UNIT 15 NUTRITIONAL MANAGEMENT IN LIVER, GALL BLADDER AND PANCREATIC DISEASES

Structure

15.1 INTRODUCTION

The last unit dealt with the gastrointestinal disorders. Did you know that when we talk about the gastrointestinal system, we include the liver, gall bladder and pancreas also because of their interrelated functioning? Thus to complete our understanding in this regard let us study the disorders of these vital components of the gastrointestinal system as well.

This section will deal with the disease, etiology, symptoms, complications and the nutritional management goals and dietary management including foods to be avoided, restricted and taken freely in liver, gall bladder and pancreatic disorders.

Objectives

After studying this unit, you will be able to:

- describe the numerous functions of the liver, gall bladder and pancreas,
- discuss the disease conditions of these organs and how the functioning of these organs are compromised in various disease conditions,
- explain the causes of the disease and symptoms produced,
- elaborate on the principles involved in the nutritional and dietary management of these disorders, and
- list the foods that can be given and those avoided in these disease conditions.

15.2 LIVER DISEASES

In our section on liver diseases, let us first get a brief input to understand the normal functioning of liver as an organ. This would help us to understand clearly the variety of conditions associated with the abnormal liver functioning namely infective hepatitis, liver cirrhosis and hepatic coma. From your understanding of the Applied Physiology Course (MFN-001), you already know that liver is the largest and the most complex organ in the body. Let us briefly recall its functions.

Functions of Liver

The functions of the liver are most varied and extensive and any change in the normal function can affect the nutritional status of an individual. The liver is considered one of the most important organs involved in the metabolism of each and every nutrient. Damage caused to liver can impair these and many vital processes in the body. The simple digested products of carbohydrates (glucose, fructose and galactose), fats (fatty acids), protein (amino acids), vitamins and minerals are taken to the liver. The liver stores many nutrients and also produces new compounds, which may contain fat and protein, products that help in clotting of blood. The liver also removes the nitrogen produced as a result of protein breakdown and converts it into urea, which is then excreted from the blood through the kidneys. It also removes several toxins. The liver has a very important role to play in the metabolism of carbohydrates fat and protein metabolism. Now, let us get to know about the various metabolisms.

a *Carbohydrate* Metabolism: Liver cells store energy in the form of glycogen and releases it as glucose when required. This conversion of sugar from carbohydrates is known as *glycogenolysis.* In the absence of carbohydrates the proteins can also be converted to glucose, which is known as gluconeogenesis in the liver cells. Both glycogenolysis and gluconeogenesis help to maintain normal blood sugar levels. Figure 15.1 highlights the connection between the gut and the liver.

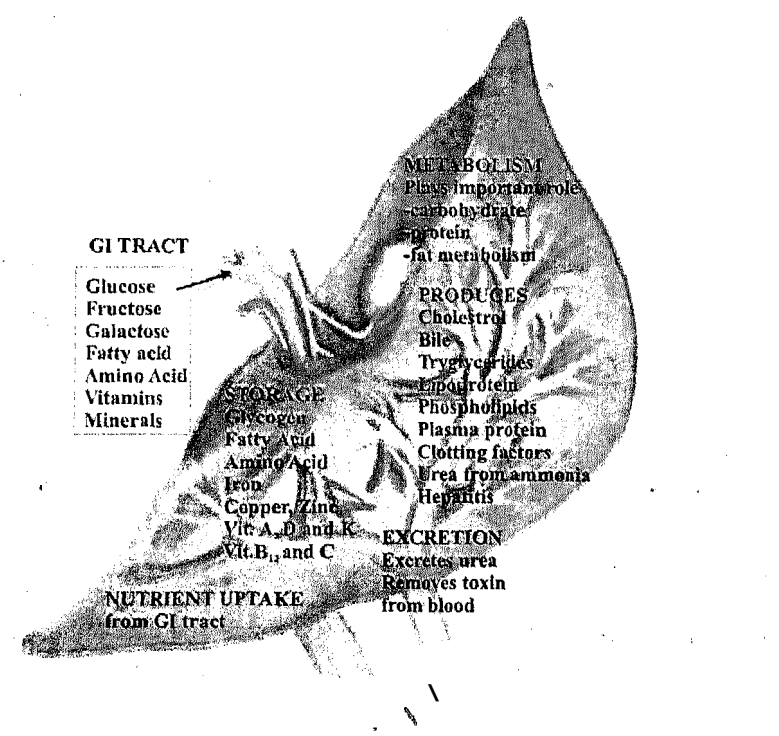

Figure 15.1: Normal functions of liver

- ***Fat metabolism:*** It relates to the production (synthesis) of triglycerides and phaspholipids. Liver synthesizes lipoproteins, which are required for the transport of lipids to peripheral tissues for use or storage. It synthesizes cholesterol and converts 80% into bile and conjugated bile salts and the remainder is transported

in the form of lipoproteins. It is also involved in the oxidation of fatty acids to acetyl CoA to give energy and the synthesis of bile and conjugation of bile salts.

- *Protein Metabolism:* Liver renioves the nitrogen from amino acids (known as deamination), which then could be used as an energy source or converted, to carbohydrates and fats. It also converts ammonia to urea (a waste product of protein breakdown). It is the site of most plasma protein syntliesis. It acts as a reserve of these proteins to replenish serum proteins. It maintains the level of non-essential amino acids, which promote tissue synthesis.

- *Mineral and Vitamin Metabolism:* Liver is a storehouse of iron and is essential for haemoglobin formation and is stored in the form of ferritin. The liver destroys the KBC and recovers the iron from it. Other minerals like zinc and copper and many vitamins are also stored in the liver and play a role in the enzymatic reactions in metabolism. Liver is a storehouse of all fat-soluble vitamins. It is involved in the conversion of carotene to retinol. It is also needed for the activation of prothrombin and conversion of vitamin D to its active form 1,25 dihydroxy cholccalciferol.

- *Other functions*: The liver converts carotene to retinol–a form of vitamin A. It syntliesizes heparin (anticoagulant) which prevents intra-vascular coagulation of blood.

We have read about the numerous functions of the liver. We can also understand that in any infection, inflammation or damage to the liver the normal working of tlie liver would be affected, this could be related to the storage function of bile, protein, fat and carbohydrates and other compounds, problems with waste production and excretion and detoxication of poisons and toxins. An infected liver becomes sluggish and the patient shows signs of lack of appetite, symptoms of jaundice (yellow colour due to bile) and even increase in liver enzymes. Jaundice is not a disease by itself but a disease. So let us know more about it.

Jaundice

Jaundice is a term given to the yellow discolouration of the skin, mucous membranes, sclera and body tissues because of accumulation of bile pigments, in the blood. It results due to an increase in bilirubin content of the blood above the normal range (0.2 to 0.8 mg/100 dl plasma). The red blood cells (RBC's) are broken down in the liver after a duration of 120 days. The haemoglobin gives a pigment known as bilirubin. Under normal conditions, the lives cells absorb bilirubin and secrete it along with other bile constituents. If the liver is infected or diseased or the flow of bile is obstructed or if excess bile is produced then it gets accumulated in the blood and eventually causes jaundice. Based on the cause of jaundice it can be of three types: haemolytic, hepatic and obstructive jaundice. Let us get to know about these types of jaundice.

- *Haemolytic Jnundice:* It is also known as *pre hepatic jaundice*. This relates to excessive destruction of RBC resulting in an increased bilirubin formation and anaemia. There is an increased unconjugated plasma bilirubin, which is excreted through the urine (pigment known as urobilinogen). A healthy liver can handle a bilirubin load 6 times greater than normal before unconjugated bilirubin accumulates in plasma. Thus, this kind of jaundice is seen normally in individuals with congenital defects like sickle cell anaemia, thalassemia, blood transfusion reactions and septicemia.

