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Liver cirrhosis: The struggling liver

Dr. Garima Singh
Assistant Professor, Nutrition and Health Department, GD Goenka University, Gurugram, Delhi

Suman Bala
Suman Bala, Assistant Professor, Department of Home Science, Kurukshetra University, Kurukshetra, Haryana, India.

Sonal Katoch
Nutrition and Health Department, GD Goenka University, Gurugram, Delhi

lakhvinder kaur
Associate professor, Manav rachna international institute of research and studies
Email: Lakhvinder.fahs@mriu.edu.in

Anil Kumar
Ex Research Scholar, Department of Botany, DDU Gorakhpur University Gorakhpur-273009
Email: Email-akdwivedinutra25@gmail.com

Abhishek Kumar
Faculty of pharmaceutical sciences, Rama University, Kanpur, U.P, India
Email: Abhiworld1997@gmail.com

Alok Bharadwaj
Assistant Professor, Department of Biotechnology, GLA University, Mathura (U P.)-281406

Ardhariksa Zukhruf Kurniullah
Faculty of Communications Science, Mercu Buana University, Jakarta, Indonesia
Email: ardhariksa.zukhruf@mercubuana.ac.id

***Abstract*---**Liver cirrhosis is a chronic liver infection. It consist of deterioration of liver cells along with fibrosis and infection generating nodules. Patients with cirrhosis frequently have either global malnutrition or alterations in specific aspects of nutritional status, such as micronutrient deficiencies, due to multiple mechanisms, including poor nutritional intake, poor absorption, and increased losses. In addition, one of the most significant nutritional problems in cirrhotic patients is muscle wasting and sarcopenia. Cirrhosis of the

liver is more common than previously thought, affecting more than 633,000 adults yearly, according to a study published in the *Journal of Clinical Gastroenterology*. People with cirrhosis had a mortality rate of 26.4 percent during a two-year interval. Most common cause of the liver cirrhosis is alcohol intake (60 to 70 percent), biliary obstruction (5 to 10 percent), biliary atresia/neonatal hepatitis, chronic Hepatitis B or C (10 percent) and hemochromatosis (5 to 10 percent). Symptoms include jaundice, fatigue, bleeding or bruising easily, nausea, swelling and confusion. But many patients have no symptoms. Cirrhosis can lead to liver failure and liver cancer. Numerous studies have attempted to develop the *Child-Pugh-Turcotte (CPT) classification* for prognosis of liver cirrhosis. Major complications of cirrhosis are, Ascites, Upper gastrointestinal bleeding, Hepatic coma or Hepatic Encephalopathy which can further lead to death. Dietary management for cirrhotic patients in general focus on suppression of hepatotoxic agents and the provision of optimal macronutrient supply in terms of energy, protein, carbohydrates and lipids together with micronutrients such as vitamins and minerals to maintain optimum nutrition status. Early identification and treatment of malnutrition in chronic liver disease has the potential to lead to better disease outcome as well as prevention of the complications of chronic liver disease and improved transplant outcomes.

***Keywords*---**Liver cirrhosis, Child-Pugh-Turcotte (CPT), lipids, Hepatic Encephalopathy.

Introduction

Human body consist of many small and functional unit of Cells, which further in group makes Tissue and group of tissue makes Organs and group of organs make an Organ System, that work together to perform a specific function in the body. Various organs like, liver, pancreas, spleen, kidney, heart, adrenal gland, etc., are the body's recognizable structure which perform various specific functions. (V.F. **Alexandra et.al. 2019) (1)**

Liver is the largest organ and gland in the human body. It carry more than 500 roles in the body. It is the only organ that can regenerate and weighs up to 1.44-1.66 kg. It is situated above and left to stomach and below lungs and is of reddish brown texture. It is the major organ for metabolism of three major nutrients i.e., carbohydrates, protein and fat.

