IJBPAS, September, 2026, 15(9): 4545-4557

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

CIRRHOSIS AND MALNUTRITION: THERAPEUTIC ROLE OF VITAMINS IN LIVER REGENERATION AND FUNCTION

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Received 14th Nov. 2025; Revised 19th Dec. 2025; Accepted 16th April 2026; Available online 1st Sept. 2026

https://doi.org/10.31032/IJBPAS/2026/15.9.10496

ABSTRACT

Liver is the primary organ for the metabolism, storage and detoxification of various nutrients and toxins. Cirrhosis is the replacement of healthy hepatic tissue by connective tissue, leading to necrosis of hepatic cells. Cirrhosis is the last stage of liver injury. It is one of the top leading causes of morbidity and mortality, primarily associated with chronic alcoholism, viral hepatitis, obesity, smoking, and metabolic syndromes. Initially the disease is asymptomatic, but with progression, symptoms like loss of appetite, nausea, weakness, fever, and weight loss appears. During cirrhosis, the liver is unable to metabolize nutrients, particularly vitamins. Deficiency of both fat-soluble and water-soluble vitamins is recorded due to reduced bile production, impaired absorption, and inadequate intake. This review emphasizes the importance of nutritional therapy and targeted vitamin supplementation as key components in the comprehensive management of patients with liver cirrhosis.

Keywords: Cirrhosis; liver disease; Hepatic metabolism; Vitamin deficiency; Fat-soluble vitamins; Water soluble vitamins; Nutritional management; Hepatic regeneration

INTRODUCTION

The term cirrhosis is derived from 'ethos', a Greek word, which means tawny and describes the tan-coloured liver. Cirrhosis is classified based on the three pathophysiological features, namely the presence or absence of liver disease, liver fibrosis, and replacement of hepatic cells with scar tissue. The initiation of cirrhosis begins with fibrous deposition along with distorted blood supply in the hepatic cells **[1, 2]**. In response to chronic liver disease or injury, the development of fibrous bands surrounding regenerative nodules of hepatic cells takes place, leading to portal hypertension and end stage liver disease or cirrhosis **[2]**.

In developed countries, increasing causes of morbidity and mortality are one of the most common reasons for cirrhosis. Cirrhosis is the 11th most common cause of deaths worldwide and 4th most common cause of deaths in Europe **[3]**. Worldwide death from cirrhosis increased from 777,800 in 1990 to 1,030,850 in 2010. It is more common among males of age between 15-49 years **[4]**. In 2017, the global estimate of deaths from cirrhosis was 1.32 million (24 percent of total deaths), among which 883,000 (66.7 percent) were male, while 440,000 (33.3 percent) were females. While in 2014, the number of reported deaths in India because of cirrhosis were 216,865, contributing about 2.44 percent of the total deaths **[5]**.

Based on the size of the nodules, cirrhosis is classified into 3 types, micronodular, macronodular, and mixed type. In micronodular Laennec's or portal cirrhosis, uniform nodules of size less than 3 mm in diameter can be seen, resulting from chronic alcoholism, obstruction in the outflow of hepatic vein, and chronic biliary obstruction. While the nodules in the macronodular form are irregular in size, and are of size more than 3 mm, caused because of hepatitis B, C, primary biliary cholangitis, and alpha-1 antitrypsin deficiency. On the other hand, mixed types have clinical and physiological features of both micro and macronodular forms **[5, 6]**. Cirrhosis is the last stage of the liver injury, necrosis of hepatic cells takes place, resulting in permanent degenerative changes in the histopathology of the liver **[2]**.

Etiology of Liver Cirrhosis

More than 90% of the total cirrhosis cases are due to alcoholism, hepatitis B, and C infections. It is highly prevalent among smokers and overweight people **[7]**. Recent studies have shown that iron and copper toxicity also contributes to cirrhosis **[3]**. The most common cause of cirrhosis includes alcohol abuse/chronic alcoholism, chronic