- ***Hepatic*** *Jaundice:* In this, there is a normal bilirubin production. The liver cannot convert fat soluble bilirubin to the water soluble form. Hence, there is a decreased conjugation leading to hepatocyte damage. An excessive amount of bilirubin is seen as a mixture of unconjugated and conjugated bilirubin. This condition leads to hepatocyte damage (jaundice). Failure of about 80% or more of hepatic functions is observed. Its clinical features include liver disease, increased unconjugated plasma biliburin and increased alanine aminotransferase (ALT)/ aspartate transaminase (AST) enzymes.

- *Obstructive Jaundice*: It is referred to as post-hepatic jaundice. This results from the interference of normal flow of bile into the duodenum due to stones, tumors or iriflammation of mucosa of the duct. This results in a backflow of bile into the blood stream and is circulated in the body giving a yellow colour.

Toxic Jaundice: It is also known as hepatocellular jaundice. It originates from poisons, drugs or viral infections of the liver.

Having learnt about jaundice, which you know is a symptom rather than a disease condition specific to liver diseases, next we shall learn about the conditions associated with the abnonnal liver functioning namely infective hepatitis, liver cirrhosis and hepatic coma. We begin with infective hepatitis.

15.2.1 Viral Hepatitis

Hepatitis is a condition of inflammation of liver. which can result in damage of the liver cells. A virus causes viral hepatitis, as liver cells are particularly susceptible to such infections. It causes damage to the liver cells and interferes with the uptake of bilirubin by the cells, and its conjugation and excretion. It can be either in form of an acute or chronic condition and is caused due to different strains of viruses such as A, B, C, D and E. Let us next learn how are these transmitted and the complications they can cause:

1. *Hepatitis A*: It is commonly called infectious hepatitis caused by a known virus Hepatitis A (HAV). It is common arnong children and young adults. It is contracted through contaminated water, food and sewage and transmitted by faecal-oral route.

2. *Hepatitis B and C :* It is caused by a virus hepatitis B (HBV) and hepatitis C (HCV). It is more severe and prolonged in nature, and can be fatal. It is transmitted by blood transfusion from a carrier, improperly sterilized medical instruments, dental drills, skin puncturing instruments that come in contact with contaminated blood, sexual contact and saliva of an infected person. Chronic active hepatitis can develop leading to cirrhosis and liver failure.

3. Hepatitis *D* : Hepatitis D virus (HDV) is dependent on the HBV for survival and propagation in humans. It may be a co-infection (occurring at the same time as HBV) or a super infection (superimposing itself on the HBV carrier state). This form of hepatitis becomes chronic.

4. *Hepatitis E* : Hepatitis E virus is transmitted via oral fecal route. Contaminated water is the major factor. Overcrowded unsanitary areas are prone to acute form of this type of hepatitis.

Acute hepatitis settles within a few (usually six) weeks and patient becomes asymptomatic. Hepatitis A virus is an example. Chronic hepatitis a more complex form of disease is caused mostly when acute hepatitis is neglected. The various symptoms of hepatitis are:

- itchy skin
- fatigue and fever
- lack of appetite
- nausea and vomiting
- weight loss
- jaundice
- enlarged liver and spleen
- mood swings
- pain in joints of the body (osteomalacia and osteoporosis)
- autoimmune problems

- associated with high risk of cancer.

These symptoms may be seen in both acute and chronic hepatitis, however, additional symptoms during severe chronic hepatitis may include Chronic inflammation of liver, fibrosis and finally death of liver cells (necrosis). Hepatitis virus B and C are known to elicit these symptoms.

Having learnt about the acute and chronic hepatitis, let us next get to know about the causes for this condition.

Etiology

The various causes of acute and chronic liver disease are enumerated herewith.

- Acute Liver Disease (recent origin)
  - Viral Infection (hepatitis)
  - Non Viral Infection (*Coxiella burnetti*)
  - Drugs (paracetamol)
  - Alcohol
  - Poisons (Aflatoxin).
  - Others e.g. complications of pregnancy.
- Chronic Liver Disease (Hepatitis)
  - Drugs and Toxins (Alcohol, Isoniazid, Methotrexate).
  - Neglected/acute infections (Hepatitis B, C virus)
  - Auto Immune Disease
  - Metabolic Disorders e.g. Wilson's Disease, I-Iaernochromatosis ,Type IV glycogen storage disease
  - Alcohol
  - Biliary Obstruction (Gall stones, narrowing of duct)

Now you can understand the differences between acute and chronic hepatitis in terms of symptoms and causes. The latter is more serious and if unattended can lead to a more serious damage to the liver. The disease is called liver cirrhosis. We shall learn about this in greater details next.

15.2.2 Liver Cirrhosis

Cirrhosis is a complication of many liver diseases that is characterized by abnormal structure and function of the liver. It is the final stage of liver injury and degeneration. We have already said that neglected chronic hepatitis can progress to liver cirrhosis. In this the liver cells get inflamed, fibrous septa get develop and the liver cells die and finally nodules develop which lead to obstructions and liver failure. In other words, the active liver tissue is replaced by inactive tissue incapable of normal functioning. Such cells get filled with fibrous tissue and fat.

Thus, cirrhosis develops when the repair that is associated with the dying liver cells causes scar tissue to form. The liver cells that do not die multiply in an attempt to replace the cells that have died. This results in clusters of newly formed liver cells (regenerative nodules) within the scar tissue.

The symptoms of liver cirrhosis are enumerated next.

Symptoms

The common symptoms include:

- GI disturbances (anorexia, nausea, vomiting, abdominal pain and distension)
- Electrolyte and fluid imbalance

- Weight loss and muscle wasting
- Abnormal serum amino acid levels
- Fatty infiltration of the liver
- Severe jaundice
- Hepatic encephalopathy (mental problems ranging from mild confusion to coma)
- Bleeding tendency
- Ascites (accumulation of fluid in the abdominal cavity)
- Osteomalacia and osteoporosis
- High drug sensitivity .
- Chronic inflammation of the liver
- Fibrosis and fatty infiltration of the liver
- Necrosis (death of cells)

So then, what is the cause for this liver condition? There are many causes of cirrhosis. These are listed next.

Etiology

The etiology of cirrhosis can be enumerated as under:

- Neglected acute/chronic hepatitis
- Alcoholism associated with malnutrition
- Virus and toxins
- Metabolic disorders
- Prolonged biliary stasis.
- Altered immune response

Wilson disease is a rare autosomal recessive disorder, characterized by an abnormal copper transport and storage mechanism resulting in an excessive copper deposition in body tissues, mainly in brain, kidney, cornea including liver causing cirrhosis.

Majority of the cases of cirrhosis are, however, due to chronic abuse of alcohol, which has a hepatotoxic effect leading to malnutrition. Since excess alcohol is a major cause of liver disorder, let us understand the complications that arise from excess alcohol consumption. Figure 15.2 depicts the complications.