Liver cirrhosis is a chronic liver infection. It consist of deterioration of liver cells along with fibrosis and infection generating nodules. It leads to portal vein hypertension. (Saeed et al. 2018)(2) Liver Cirrhosis results from different mechanisms of liver injury that lead to necroinflammation and fibrosis. Histologically, LC is characterized by diffuse nodular regeneration surrounded by dense fibrotic septa with subsequent collapse of liver structures and thus causes pronounced distortion of vascular architecture in the liver. (D. Schuppan et.al.2008) (3)

Patients with cirrhosis frequently have either global malnutrition or alterations in specific aspects of nutritional status, such as micronutrient deficiencies, due to multiple mechanisms, including poor nutritional intake, poor absorption, and increased losses. Malnutrition is present in almost every patient with alcoholic cirrhosis and is frequent in most other types of cirrhosis. In addition, one of the most significant nutritional problems in cirrhotic patients is muscle wasting and sarcopenia. Patients with cirrhosis often go into a catabolic phase overnight due to limited glycogen stores in the liver. (McClain C. J. et.al.2016) (4) Alcohol is the most common cause of cirrhosis of liver and the burden of alcohol-related cirrhosis is significantly increasing in comparison to other causes including viral infection, nonalcoholic steatohepatitis (NASH), and autoimmune hepatitis. (Mishra, D.,et.al.2020) (5)

Based on these backgrounds, several methods for evaluating nutritional status in patients with chronic liver disease such as indirect calorimetry, dual-energy X-ray absorptiometry (DEXA), bio impedance analysis (BIA), and anthropometry have been developed and they have been preferably used in the clinical settings. (A. J. Montano-Loza.et.al.2014) (6)

Prevalence

Cirrhosis of the liver is more common than previously thought, affecting more than 633,000 adults yearly, according to a study published in the *Journal of Clinical Gastroenterology*. (Dr. Scaglione ,et.al.2015) (7). According to the latest WHO data published in 2017, liver disease deaths in India reached 259,749 or 2.95% of total deaths, accounting for one-fifth (18.3%) of all cirrhosis deaths globally. With the rapidly growing economy and changes in lifestyle and nutrition, it is presumed that the etiological factors of liver cirrhosis in India might have changed over the past few years. It has been reported that in India, alcohol consumption increased by 55% from 1992 to 2012 with doubling of per capita consumption between 2005 and 2016. Singh et al. in a study from eastern India reported that 50% of the patients with alcoholic liver disease started drinking before the legal age of drinking. (Mishra, D.,et.al.2020) (5)

People with cirrhosis had a mortality rate of 26.4 percent during a two-year interval, compared with an 8.4 percent two-year mortality rate among similarly matched adults who did not have cirrhosis. Compared with the general population, people with cirrhosis tended to be older. Men were more at risk for cirrhosis than women. Prevalence was higher among poor people and people without a domestic partner. Prevalence declined with increasing levels of education. (Dr. Scaglione ,et.al.2015) (7)

Etiology

Most common cause of the liver cirrhosis is alcohol intake (60 to 70 percent), biliary obstruction (5 to 10 percent), biliary atresia/neonatal hepatitis, chronic Hepatitis B or C (10 percent) and hemochromatosis (5 to 10 percent). (Saeed et al. 2018)(2). Chronic hepatitis C virus (HCV) infection affects about 170 million people worldwide and is the most common cause of chronic liver disease. Of these HCV-infected individuals, 20–30% eventually develop liver cirrhosis (LC) or

hepatocellular carcinoma (HCC). In our country, about 30,000 persons per year die from HCC, with 70–80% of these deaths ascribed to HCV. (D. Schuppan et.al.2008) (3)

