liver cancer, and fulminant failure not responding to supportive measures
- Need to consider in all decompensated cirrhotics-- encephalopathy, ascites, SBP, variceal bleeding, albumin <2.5
- Used technique is orthotropic transplantation in which native liver is removed and replaced by donor organ in the same anatomic location as the original liver.
- Hepatocyte transplantation. Transplanting only the cells of the liver — not the entire organ — may temporarily delay the need for a liver transplant. In some cases, it could lead to a complete recovery. A shortage of good-quality donor livers has limited the use of this treatment.


For the patient
For the donor
Liver Failure Patient
Donor Identification
Patient Evaluation
Surgical and Donor Evaluation
Suitable?
Psychiatric Review
Independent Doctor Review
Medical Social Review
Transplant Ethics Committee
Transplant

Prevention

- Practice healthy habits—do not drink or eat after anyone with hepatitis. Wash your hands often. Do not touch your mouth or eyes with dirty hands. Do not touch others' blood.
- Maintain a healthy diet—avoid high-fat, acidic foods that can increase the chance for gallstones.
- Get yearly physicals with laboratory blood tests.
- Limit alcohol consumption.
- Stop smoking or taking drugs.
- Limit over-the-counter pain relievers, vitamin supplements and prescription medications that aggravate the liver.
- Get vaccinated against hepatitis A and B.
- Maintain a healthy weight with light exercise and eating a healthy diet.

References

- Mahan L.K., Raymond J.L.; Food and The Nutrition Care Process
- Srilakshmi B.; Dietetics


thank you