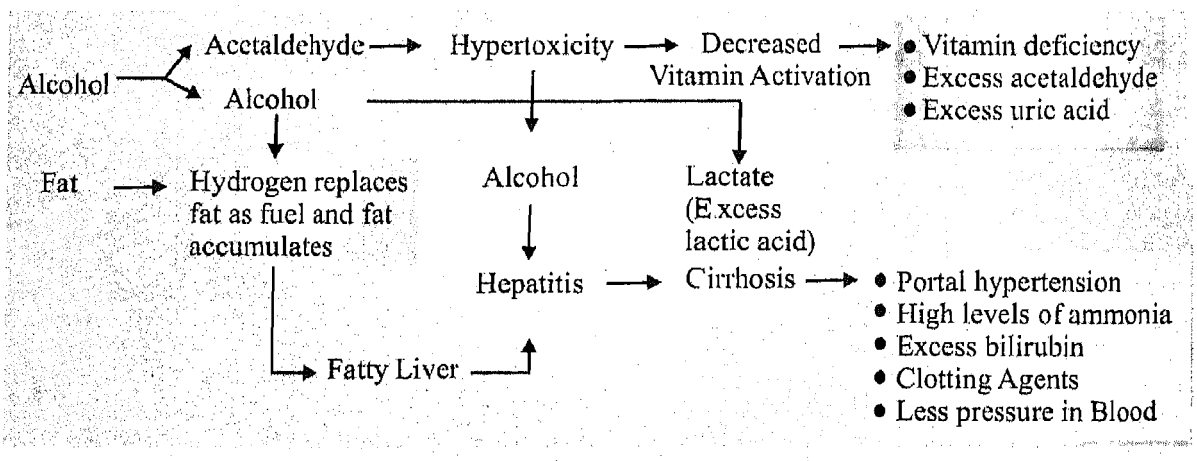

Figure 15.2: Complications of excessive alcohol consumptions

Alcohol consumption can harm the liver by causing inflammation, necrosis due to fat accumulation in the cells which reduces the normal functioning of the liver causing serious vitamin and protein deficiency leading to malnutrition and changes in the metabolism of carbohydrate, protein and fat. For example, clotting defects could

precipitate excess bleeding, protein deficiency could cause cncephalopathy and result in coma. Portal hypertension could result in ruptured varices and ascites. Excess uric acid could precipitate gout and mineral deficiencies. Excess alcohol intake can cause multiple complications and malnutrition. Box 15.1 gives the various causative factors of malnutrition in liver disease. It can also affect the kidney, heart and the vessels and may cause other problems such as oedema or ascites, bleeding. gout and acidosis. This is clearly seen in figure 15.2 also.

The pathogenesis of alcoholic liver disease progresses in 3 stages. Let us briefly understand these stages.

Stage 1: Hepatic Steatosis or ***Fatty Liver***

During this stage, the fat infiltrates into the functioning liver cells and cause problems in normal functioning of liver. The excess fat could come from the fat stores of the body, increased production of fat in the liver. This stage is reversible with abstinence from alcohol and if abuse continues, it can lead to hepatitis and cirrhosis.

Stage 2: Alcoholic Hepatitis

Alcoholic Hepatitis is characterized by hepatomegaly (enlargement of liver). Patients have abdominal pain, anorexia, nausea, vomiting, weakness, and diarrhoea, weight loss and fever. If patient discontinues the alcohol intake, hepatitis may resolve or else it progresses to the third stage of alcoholic cirrhosis.

Stage 3: Alcoholic Cirrhosis

In this stage, patients develop further complications of ascites, gastrointestinal bleeding, portal hypertension, hepatic encephalopathy and other symptoms of liver disease.

At this stage, let us look at the general complications linked with liver cirrhosis. These are listed herewith.

Complications

Major complications of cirrhosis include:

- Ascites (accumulation of water in abdomen)
- Upper gastrointestinal bleeding (oesophageal varices)
- Hepatic coma or Hepatic Encephalopathy

A brief discussion on each of these follows.

- *Ascites:* It is a characteristic symptom of advanced stage of liver cirrhosis. It relates to the accumulation of massive quantities of fluid in the peritoneal cavity of the abdomen. This may be due to:
  a) Portal hypertension (obstruction of portal blood vessels that increase intra hepatic pressure),
  b) Hypoalbuminaemia (a fall in colloidal osmotic pressure due to inadequacy of serum albumin), and
  c) Renal dysfunction (increased renal tubular sodium resorption; and water retention).
- *Oesophageal* ***varices:*** It relates to a state of varicose (distended or dilated) veins in the oesophagus and upper part of the stomach, which develops as a consequence of portal hypertension. Upper GI tract bleeding may be the risk associated with this state.
- Hepatic *Coma*: It relates to a state of confusion, apathy, personality changes, asterixis (tremor of the hands when extended in front of the chest) and spasticity. We shall deal with it in much more details later in this unit.

Box 15.1 Malnutrition in Liver Disease

Malnutrition is predominant in liver disease and it can be related to a number of factors such as decreased intake of food, impaired digestion and malabsorption, increased energy needs, inefficient protein synthesis, accelerated protein breakdown and increased protein oxidation. These factors are reviewed herewith.

1. Decreased intake of food is generally due to:
   - Anorexia
   - Nausea and vomiting
   - Early satiety
   - Unpalatable diet
   - Drugs used
2. Impaired digestion and absorption could also result due to:
   - Pancreatic insufficiency
   - Bile salt deficiency
   - Impaired absorption
   - Mucosal defect (portal hypertensive enteropathy), major defect is in fat digestion and absorption leading to steatorrhoea.
3. Increased energy requirements: the energy needs increase considerably and if not met worsens the condition of liver
4. Insufficient protein synthesis
5. Accelerated protein breakdown
6. Increased protein oxidation

All 4,5 and 6 factors related to protein metabolism, require an increased protein intake and if not met leads to malnutrition.

7. Alcohol consumption replaces food in the diet and supplies empty calories. Alcohol is a source of empty calories (7 Kcal/g) and does not contain any other vital nutrient. The energy supplied also is lost to a great extent due to the wasteful pathways which alcohol metabolism takes. Alcohol causes inflammation of the stomach, pancreas and intestine and interferes with the normal processes of digestion and absorption. It may lead to malabsorption of nutrients like thiamin, vitamin B_{12}, folic acid and vitamin C, Alcohol gets converted to acetaldehyde, which in turn interferes with the activation of vitamins by the liver cells. So numerous dietary nutrients are not synthesized or activated which leads to malnutrition. Alcoholism and malnutrition forms a vicious circle that is difficult to break.

Finally, let us get to learn about hepatic coma.

15.2.3 Hepatic Encephalopathy (HE) or Hepatic Coma

Hepatic encephalopathy is brain and nervous system damage that occurs as a complication of liver disorders that reduce liver functioning (as in hepatitis or cirrhosis). It is a complex syndrome characterized by neurological disturbances. The symptoms associated with it are: changes in mental state, consciousness, personality and behaviour changes characterized by the following signs – mild confusion, euphoria or depression, decreased attention, slowing of activity to perform mental tasks, irritability, and disorder of sleep pattern, drowsiness, lethargy, speech disorientation, incomprehensive speech and finally coma.

Besides the neurological changes the blood picture shows some abnormalities. These include:

1. Elevated blood ammonia levels (ammonia not converted to urea by liver)
2. High blood concentration of aromatic amino acids (AAA), especially phenylalaninc, tyrosine and tryptophan, an increase in methionine, lysine, glutamine, asparagine and histidine, threonine, glycine and serine which are the ammoniogenic amino acids, In other words they can produce ammonia which worsen the condition.
3. Low levels of branched chain amino acid (BCAA), leucine, isoleucine and valine in plasma, due to a depression in the process of gluconeogenesis and ketogenesis – (the processes through which they are used as a source of energy by skeletal muscle, heart and brain).
4. Altered plasma amino acids composition (decreased ratio of BCAA to AAA).

There are four clinical stages of hepatic encephalopathy.

1. Stage I – Mild confusion, euphoria or depression, decreased attenlion, agitation, irritability, sleep disturbance, slowing of ability to perform mental tasks.
2. Stage II – Lethargy, disorientation, inappropriatc behaviour, irritability in performing mental tasks.
3. Stage III – Somnolent but arousable, incomprehensible speech and confused aggressive behaviour when awake.
4. Stage IV – Coma.

The causes for this liver condition are enumerated next.

Etiology

The cause of encephalopathy is unknown, but there are three proposed mechanisms leading to it. These include:

1. Accumulation of increased toxins due to impaired liver functions. Excess ammonia being the major toxin.
2. Altered plasma amino acid composition. Decreased ratio of BCAA to AAA, which leads to false neurotransmitter impulses in the brain and hence neurological symptoms.
3. An increase in serum and brain neuro-inhibitory substances, like increased gamma-amino butyric acid (GABA) levels.

Before we move on the management of liver disease, let us review what we have learnt so far.