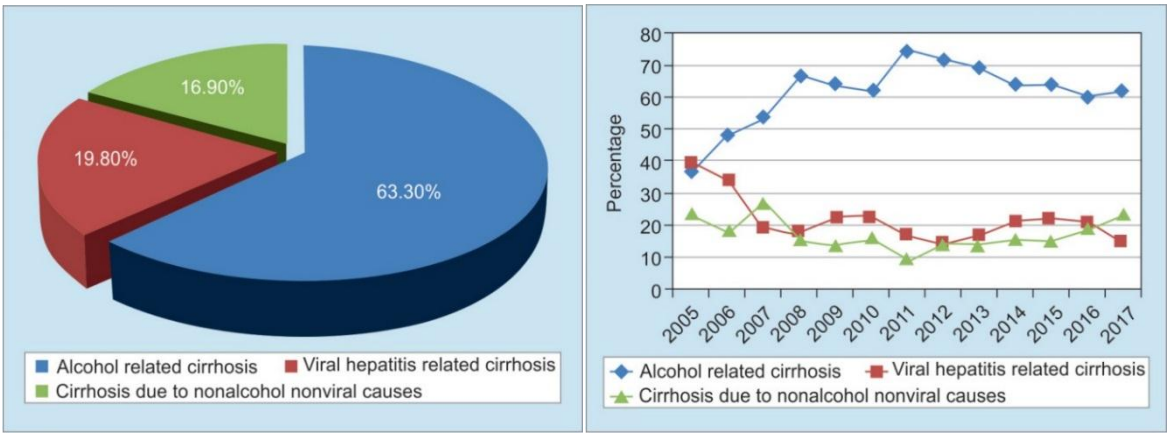
A study of 5,138 patients admitted to the Institute of Liver and Biliary Sciences in New Delhi, India with cirrhosis were hospitalized from 2010 to 2017 without acute-on-chronic liver failure (ACLF) and with at least a 1-year follow-up after their index hospitalization. Most of the patients included in the study (84.8%) had decompensated cirrhosis at inclusion. The most common etiology was alcohol-related liver disease (39.5%), followed by NASH (18.2%) and HBV-related cirrhosis (10.8%). (Jain, P.,et.al.2021) (8)

Data were obtained from hospital records of all patients admitted to the Department of Gastroenterology, Srirama Chandra Bhanja Medical College and Hospital, Cuttack, Odisha. A total of 16,902 patients were hospitalized in the gastroenterology ward. Out of which, 4,331 patients were diagnosed to have cirrhosis of liver and were included in the analysis, of whom 2,742 (63.3%) had alcohol-related cirrhosis, 858 (19.8%) had viral hepatitis-related cirrhosis, and 731 (16.9%) had cirrhosis of liver due to nonalcoholic and nonviral causes. Majority of the cirrhosis patients were male, i.e., 3,663 out of 4,331 (84.6%). Among the patients with alcohol-related cirrhosis, 97.5% were male, and among patients with viral hepatitis-related cirrhosis, 69.9% were male. (Mishra, D.,et.al.2020) (5)

Easy access to calorie-dense food and sedentary lifestyle together with the modern epidemics of diabetes mellitus (DM) and obesity have catapulted NAFLD into a substantial public health problem in India as in other parts of the world. NAFLD has emerged as one of the leading causes of cirrhosis, hepatocellular carcinoma (HCC), and liver transplant in India. (Duseja, A. et.al.2021) (9)

Thus, causes of cirrhosis can be enumerated as-

- Alcohol consumption
- Infections (hepatitis B, C and D)
- Metabolic disorders (Wilson’s disease)
- Autoimmune hepatitis
- Non- alcoholic fatty liver disease (NAFLD)
- Biliary tract disease
- Obesity
- Genetic factors
- Bad eating habits