viral infections including hepatitis B infection, and non-alcoholic steatohepatitis (fatty liver caused because of diabetes and obesity) **[8]**. Other causes of cirrhosis includes inherited diseases like Alpha antitrypsin deficiency (build-up of an abnormally high levels of protein in the liver), Hemochromatosis (excess iron stores in the liver), Cystic fibrosis (sticky, thick mucus builds up in the liver), Autoimmune hepatis (body's immune system attacks hepatic cells, causing damage), Wilson's disease (excess copper stores in the liver), Glycogen storage disease, Alagille syndrome (congenital disease, child is born with lesser number of bile ducts than normal, affecting bile flow and leading to jaundice), diseases that damage or block bile ducts in the liver, Biliary atresia (a child is born with either deformed or blocked bile ducts) **[9, 10]**. Changes from liver diseases that lead to cirrhosis are gradual. Over a period of prolonged time, scar tissue replaces the damaged liver cells, and the liver can't function properly, thus leading to cirrhosis **[11]**.

Signs and Symptoms

The symptoms of cirrhosis depend on the stage of the disease. In the beginning stages, the disease is generally asymptomatic **[12]**. Early symptoms and signs of cirrhosis include loss of appetite, weakness, nausea, fever, and unexpected weight loss **[13]**. As the disease progresses, liver function gets declined and body starts showing various signs and symptoms, including bruising and bleeding, yellowing of skin and the sclera of eyes, itchy skin, swollen legs, feet and ankles, oedema, dark coloured urine, light coloured stools, confusion, difficulty thinking, memory loss, personality changes, blood in stool, telangiectasias (spider-like blood vessels), loss of sex drive, enlarged breasts, shrunken testicles in man, and premature menopause in woman **[14]**.

Role of Liver in Nutrition and Metabolism

The primary role of the liver is the synthesis of macronutrients, metabolism, storage, and detoxification. Liver is the primary storehouse of various macro as well as micro-nutrients namely, glycogen, fatty acids, vitamin A, iron, copper, manganese, Vitamin D, and B12. With changes in the normal hepatic cells, there occur a wide variety of derangements in the liver function. After skeletal muscles, liver is the second largest storage site for glycogen. Cirrhosis leads to decline in the storage capacity of the liver cells, resulting in the derangement of glucose homeostasis and insulin resistance. More than 90% of the plasma proteins and 15% of the total protein mass are produced in the liver. Liver also produces several cytokines and chemokines

and serve as primary site for the metabolism of fatty acids and triglyceride synthesis **[15]**.

Liver is the primary organ to produce energy because of its role in the storage and metabolism of fatty acids as well as carbohydrates. During the conditions of starvation/fasting when glucose/glycogen stores are limited, fatty acid breakdown provides an alternate source of energy by switching to gluconeogenesis **[16]**. Liver also helps in the absorption and assimilation of fats and fat-soluble vitamins by producing and excreting bile salts. Liver is the primary site for the detoxification of various toxic metabolites and substances **[17, 18]**. The structural as well as functional integrity of the liver is essential not only for the metabolism of various nutrients, but also for supply and inter-organ trafficking of various nutrients in the body **[19]**.

Many factors contribute to the disruption of metabolic homeostasis in liver, including declined hepatic and skeletal muscle glycogen synthesis along with an elevation in the lipolysis and protein catabolism. The pathogenesis of cirrhosis and malnutrition are corelated, as restricted food intake because of dietary restrictions as well as anorexia, impaired intestinal nutrient absorption, reduced protein intake and increased protein loss, abnormality in the catabolism of carbohydrates, protein, and fat metabolism, irregularities in the nutrient uptake and use, and elevated levels of pro-inflammatory cytokines, all lead to hypermetabolic state, thus contributing to disruption in the liver metabolism and malnourishment **[20]**.

Malnourishment in Cirrhosis

Malnutrition is very common in cirrhosis, depending upon the severity of the liver disease and nutritional assessment method used, the prevalence rate of malnourishment in cirrhosis ranges from 65- 100% cases **[21]**. The criteria for the assessment of malnourishment in cirrhosis patients include, defining loss in muscle mass or muscle strength along with the decrease in visceral fat mass and decreased subcutaneous fat mass **[18]**. Causes of malnutrition in liver cirrhosis includes diminished/reduced dietary intake of nutrients, inadequate absorption and assimilation of macro as well as micro-nutrients **[22]**. Ascites in patients cause impaired stomach expansion and impaired gastric accommodation causes early satiety, causing decline in the nutrient intake **[23]**.