Check Your Progress Exercise 1

1. List the various liver functions and briefly give the role of liver in fat metabolism.

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2. Mention one symptom common to all liver diseases? Mention the physiological changes, which occur as a consequence of this symptom.

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3. What do you understand by the term 'liver cirrhosis"? Enumerate the various etiological factors involved in its pathogenesis.

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4. Discuss the pathogenesis and major complications of alcoholic liver disease.

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The discussion so far focused on the numerous functions of the liver. We also looked at the fact that in any infection, inflammation or damage to the liver the normal working of the liver is affected. Common liver diseases include vital hepatitis, cirrhosis, hepatic coma. An infected liver becomes sluggish and the patient shows signs of lack of appetite, symptoms of jaundice (yellow colour due to bile) and even increase in liver enzymes. Jaundice is not a disease by itself. It is a symptom. So what can be done to manage these liver diseases? The nutritional management of liver diseases is highlighted next.

15.3 NUTRITIONAL MANAGEMENT OF LIVER DISEASES

Having gone through the information presenled in the section(s) above, it must be evident that malnutrition is predominant in liver diseases and it can be related to a number of factors such as decreased intake of food, impaired digestion and malabsorption, increased energy needs, inefficient protein synthesis, accelerated protein breakdown and increased protein oxidation. The diet of the patient must bc individualized. The main goals of dietary management for a liver disease patient should be:

- Maintain adequate nutrition
- Prevent breakdown of body protein tissue
- Control of oederna and ascites, and
- Prevent symptoms of encephalopathy

However, since each disease condition is specific the dietary recommendations too are specific. We shall look at the dietary recommendations for each disease condition separately. Table 15.1 gives broad dietary guidelines for liver patients.

Table 15.1: Dietary guidelines for liver patients

Nutrients	Hepatitis	Cirrhosis	Encephalopathy
Kilocalories (Kcal)	3000 and above	2000-3000	1800
Carbohydrates (g)	300-400	300-400	450
Protein (g)	High 1.5-2 g/ kg (100 g or more)	Moderate 0.8-1g/kg (70 g)	Low 0.5 g/kg (20-40 g)
Fat (g)*	Moderate 30% total Kcal	Low 25% total Kcals	Initially restricted slowly increase to 25-30%
Vitamins and minerals**	Vitamin B and C, Folic acid, calcium, magnesium and zinc	B group and C, Fe if anaemia, Ca, Mg and Zn	Vitamin B and C, Fe if anaemia, Ca, Mg and Zn

*MCT (medium chain triglycerides): more digestible and shown to decrease fatty infilteration.

**Restrict Sodium if oedema or ascites

Let us now review the dietary guidelines for each condition one by one. We begin with the dietary recommendations for viral hepatitis.

15.3.1 Dietary Recommendations for Viral Hepatitis

An increased carbohydrate, increased protein and moderate fat diet is advised in the case of viral hepatitis with vitamin and mineral supplementation as you may have noted in Table 15.1 above. The requirements for individual nutrients are reviewed further.

Carbohydrates: Liberal intake of CHO is advised (300-400 g). This is to prevent endogenous breakdown of protein thus having a protein sparing effect, increase the (intra hepatic) glycogen stores to improve the functioning and protect the liver against infectious agents.

The caloric *intake* advised for adults is 35-40 Kcal/kg IBW or as per the requirement to maintain a desirable body weight.

Proteins: Moderate protein intake in the diet is required for the following reasons:

- to prevent negative N_2 balance, which may lead to hypoproteinemia,
- for adequate tissue regeneration especially of parenchymal cells; and
- prevent fatty infiltration of liver cells

Thus, 1.5 to 2.0 g/kg IBW protein is recommended. Supplements of high protein beverages are recommended in between the meals.

Fats: Fats should not be severely restricted as they can make the food unpalatable. About 20% of the total calories should be from Eat. MCT are preferred as they are easily digestible and assimilable (40-50 **g).** For examples dairy fat, cream and butter are preferable.

Vitamins: Supplementation of B complex vitamin and **C** should be given.

Minerals: Sodium restriction is required only if there is fluid retention. Potassium supplements are necessary with diuretic therapy. Iron supplementation is needed only if there is anaemia.

Keeping these considerations in mind, the food items for a patient with viral hepatitis, showing symptoms of obstructive jaundice have been included in Table 15.2 for your reference. You can use this information while planning diets for hepatitis patients in the Practical Manual (MFNL-005).

Table 15.2: Food items for a patient with viral hepatitis or obstructive jaundice

Freely given foods	Foods to be avoided
Cereals - Bread or chapatties of wheat; rice, maida, suji, maize, jowar, bajra or ragi Breakfast cereal of broken wheat, rice, oatmeal or maize Milk or milk products Soups Vegetable salad Vegetables, cooked Potato, sweet potato, or yam Fruits, fruit juices Sugar, jaggcry or honey Jam or murabba, jellies and other sugar concentrates Biscuits Desserts as light custard or ice-cream Beverages, water (liberal), glucose water	Whole pulses (dal) or beans Red Meat, high fat organ meats Egg Fried Eoods, butter (restricted) Nuts and oilsecds, dry fruits Condiments and spices Papad, chutney or pickles Strong tea or coffee Alcoholic beverages

Next, we move on to the dietary recommendations for liver cirrhosis.

15.3.2 Dietary Recommendations for Liver Cirrhosis

Refer to Table 15.1. A high carbohydrate, moderate protein and low fat diet is advised to a patient with liver cirrhosis along with vitamin and mineral supplementation. Since anorexia is at its peak, the food should be given in several feedings with moderate portions (6-8 feedings). The diet needs progression from liquid, soft to normal diet, depending on the acute stage and recovery. Judicious use of spices and condiments to stimulate appetite is needed. High calorie and protein beverages are useful in between meals. In case of complications of encephalopathy low protein diet needs to be given. Fat also needs to be less and gradually increased as the subject improves. Nutrition care should maintain or improve the nutritional status. Individualized diets must be given depending on the degree of malnutrition and tolerance level of the patient. Counseling the patient on diet and food choices is helpful.

What should be the ideal dietary intake that would help to minimize the symptoms and complications? Let us read and find out.

Proteins: Intake to be adjusted as per the individual requirement, depending on the pathological state. A protein intake high enough to maintain nitrogen balance and low enough to prevent hepatic coma in the initial stages is recommended. In uncomplicated hepatitis or cirrhosis without encephalopathy, a protein requirement 1 g/kg of dry weight/day to achieve nitrogen balance is advised. To promote nitrogen accumulation or positive balance, at least 1.5 to 1.2 g/kg daily is needed. Protein intake is reslricted to 0.5 g/day if there are signs of impending coma. In situations of stress such as alcoholic hepatitis or sepsis; infection, GI bleeding, severe ascites, at least 1.5 g of protein /kg/day should be provided.

Carbohydrates: To ascertain the carbohydrate need is challenging because of deteriorating functional state of liver, which favours preference for alternative fuels. Depending on the state of the liver, the carbohydrate content is kept adequate (300-400 g/day) for its protein sparing effect. It protects and supports the liver function. Adequacy of calories for maintaining weight needs to be emphasized. Emphasis should also be on improving the total intake of the patient. An intake of 25 to 35 Kcal/kg estimated dry body weight should be used in calculations to prevent overfeeding. Intravenous glucose administration must be done only if there is severe nausea and vomiting.

Fats: Steatorrhoea or fatty infiltration of liver may be seen in a cirrhosis patient, thus a moderate intake with the substitution of medium chain triglyceridcs (MCTs) may prove to be effective in reducing malabsorption of fat.

Vitamins: Supplementation with vitamins is desired to replenish liver stores and repair tissue damage especially if the patient has anorexia. This is due to the intimate role of liver in nutrient transport, storage and metabolism, in addition to the side effects of the drugs used. The vitamins of importance are water-soluble vitamins – pyridoxine, cyanocobalamin, folate, niacin and thiamin associated especially with alcoholic liver disease leading to Wernicke's encephalopathy. Deficiency of fat-soluble vitamin has been observed due to malabsorption and decreased storage capacity of diseased liver, Therefore supplementation is necessary using water-soluble forms.