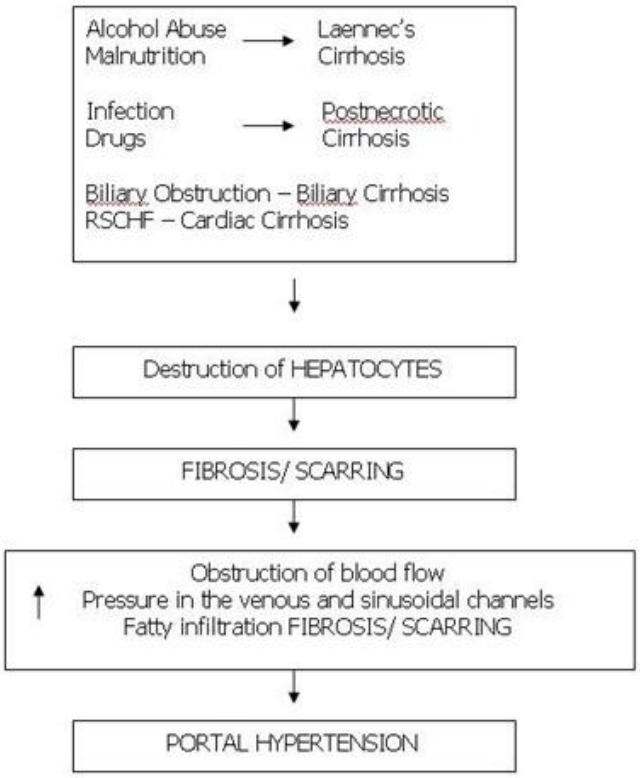


Pathophysiology of liver cirrhosis

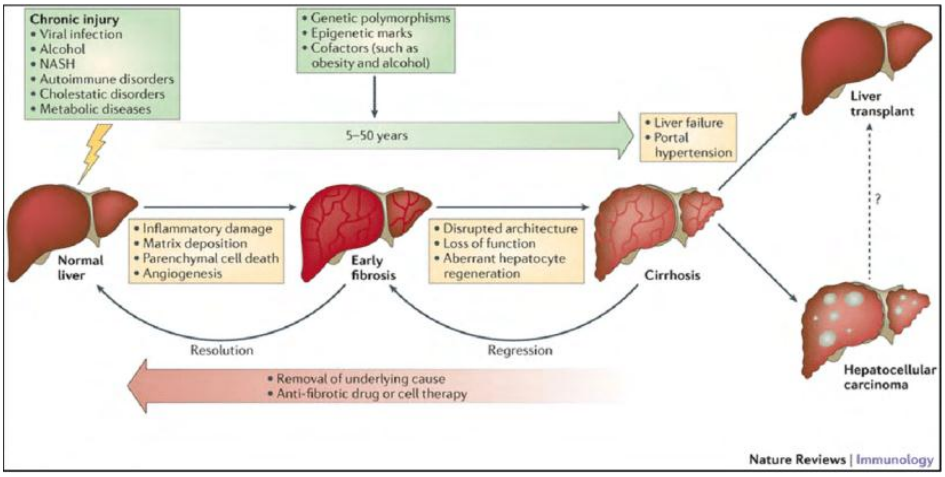
The liver plays a vital role in the synthesis of proteins (for example, albumin, clotting factors and complement), detoxification, and storage (for example, of vitamin A and glycogen). In addition, it participates in the metabolism of lipids and carbohydrates.

Cirrhosis is often preceded by hepatitis and fatty liver (steatosis), independent of the cause. If the cause is removed at this stage, the changes are fully reversible.
The pathological hallmark of cirrhosis is the development of scar tissue that replaces normal tissue. This scar tissue blocks the portal flow of blood through the organ, raising the blood pressure and disturbing normal function. Research has shown the pivotal role of the stellate cell, normally stores vitamin A, in the development of cirrhosis. Damage to the liver tissue from inflammation leads to the activation of stellate cells, which increases fibrosis through the production of myofibroblasts, and obstructs hepatic blood flow. In addition, stellate cells secrete TGF beta 1, which leads to a fibrotic response and proliferation of connective tissue. TGF-β1 have been implicated in the process of activating hepatic stellate cells (HSCs) with the magnitude of fibrosis being in proportion to increase in TGF β levels. ACTA2 is associated with TGF β pathway that enhances contractile properties of HSCs leading to fibrosis. Furthermore, it secretes TIMP1 and TIMP2, naturally occurring inhibitors of matrix metalloproteinases, which prevents them from breaking down the fibrotic material in the extracellular matrix.

As this cascade of processes continues, fibrous tissue bands (septa) separate hepatocyte nodules, which eventually replace the entire liver architecture, leading to decreased blood flow throughout. The spleen becomes congested, and enlarged, resulting in its retention of platelets, which are needed for normal blood clotting. Portal hypertension is responsible for the most severe complications of cirrhosis. (Hammer GD,et.al.2010) (11)



http://patrickaguas.blogspot.com/2014/05/liver-cirrhosis.html



https://www.researchgate.net/publication/333172634_Pros_and_Cons_of_Existing_Biomarkers_for_Cirrhosis_of_Liver

Symptoms

Early symptoms of cirrhosis may include (NIDDK.et.al.2021) (12)

- feeling tired or weak
- poor appetite
- losing weight without trying
- nausea and vomiting
- mild pain or discomfort in the upper right side of your abdomen