Cirrhosis and Obesity

Obesity is defined as BMI 30 kg/m^2 or more. Obesity is considered as one of the most potential aetiological factors for the progression of liver diseases including cirrhosis. NAFLD (Non-alcoholic fatty liver

diseases) are linked directly with obesity. NAFLD or Non-alcoholic fatty liver diseases are a class of diseases ranging from benign steatosis to NASH or non-alcoholic steatohepatitis, all contributing to advanced fibrosis, sometimes cirrhosis as well as nutrient deficiency leading to obesity. With the increase in the incidences of obesity, there is an expected incline in the number of cirrhosis cases due to NAFLD **[24]**.

The prevalence rate of NAFLD in lean person is 16.5%, while is 75% in obese people. NAFLD is prevalent in all age group and is strongly associated with obesity. With age, the prevalence of NAFLD increases. The highest prevalence rate is found in the age group of 40-49 years **[25]**.

Nutritional Management

The aim of the nutritional management in patients with cirrhosis is hepatic regeneration, prevent or correct the complications associated with cirrhosis, and correct or prevent malnourishment. From studies it has been found that nutritional management or diet therapy in cirrhosis aided in the increased survival rate, improved liver function, surgical outcomes and helped against the complications of cirrhosis **[26]**.

Vitamins

Hepatic dysfunction and decline in the level of stored nutrients leads to various deficiencies, particularly of vitamins and minerals. The stores deplete even more with malabsorption, inadequate dietary intake, as well as with the progression of the disease. Stores of fat-soluble vitamin are more affected, than water soluble vitamins **[27]**. Nutritional deficiencies are very prevalent in cirrhosis, because of the decline in bile production. Low bile production leads to decrease in the bile salt concentration. Decreased bile salt concentration leads to reduced uptake of vitamins, particularly fat-soluble vitamins **[28]**. So proper administration is to be done. Deficiency of water-soluble vitamins is generally seen in end stage liver diseases. While deficiency of fat-soluble vitamin is a result of malabsorption **[29]**.

Fat Soluble Vitamins

Vitamin A

Vitamin A or retinol, deficiency is associated with night-blindness and is very common in patients with primary biliary cirrhosis **[27]**. Vitamin A is stored in the hepatic cells. Vitamin A deficiency is reported in 50% of the alcoholic cirrhosis cases **[30]**. Liver related death is associated with serum retinol levels below ≤0.78 μmol/L, also high levels of retinol have an hepato-toxic effect **[31]**. Low levels of vitamin A results in dry cornea and nyctalopia, supplementation of 100,000-200,000 IU every four weeks is advised **[22]**.

Vitamin D

From several studies, a close corelation has been found in vitamin D deficiency and liver diseases (both cholestatic, and non-cholestatic) **[27]**. The process of 25-hydroxylation of vitamin D takes place in the hepatic cells. Chronic liver diseases, particularly because of chronic alcoholism results in vitamin D deficiency. Low vitamin D level in chronic liver disease patients is associated with increased mortality **[31]**. Osteoporosis is one of the most common complications of end stage liver disease, particularly in people of older age, history of fractures and smoking. Supplementation of 400-800 IU vitamin D is advised along with 1200-1500 mg calcium **[22]**.

Vitamin E

Vitamin E reduces the membrane injury precipitated by reactive oxygen species (ROS), thus acting as an antioxidant, particularly in NAFLD **[32]**. From a meta-analysis conducted, it was found that vitamin E acts as chain breaker in the chained reactions like lipid peroxidation. Vitamin E protects against liver fibrosis caused because of cytokine TGF-β1 (Transforming growth factor beta 1) **[33]**. From studies, it was concluded that vitamin E helps in improving hepatic cell integrity by reducing response to membrane transporter in the uptake of fatty acid **[34]**.

Vitamin K

Vitamin K helps in the production of factor II, VII, IX, and X. It acts as a cofactor in the gamma-carboxylation of glutamic acid residue, resulting in the production of co-factors **[28]**. During cirrhosis, vitamin K supplementation is prescribed because of the increased PT (prothrombin time) along with the presence of oesophageal varices. Increased PT can lead to nosebleed and can worsen the situation. Vitamin K is administered via parenteral route. A dosage of 10 mg vitamin K is administered every 4 weeks **[35]**.