Minerals: Calcium, Magnesium and Zinc are the important minerals as the serum levels tend to decrease in cirrhotics due to malabsorption associated with steatorrhoea. Thus adequate doses as per the requirements should be supplemented.

Sodium: Restriction of sodium is essential if oedema and ascites are present. Sodium restriction up to 500 mg/day is seen with ascites but generally relaxed to 2 g/day with diuretics. An extremely low sodium diet can affect the palatability, as well as, increase risk to hyponatremia,. Emphasis must be on low sodium foods and avoidance of table salt or salt in food preparation. Look up Unit 11, sub-section 11.3.3 for low sodium food items. Also protein intake must be adequate without increasing sodium intake. Serum sodium and potassium levels need to be closely monitored.

Fluids: These may need not be severely limited if sodium restriction is effective in correcting oedema and ascites. No more than 1500 ml of fluid/day is given. Fluid requirement is generally worked out as per the previous day urinary output coupled

with the insensible losses (perspiration, breath, feces etc) which normally amounts to 500 ml/day as well as loses due to diarrhoea or vomitting (if any).

Fibre: Reduction in fibre content is necessary in advanced cirrhosis to prevent danger of haemorrhage from oesophageal varices. Hence, liquid and soft diet and small meals are emphasized.

Considering these dietary guidelines, the foods which may be permitted and which should be excluded from the diet of a patient suffering from cirrhosis is given in Table 15.3. A sample menu for a cirrhotic patient providing roughly 2000 Kcal and 60 g proteins is given in Box 15.2.

Table 15.3: Permitted and excluded foods

Permitted Foods	Excluded Foods
Bread (wheat), rice, maize, jowar, bajra, breakfast cereals, pasta and other refined cereals like maida, suji etc.	Fried foods
Tonned milk and its products like paneer, curd etc.	Organ meat, egg yolk
Washed and split pulses and beans	Whole pulses and fibre rich cereals like oats, barley
Sugar, jaggery, honey, jam or murabba,, jellies	Extra salt and baking soda, preserved foods and foods containing salt like papads, chutneys, pickles etc.
Lean meat, egg white, fish or chicken	
Fat or butter, cream	
Potato, sweet potato or yam	
Pastries, dessert, sweetmeat	
Beverages, lemonade, fruit juices	

Box 15.2 Sample Menu for a Patient Suffering from Liver Cirrhosis

Bed Tea :
Tea/Coffee/Lime Juice/Fruit Juice
Biscuits (2)

Breakfast :
Milk (Skimmed/Tonned) 1 Glass
or Egg white (2 No.)
Porridge (1 bowl) - Cornflakes/Semolina
2 Slice of bread with Jam OR 2 small chapatti with vegetable
fruit (1)

Mid-morning :
Sweet Lassi/ Fruit Juice/Coconut water/Roohafza water/Lime Juice/Soup (1 glass)

Lunch :
Chapatti (2-3) or Rice (1 big bowl - 40 g)
Dal - 1 Katori (Fish/Chicken may be given but not more than twice a week)
Soft vegetable - 1 katsri
Curd (1 katori)
Fruit (1)

Evening Tea :
Milk/Tea/Coffee/Juice
Biscuit/Rusk/Puffed Rice snack/sprouts/tofu etc.

Dinner :
Soup (1 bowl)
Same as Lunch (but avoid meat, chicken etc.)
Custard with Jelly/Kheer/Pudding

For cirrhotic patients with Wilson disease, binding agents also known as chelating agents are often used for removing copper. Also a vegetarian diet which is low in copper is recommended. Table 15.4 gives a list of foods high and low in copper. Inclusion of foods low in copper will help prevent symptoms of Wilson disease.

Table 15.4: Copper content of foods

Foods **High in Copper (to be avoided)**	**Foods Low in Copper (desirable)**
Meat: organ meats, lamb, pork, fish, liver and shellfish	Eggs
Milk: chocolate, cocoa and soyamilk, tofu	All other dairy products
Pulses: Beans Peas and lentils	Bread and pasta from refined flour, rice, sweet potatoes
Cereals: Bran containing cereals, soyaflour	All other vegetables including tomatoes
Vegetables: Mushrooms and juice	All other fruits (including jams, jellies) Lemonade and fruit flavoured beverages
Fruits: Dried fruits, dates, raisins and prunes	
Brewer's yeast	

Now, let us learn about the nutritional management of the last stage of liver disease, which is called hepatic encephalopathy or hepatic coma.

15.3.3 Dietary Recommendations for Hepatic Encephalopathy (HE) or Hepatic Coma

The nutritional management goals for hepatic coma include:

- reduction in protein intake to a minimum in order to decrease amount of ammonia produced,
- correcting plasma amino acid profile, and
- prevention of catabolism of tissue protein.

There is no general treatment. The diet needs to be individualized. The dietary recommendations include:

Calories: A 1500 to 2000 Kcal diet is recommended to prevent breakdown of tissue protein for energy. It is provided chiefly in the form of carbohydrates. It can be given by parenteral or tube feeding if needed. Carbohydrates help to build up liver glycogen reserves and have a protective role in the healing process.

Carbohydrates: An increase in carbohydrates in the diet is recommended because it is the main source of energy and thus spares the protein. It promotes glycogen repletion, which improves with carbohydrate adequacy. It also prevents hypoglycemia.

Proteins: It has not yet been proved that severe protein restriction improves the mental state of the patient in hepatic encephalopathy. Unnecessary protein restriction may only worsen body protein losses and therefore must be avoided. More than 95% of cirrhotic patients can tolerate mixed protein diets .The protein intake may begin with 0.2 g/ kg IBW /day. If the patient remains asymptomatic for a week it may gradually be increased by 10-15 g per week, and then 20-40 g and gradually to 0.5 g/kg IBW per day as indicated in Table 15.1 above,

Research postulates that vegetable proteins and caesin may improve mental status compared to animal protein. Vegetable based diets are lower in AAAs and higher in BCAAs than meat based diets. The potential advantage of vegetable protein is that it is low in methionine and ammoniogenic amino acids. The BCAA are desirable supplements in liver disease. These amino acids are metabolized by the muscles independent of the liver to provide energy, other amino-acids or small nitrogenous compounds and help in obtaining a positive nitrogen balance. Vegetables proteins are rich in BCAA. The common food sources rich in BCAA include dairy products and red meat. Whey protein and egg protein supplements are among the other sources.

Experts agree that BCAA enriched formulas should be indicated for patients with encephalopathy who do not tolerate standard proteins. Both enteral and parenteral BCAA supplement formulas are available commercially and could be used, if required.

An increase in BCAA helps in a number of ways. These include:

- enhances the uptake of AAA by muscles.
- increases protein synthesis in muscles.
- increases hepatic protein synthesis.
- reduces the cerebral AAA levels by competing for a common transport system across the blood brain barrier.

Fats: Fats require restriction, as diseased liver cannot metabolize fats. Substitution with MCT is recommended as they do not require bile salts and micelle formation for absorption and are readily taken up by the portal route.

Vitamins: Increase in intake of B-complex vitamins such as folate, thiamin, B_{12} and vitamin C is recommended as these vitamins act as coenzymes in various metabolic reactions.

Sodium: Depending on the state of the patient, a restriction of 2 g/day along with use of diuretics is recommended.

Fluid: Hyperaldosteronism is associated with liver failure, which results in increased renal sodium exchange for potassium. This urinary potassium loss further gets aggravated by diuretic therapy. There is an evident fluid retention. Thus, depending on the patients' state of hydration, urine output, presence of oedema and diuretic therapy, the fluid intake should be decided and recommended.