As liver function gets worse, you may have other symptoms, including

- bruising and bleeding easily
- confusion, difficulties thinking, memory loss, personality changes, or sleep disorders
- swelling in your 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 your urine
- blood in vomiting
- yellowish tint to the whites of your eyes and skin, called jaundice

Cirrhosis is end-stage scarring of the liver. Symptoms include jaundice, fatigue, bleeding or bruising easily, nausea, swelling and confusion. But many patients have no symptoms. Cirrhosis can lead to liver failure and liver cancer. (Dr. Scaglione ,et.al.2015) (7)

Cirrhotic patients often have multiple micronutrient deficiencies like, Magnesium, Selenium, Fat soluble and water soluble vitamins, Zinc, Niacin, etc. They show signs and symptoms like, insulin resistance, muscle cramps, myopathy, neurological symptoms, glossitis, cheilitis, oxidative stress, confusion, metabolic abnormalities, etc. (McClain C. J. et.al.2016) (4)

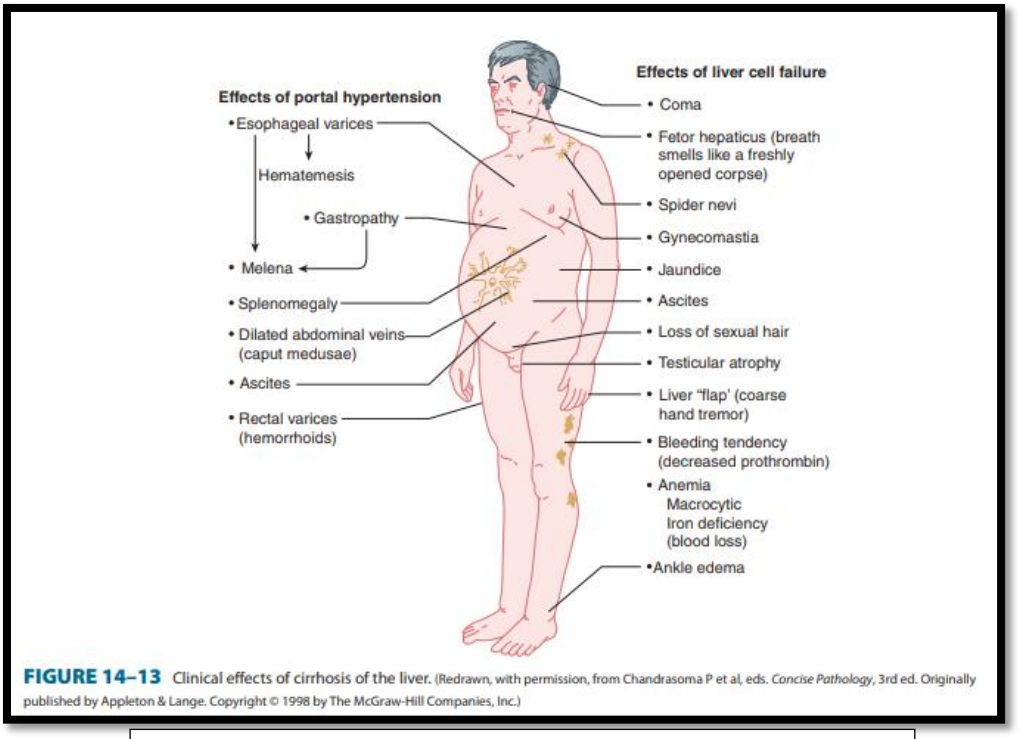


FIGURE 14–13 Clinical effects of cirrhosis of the liver. (Redrawn, with permission, from Chandrasoma P et al, eds. *Concise Pathology*, 3rd ed. Originally published by Appleton & Lange. Copyright © 1998 by The McGraw-Hill Companies, Inc.)

SYMPTOMS OF LIVER CIRRHOSIS

Prognosis of liver disease

Numerous studies have attempted to develop a classification system that can both characterize the degree of liver injury and predict the prognosis of patients with cirrhosis based on clinical and laboratory parameters. Due to its low level of complexity and its fairly good predictive value, the *Child-Pugh-Turcotte (CPT) classification* is widely used.