Water Soluble Vitamins

Vitamin B1

Vitamin B1 or Thiamine deficiency is prevalent in cirrhosis, particularly alcoholic liver diseases, the reason being decreased hepatic storage, inadequate dietary intake, and impaired intestinal absorption in the presence of ethanol **[36]**. Ethanol not only impairs intestinal absorption of vitamin but also impairs the transformation of inactive form of vitamin to its active form **[37]**. Vitamin B1 supplementation is recommended in subjects with ESLD (end stage liver disease), irrespective of the cause **[31]**.

Vitamin B2

Vitamin B2 or Riboflavin is a cofactor used in the energy metabolism as well as in antioxidant responses. Vitamin B2 deficiency is present in both alcoholic as well as non-alcoholic cirrhosis. The reason being abnormal vitamin metabolism, inadequate intake, malabsorption or improper storage, and increased utilization **[31]**.

Vitamin B3

Vitamin B3 or Niacin is the precursor of the coenzyme namely, nicotinamide adenine dinucleotide phosphate (NADPH) and nicotinamide adenine dinucleotide. NADPH plays a crucial role in the lipid metabolism. It is used in the treatment of CVDs and dyslipidaemia **[34]**. From studies it has been found that treating dyslipidaemia patients with niacin helped in reducing hepatic fat content, reduced plasma triglyceride levels and helped lower liver enzymes **[38]**.

Vitamin B6, And B9

Vitamin B6 or pyridoxine, and B9 or folate, deficiency is commonly seen in chronic liver diseases, especially cirrhosis. The reason being reduced hepatic storage. From study, it was found that alcoholic liver cirrhosis patients have declined pyridoxine levels along with increased cystathionine and declined alpha-aminobutyrate- cystathionin ratio. From study it was found that the plasma folate levels decrease in chronic liver diseases **[31]**.

Vitamin B12

Vitamin B12 or cobalamin helps in DNA synthesis and repair. It acts as a cofactor for the enzyme namely methyl malonyl CoA mutase, thus playing an important role in the regulation of lipid metabolism **[39]**. Vitamin B12 is stored in liver, and low serum levels during cirrhosis hampers lipid metabolism, and a marked increase in the serum ALT (Alanine aminotransferase) levels **[34]**. It also acts as a cofactor for the metabolism of homocysteine to methionine. During alcohol abuse, the metabolism of homocysteine is altered, and is related positively with alcohol related liver disease markers **[40]**.

Vitamin C

Cirrhosis patients have clinical signs of intra-hepatic endothelial dysfunction, which is characterised by flow dependent impaired vasorelaxation. The impaired vasodilation is responsible for marked elevation in the postprandial portal pressure, attributing to decline in the release of NO (Nitric oxide). From a study, it was found that Vitamin C or Ascorbic acid increases the bioavailability of NO through the neutralization of superoxide anions, which prevents the NO from the scavenging action of superoxide, thus reversing/ slowing the progression of endothelial dysfunction **[41]**. From another study, a 12-week Vitamin C supplementation

at 1,000 mg/day in patients with NAFLD, it was found that there was an improved glucose metabolism as well as liver function **[42]**.

CONCLUSION

Cirrhosis derived from Greek word 'ethos' meaning "tawny" is an irreversible end stage of chronic liver disease marked by fibrosis and necrosis of hepatic cells. Its prevalence is attributed to the increased incidences of alcoholism, viral hepatitis and obesity. Cirrhosis not only impairs the hepatic metabolism but hampers nutrient storage and detoxification done by liver, leading to malnutrition. Nutritional therapy plays a vital role in improving liver function, promoting hepatic regeneration, and managing complications. Supplementation of both fat soluble (A, D, E, K) and water soluble (B-complex, C) is essential, but careful monitoring should be done to avoid toxicity, especially of fat-soluble vitamins. Managing adequate nutrient deficiencies, calorie intake, and addressing metabolic imbalances can enhance survival, improve the quality of life, and slow down the disease progression. A well-structured nutritional intervention, integrated with medical treatment, remains a cornerstone in the management of cirrhosis.

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