With this we end our study on the nutritional management of liver diseases. Try to recall what you have learnt so far by answering the questions given in check your progress exercise **2.**

Check Your Progress Exercise 2

1. What dietary advice would you give to a patient suffering from viral hepatitis?

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2. Which is the last stage of liver disease? Discuss why BCAA are preferred over AAA? Also identify the food sources rich in BCAA.

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3. What is Wilson's disease? List at least five food items that must be avoided.

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4. Mention three foods that you would avoid in liver cirrhosis.

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Having attempted these questions successfully we hope you have understood the nutritional management in liver diseases. Next, we shall review the gall bladder and biliary tract diseases and learn about their nutritional management.

15.4 GALL BLADDER AND BILIARY TRACT DISEASES

Gall Bladder, as you would recall studying in your Applied Physiology Course (MFN-001) in Unit 6, section 6.9, is an organ attached to the right side of the undersurface of the liver. Its main function is to concentrate and store the bile formed in the liver until the body needs to digest fat. At that time, the gall bladder contracts and pushes the bile into a tube, called the common bile duct that carries it to the small intestine, where it helps with digestion.

Bile contains water, cholesterol, fats, bile salts, proteins, and bilirubin. Bile salts break up fat, and bilirubin gives bile and stool a yellowish colour. If the liquid bile contains too much cholesterol, bile salts, or bilirubin, under certain conditions it can harden into stones, The two types of gallstones are *cholesterol* stones and *pigment* stones. Cholesterol stones are usually yellow-green and are made primarily of hardened cholesterol. They account for about 80 percent of gallstones. Pigment stones are small, dark stones made of bilirubin.

The disorders of the biliary tract and gall bladder are closely associated with liver disorders. The common diseases of the biliary tract are *cholelithiasis* , *choledocholithiasis* and *cholecystitis.* Let us get to know what these terms mean.

- *Cholelithiasis*: it is the formation of gallstones in the absence of infection of the gall bladder. These may cause no symptoms and the patient might be unaware of their presence.
- *Choledocholithiasis*: when stones slip into the bile duct producing obstruction, pain and cramps, it is referred to as *choledocholithiasis.*
- *Cholecystitis*: inflammation of gall bladder. It is usually caused by, gallstones obstructing the bile ducts causing a backflow of bile. The walls of the gall bladder become inflamed and distended and infection can occur. During such episodes, the patient experiences upper quadraut abdominal pain accompanied by nausea, vomiting and flatulence. Jaundice can also occur during this disease.

Cholecystitis can be either acute or chronic. Let us get to know about these two phases.

1. *Acute*: This can occur without stones mainly in critically ill patients or when the gall bladder and its bile are stagnant.
2. *Chronic:* This appears to be due to diminished spontaneous contractile activity of the gall bladder and decreased contractile responsiveness to the hormone cholecystokinin.

Having learnt about the common diseases of the billiary tract, let us next review the risk factors associated with gall stone formation, particularly the cholesterol stones. These can be listed as:

- Female Gender
- Pregnancy
- Older **Age**
- Family History
- Obesity
- Trunkal body fat distribution
- Diabetes Mellitus

- Inflammatory Bowel Disease (IBD)
- Drugs like lipid lowering medications, oral contraceptives
- Rapid weight loss through severe calorie restriction – biliary sludge

  Low grade chronic infections
- High dietary fat intake

Other factors include sickle cell anaemia, thalassemia, biliary tract infection, cirrhosis, alcoholism and long term parenteral nutrition.

Next, let us get to know about the treatment and management of gallstones.

Treatment of gallstones

Removal of gall bladder is called *cholecystectomy.* This can be done by the traditional laparotomy or non-invasive laparoscopic procedure. Conservative treatment is by using chemicals to dissolve the stones. Currently the shock wave lithotripsy is also being used extensively. After the removal of the gall bladder, the biliary tract dilates forming a simulated pouch over time to allow bile to be held in a manner similar to the original gall bladder. We shall not dwell any further on the medical treatment of gallstones, since our focus here is on the nutritional management which is described next.

Nutritional *Management*

The dietary considerations for management of gall bladder stones are enumerated herewith:

- The main aim of the treatment is to reduce discomfort by providing a diet restricted in fat. In an acute condition it is advisable to keep the gall bladder at rest and minimize contractions by excluding fat from the diet. In an acute attack a 'nil by mouth diet', followed by an extremely low fat intake to prevent gall bladder stimulation is recommended. Fat content is gradually increased by providing 2 hourly liquid diet for a few days. In chronic phase 20-30 g of fat may be provided and later increased gradually to 40-45 g thus increasing the palatability of the diet. After the symptoms settle clear soups, toned milk, refined cereals could be given. The patient is put on a 30-45 g of fat intake per day, which can be achieved by incorporating a variety of low fat options.
- A calorie-restricted diet with a restriction on the intake of refined carbohydrates is beneficial. Refined sugars increase cliolesterol saturation and lithogenicity of the bile. Control on fat intake also contributes to weight loss due to calorie restriction,
- A moderate fat restriction can be considered. Around 25% of total calories can come from fat with a prudent usage of Medium Chain Triglycerides and good quality fat (high MUFA and low N6: N3 ratio) from invisible sources. Use of MUFA has shown a powerful effect on gall bladder emptying. This is because monounsaturated fats increase the ratio of HDL cholesterol to LDL cholesterol and it may therefore provide important protection against gallstone formation.
- An appropriate selection of low fat options like toned/ skim milk, paneer, low fat meat options, fish and egg in restricted quantity may help in getting the good quality proteins. Studies reveal that animal proteins in the diets are more lithogenic (stone forming) as compared to vegetable proteins.
- A high intake of soluble fibre through pulses/ sprouts/ dals, vegetables, fruits and oilseeds (flax seeds and methi seeds) can help the body to get rid of bile acid sterols out of the gut. This also gives a desirable micro flora to favour inhibiting endogenous production of cholesterol. It also provides less time for colonic bacteria to produce secondary bile acids like deoxycholic acid from cholic acid, hence less deoxycholic acid is absorbed.

- The degree of food intolerance needs to be individualized, as it may vary from patient to patient.
- The distribution of meals throughout the day may be of use to give symptomatic relief and better tolerance to food.
- Administration of water-soluble forms of fat-soluble vitamins may be of benefit in chronic gall bladder disease patients, as fat malabsorption is suspected.
- Vitamin C is beneficial as it decreases the incidence of cholelithiasis.
- Coffee consumption has shown to be having a protective effect on gallstones formation as coffee and various constituents of coffee affect various metabolic processes that are involved in cholesterol lithogenicity. Coffee stimulates cholecystokinin release, increases gall bladder motility and possibly enhances large bowel motility. Caffiene inhibits biliary cholesterol crystallization, decreases gall bladder fluid absorption and increases hepatic bile flow.

Table 15.5 provides a list of foods permitted and excluded from the diet of a patient suffering from gall bladder disease.

Table 15.5: Food items for a patient with gall bladder disease

Permitted	Excluded
Bread or chapatties from wheat, maize, jowar, bajra or ragi	Fried foods
Breakfast cereal of wheat, rice, oalmeal or maize	Fatty meats
Rice, cooked	Fruits, dried
Pulses (dal) or beans as thin dal, 1 cup	Nuts
Fish or chicken	Sweets or sweetmeats
Egg (if no discomfort)	Condiments and spices
Milk or milk products (with cream removed)	Papad, chutney or pickles
Soup (thin soups)	Thick soups and gravies whole milk/khoya
Vegetable salad	
Vegetables, cooked	
Potato, sweet potato, or yam	
Fat for cooking or butter if no symptoms	
Sugar, jaggery or honey	
Fruits, fresh	
Fluid liberal	
Jam or murabba	
Pastry only as biscuits or light cakes	
Desserts as light custard, jelly ice-cream	
Beverages	

Now let us check our understanding of the gall bladder and billiary duct diseases and their nutritional management by answering the questions included in the check your progress exercise 3.

Check Your Progress Exercise 3

1. What is the main function of gall bladder? List the common disorders of the biliary tract.

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2. Enumerate the risk factors involved in the formation of cholesterol stones.

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3. Discuss the dietary considerations in the management of gallstones.

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4. Kumar is a patient suffering from gallstones. Would you suggest consumption of coffee to him? Why?

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Let us move on further and understand the details of pancreatic disorders.

15.5 PANCREATIC DISEASES

The pancreas is located deep in the upper abdomen behind the stomach. Look up Unit 6, Figure 6.10 in the Applied Physiology Course to view the pancreas. What functions does the pancreas perform in our body? Yes, the main functions of pancreas include insulin manufacture and secretion of enzymes that participate in the digestion of various nutrients. One of the conditions linked to the abnormal functioning of pancreas is pancreatitis.

What is pancreatitis ?

Pancreatitis relates to the inflammation of the pancreas and is characterized by oedema, cellular exudates and fat necrosis. The disease can range from mild and self-limiting to severe condition which results in auto digestion, necrosis and haemorrhage of pancreatic tissue. Pancreatitis can be classified as acute and chronic. Let us read and find out what are these, how do they differ and what is their dietary management. But first we need to understand that pancreatitis is closely linked with liver and gall bladder diseases. What is the relationship? Let us find out.

Bile, we learnt above, is synthesized by the liver, stored in the gall bladder and secreted in the intestine. It has a distinct role to play in fat metabolism and decreased production and secretion in the intestine can affect fat digestion and lead to fat malabsorption. A building up of back up bile pressure due to renal or post renal causes can lead to precipitation of jaundice and can be a cause of secondary biliary cirrhosis, cholelithiasis. Obstruction of distal common bile duct can lead to pancreatitis if the pancreatic duct is blocked, Thus, liver, gall bladder and pancreatic diseases can have an overlapping etiology.

Now, with this basic understanding let us get to know about acute pancreatitis.

15.5.1 Acute Pancreatitis

The pancreas is protected against its own enzyme by their synthesis as proenzymes. Acute pancreatitis develops when activated pancreatic enzymes (activated prematurely in pancreas) are liberated within the pancreatic system.

The clinical feature of pancreatitis results from auto digestion of tissue and toxic effects of digestion products. Elevated serum and urinary amylase concentration due to enzyme released by necrosed pancreatic cells remain one of the main diagnostic criteria in acute pancreatitis. The severity of pancreatitis can be seen by the Ranson's criteria to classify pancreatitis. We shall not go into the details of this criteria, since it is not within the purview of this unit. You may refer to the practical manual (005) for details. We will however move on to the etiology of pancreatitis.

Etiology

The etiological factors involved in the disease are biliary tract disease, such as gallstones, alcohol abuse, trauma and hyperlipidemia {rarely).

Let us study what are its symptoms and complications.

Symptoms

The symptoms of pancreatilis include:

- Continuqus or intennittent pain of varying intensity in the upper abdominal region that radiates to the back. Symptoms worsen with ingestion of food.
- Swollen and tender abdomen.
- Nausea and vomiting – precipitated by large meal and alcohol consumption.
- Steatorrhoea and malabsorption.
- Sweating, fever,. mild jaundice and rapid pulse are also seen.

Complications

Some of its complications include low blood pressure, heart failure, kidney failure, diabetes, ascites and cysts in pancreas.

Now then what can we do to manage this problem? The nutritional-management is highlighted next.

Nutritional Management

Acute pancreatitis often results in a catabolic state characterized by profound haemodynamic, metabolic, cardiovascular, pulmonary, haematological and renal aberrations. Parenteral nutrition and metabolic support becomes essential in order to minimize mortality. Since TPN feeding needs specialized set ups, increased costs and long term maintenance, it is difficult for patients to afford the treatment. Hence, enteral nutrition is a preffered mode. This type of feeding is safe, well tolerated with less infections/non-infectious complications. The nutritional management goals of acute pancreatitis include:

1. Conservative management involves resting pancreas and maintaining fluid balance.
2. Nil by mouth till the pain and fever subsides, as the oral intake further aggravates the symptoms caused by an increased secretory mechanism of pancreatic enzyme and bile.
3. The patient needs to be supported by early enteral nutrition with the formulation of nutrient in predigested forms and supplementation with low fat intake to prevent further precipitation of malnutrition. Sometimes TPN is required.
4. When oral feeding is resorted it should be a clear liquid diet with a waiting period to see the response of the patient in terms of undesirable symptoms.
5. A low fat diet with an intake of total fat as 30 g/day, which may gradually be increased as per the patient's tolerance. MCT may be incorporated for better

digestibility and assimilation, as they do not require the pancreatic enzyme system for the same. They also help in increasing the total caloric intake of the patient.

6. A careful monitoring of all the biochemical parameters starting from enzymic assay to serum albumin concentration should be done on regular basis.

7. Decreased calcium levels are observed during acute pancreatitis. This can be due to (i) hypoalbuminemia (as calcium is bound to protein), and (ii) soap formation of calcium with fatty acids created by fat necrosis. Hence calcium supplementation may be required.

Next, we move on to chronic pancreatitis.

15.5.2 Chronic Pancreatitis

Chronic pancreatitis mainly results following the repeated attacks of acute pancreatitis or the effect of digestive enzymes on pancreas or may be associated with chronic inflammation of the biliary tract. There is also a strong relationship of alcohol abuse and development of chronic pancreatitis as it acts as an intestinal irritant and leads to reoccurrences.

What causes this chronic condition? Let us find out.

Etiology

The etiological factors include:

- Neglect of acute pancreatitis
- Alcohol abuse
- Excessive iron in the blood
- Unknown factors

The symptoms of this acute condition are highlighted next.

Symptoms

Common symptoms include:

1. Pain
2. Malabsorption
3. Weight loss
4. Malnutrition (could be due to alcohol abuse).
5. Steatorrhoea

Finally let us get to know about the nutritional management of chronic pancreatitis.

Nutritional Management of Chronic Pancreatitis

The nutritional management goals for chronic pancreatitis include:

- Rest to pancreas
- Prevention of diabetes
- Enteral supplementation
- Diet control and special feeding

The nutritional management ranges from fundamental dietary modifications to administration of appropriate digestive enzymes to enteral supplementation. It however, depends on the stage, severity and manifestations of pancreatitis. Pancreatic enzyme supplementation is important in long term patient management and it helps to control

and reduce malabsorption. At times appropriate digestive enzymes help and other times it needs special feeding methods to be employed especially enteral feeding.

In chronic cases with extensive pancreatic destruction, the insulin secretory capacity of the pancreas decrease and glucose intolerance develops. Treatment with insulin and nutritional care is similar to diabetes.

The dietary guidelines include:

- A caloric intake of 35 Kcal/kg IBW is ascertained keeping in mind tlie'moderate stressful state. The patient needs to be kept on a low fat diet (40 to 60 g/day), the levels are ascertained with the tolerance of the patient.
- Calcium and vitamin B_{12} are important as deficiency of pancreatic protease may prevent cleavingof vitamin B_{12} from its carrier protein there by leading to vitamin B_{12} deficiency.
- Fat-soluble vitamin malabsorption may occur. Thus, parenteral administration of fat-soluble vitamin is necessary.

In the end, let us remember some important guidelines for managing chronic pancreatitis. These include:

- Avoid alcohol
- Avoid meal with a high fat content. Use MCT's in severe steatorrhoea
- Give adequate energy and protein intake
- Monitor blood glucose levels regularly
- Give vitamin and mineral supplementation (fat soluble vitamins, folic acid and calcium)
- Pancreatic enzyme supplementation should be taken adjusted to quantity of food and fat content of meals)

We shall learn the details of dietary management in the practical manual. However, you may now attempt the questions mentioned below to check you understanding of the concepts discussed above in section 15.5.

Check Your Progress Exercise 4

1. What do you understand by the term 'pancreatitis? List its characteristic features.

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2. Discuss the dietary management of a patient suffering from acute pancreatitis,

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3. What are the symptoms of chronic pancreatitis? List a few dietary guidelines for maintaining such a disease condition.

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4. Why do you think a chronic pancreatitis patient is at risk of developing diabetes?

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15.6 LET US SUM UP

In this unit, we learnt about the important liver, pancreatic and gall bladder disorders such as hepatitis, cirrhosis, pancreatitis, gall bladder disorders (stones), hepatic encephalopathy or coma. To have a better understanding of the diseases involved, wc first reviewed our knowledge about the functions of these organs.

Next, we studied about thcse disorders separately in a greater detail, discussing their etiology, symptoms, associated complications and clinical manifestations.The nutritional aspects of these disorders and their corresponding dietary management were also emphasized. The discussion also included the types of foods to be included, as well as, excluded, a few dietary tips to remember and some sample diets.

15.7 GLOSSARY

Atresia : inadequate development of an organ or a part of organ during pregnancy.

Cholangitis : an inflammation of the bile duct; can be acute as an outcome of infection or liver failure.

Cholestasis : a condition of sludge like buildup in the gall bladder as a result of lack of stimulation or release of bile.

Chorea : rapid jerky, dance-like movements of the body especially extremities and head.

Cystic fibrosis : an inherited autosomal recessive condition that causes the secretion of abnormal mucus in the lungs and problems with pancreas function and food absorption.

Dystonic facies : the appearance or expression of the face, especially when typical of a certain disorder or disease.

Fecal-oral route : Many diseases can be passed when the stool (or remnants thereof) of one host ends up in someone else's mouth. This is referred to as the fecal-oral route (or alternately, the oral-fecal route or oro-fecal route).

Patty Liver : a condition characterized by the accumulation of excess fat in the liver commonly caused by alcohol abuse but also associated with obesity, starvation, intestinal bypass etc.

Fulminate liver disease : absence of pre-existing liver disease with development of hepatic encephalopathy within 2 months of onset of illness.

Hemochromatosis : a disorder of iron metabolism characterized by excessive absorption of ingested iron, saturation of iron-binding protein and deposition of haemosiderin in tissue, liver cirrhosis, diabetes, pigmentation of the skin and eventually heart failure.

Hepatic coma	:	coma (a state of unrousable unconsciousness) that can occur in severe cases of liver disease.
Neurosis	:	a disease of the nerves without any appreciable change of nerve structure leading to a mental or personality disturbances.
Portal hypertension	:	high blood pressure in the portal vein that carries blood to the liver.
Psychosis	:	a major mental disorder that affects the ability to function normally on a daily basis.
Thrombocytopenia	:	an abnormally low number of thrombocytes resulting in abnormal bleeding and bruising.
Wernicke's Encephalopathy	:	an inflammatory degenerative disease of the brain caused by thiamin deficiency that is usually associated with alcoholism.

15.8 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check **Your** Progress **Exercise 1**

1. The liver plays an important role in fat, carbohydrate, protein functions, vitamin and mineral metabolism. In fat metabolism it relates to the synthesis of triglycerides and phospholipids. Liver synthesizes lipoproteins, cholesterol and converts 80% into bile salts and the remainder is transported in the form of lipoproteins. It is also involved in the oxidation of fatty acids to acetyl CoA and synthesis of bile and conjugation of bile salts.

2. Jaundice is one symptom common to all liver diseases. It consists of yellow pigmentation of the skin and body tissues because of accumulation of bile pigments, in the blood. It results due to an increase in bilirubin content of the blood above the normal range (0.2 to 0.8 mg/100 ml plasma).

3. Cirrhosis is the final stage of liver injury and degeneration. Cirrhosis refers to all forms of chronic *diffuse* liver *disease* characterized by significant loss of functional liver cells (necrosis), nodular regeneration of new tissue that limits liver functioning by interfering with blood flow due to distortion of vascular bed.

   The various etiological factors include chronic alcoholism in association with malnutrition, underlying metabolic disturbances, hepatotoxins (virus, drug etc), prolonged biliary stasis, cystic fibrosis and chronic hepatitis

4. It involves metabolic derangements caused by toxic effects on mitochondria1 function produced by acetaldehyde and hydrogen. Acetaldehyde, a toxic by product of alcohol metabolism, causes damage to mitrochondrial membrane structure and function. Ethanol is metabolized primarily in the liver by alcohol dehydrogenase (ADH). This results in acetaldehyde production with the transfer of hydrogen to nicotinamide adenine dinucleotide (NAD), reducing it to NADH. The acetaldehyde then loses hydrogen and is converted to acetate, most of which is released into the blood. These include hyperlactic acidemia, acidosis, hyperuricemia, ketonemia, and hyperlipidmia.

Check Your Progress Exercise 2

1. An increased carbohydrate, protein and moderate fat diet are advised in the case of viral hepatitis. Supplementation of B complex vitamins, may be given, as they have an important role in carbohydrate and protein metabolism. Sodium restriction is required only if there is fluid retention. Potassium supplements are necessary with diuretic therapy. lron supplementation is needed only if there is anaemia.

2. Hepatic coma or hepatic encephalopathy is the last stage of liver disease. Caesin-based diets are lower in AAAs and higher in BCAAs than meat based diets. The potential advantage of vegetable protein is that it is low in methionine and ammoniagenic amino acids and it is BCCA rich. The common food sources of BCAA include dairy products, red meat, whey protein and egg protein supplements.

3. Wilson Disease is a rare autosomal recessive, inherited disorder characterized by an abnormal copper transport and storage mechanisms resulting in an excessive copper deposition in body tissues. High Copper Foods (20.2 mg/portion) include organ meats, lamb, pork, fish, liver, shellfish, turkey, chocolate, cocoa, broccoli, beans, pulses, lentils, soya, millets, bran products, mushrooms, dried fruits such as raisins dates, prunes and brewer's yeast.

4. Foods to be avoided in liver cirrhosis are organ meat, fried foods and pickles, chutney, papad.

Check Your Progress Exercise 3

1. The main functions of the gall bladder include concentration and storage of bile, which is secreted by the liver. The common disorders of the billiary tract are cholelithiasis, choledocholithiasis and cholecystitis.

2. The risk factors include female gender, pregnancy, family history, obesity, truncal body fat distribution, diabetes mellitus, inflammatory bowel disease (IBD), rapid weight loss through severe calorie restriction – biliary sludge, low grade chronic infections – changes in gall bladder mucosa – excess bile acid absorption etc.

3. The dietary considerations in the management of gallstones are given in section 15.4. Read the considerations carefully and answer accordingly.

4. Coffee consumption has shown to be having a protective effect on gallstones formation. Coffee stimulates cholecystokinin release, increases gall bladder motility and possibly enhances large bowel motility. Caffiene inhibits biliary cholesterol crystallization, decreases gall bladder fluid absorption and increases hepatic bile flow.

Cheek Your Progress Exercise 4

1. Pancreatitis refers to the inflammation of the pancreas and is characterized by oedema, cellular exudates and fat necrosis.

2. Dietary management of a patient suffering from acute pancreatitis is given in sub-section 15.5.1. Read carefully and answer on your own.

3. The salient features are pain, malabsorption, weight loss/malnutrition due to alcohol abuse and steatorrhoea.

   The nutritional management ranges from fundamental dietary modification to administration of appropriate digestive enzymes to enteral supplementation, depending on the stage, severity and manifestation of the disease. Pancreatic enzyme supplementation is important in long term patient management and helps in two ways by assisting in pain control and reducing malabsorption.

4. In chronic cases with extensive pancreatic destruction, the insulin secretory capacity of pancreas decrease and glucose intolerance develops. Treatment with insulin and nutritional care similar to diabetes is required.