Child Pugh Turcotte (cpt) Classification (schuppan, d.et.al.2008) (13)

points	**1**	**2**	**3**
Encephalopathy	absent	medically controlled	poorly controlled
Ascites	absent	controlled medically	poorly controlled
Bilirubin (mg/dL)	< 2	2-3	> 3
Albumin (g/dL)	< 3.5	2.8-3.5	< 2.8
INR	< 1.7	1.7-2.2	> 2.2

	CPT A: 5-6 POINTS	CPT B: 7-9 POINTS	CPT C: 10-15 POINTS
Life expectancy (years)	15-20	4-14	1-3
Perioperative mortality (abdominal surgery) (%)	10	30	80

INR, international normalized ratio.

Complications and Prevention (Schuppan, D. et.al.2008) (13)
Major complications of cirrhosis include-

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

Complication	Prevention	Treatment
Variceal bleeding	Non selective beta blockers Variceal band ligation	*Acute:* Resuscitation, Vasoconstrictors, Sclerotherapy Band, Ligation, TIPSS, Surgical Shunts
		Chronic: Variceal obliteration, TIPS, Surgical Shunts
Ascites	Low Na diet	Low Na diet, Diuretics, Large volume paracentesis, TIPSS
Renal failure	Avoid hypovolemia	Discontinue diuretics, Rehydration, Albumin infusion
		Hepatorenal syndrome: Add Terlipressin or Midodrine (Noradrenaline) and Somatostatin (Octreotide)
Encephalopathy	Avoid precipitants	*Treat precipitating factors:* Infection Bleeding Electrolyte imbalance Sedatives High protein intake
		Lactulose Neomycin, Metronidazole, Rifaximin
Spontaneous bacterial peritonitis	Treat ascites	Early diagnostic paracentesis: Neutrophils >250/cc → antibiotics iv Secondary prophylaxis with a po-antibiotic such as Levofloxacin

Diagnostic Markers (shashidhar kn.et.al.2019) (10)

biomarkers	description	etiology
AST and ALT	Often normal or moderately elevated	Leakage from damaged hepatocytes; AST to ALT ratio often above 1, especially in alcoholic cirrhosis (relative vitamin B6 deficiency)
ALP	Elevated <3-fold, except for PBC and PSC	Cholestasis
GGT	More specific for liver than ALP, high in active alcoholics	Cholestasis
Bilirubin	Elevated later than GGT and ALP, important predictor of mortality	Cholestasis, decreased hepatocyte and renal excretory function (exacerbated by systemic inflammation)
Albumin	Decreased in advanced cirrhosis	Decreased hepatic production, sequestration into ascites and interstitial (exacerbated in systemic inflammation), DD: malnutrition, protein losing enteropathy
Prothrombin time	Decreased in advanced cirrhosis	Decreased hepatic production of factor V/VII (While thrombin production is maintained), DD: vitamin K deficiency (e.g., due to mechanical biliary obstruction)
Immune globulins	Increased (mainly IgG)	Shunting of portal venous blood carrying (intestinal) antigens to lymph tissues with resultant stimulation of plasma cells
Sodium imbalance	Hyponatremia	Inability to excrete free water via the kidneys due to increased activity of antidiuretic hormone (vasopressin 2 receptor effect)
Anemia	Macro-, normo- or microcytic anemia	Folate deficiency, Hypersplenism, direct toxicity (alcohol), gastrointestinal blood loss (e.g., via esophageal varices)
Thrombocytes and leukocytes	Thrombocytopenia (Leukopenia)	Hypersplenism, dysfibrinogenemia, reduced hepatic thrombopoietin production

(Schuppan, D.e.tal https://doi.org/10.1016/S0140-6736(08)60383-9)

Dietary Management

Dietary management for cirrhotic patients in general focus on suppression of hepatotoxic agents and the provision of optimal macronutrient supply in terms of energy, protein, carbohydrates and lipids together with micronutrients such as vitamins and minerals. Energy, macro- and micronutrient supplies should be based on the results of individual nutritional assessments and adjusted for weight maintenance and/or repletion. (Bémeur, C et.al.2014) (14)

PRINCIPLES OF DIET FOR CIRRHOTIC PATIENT
A high calorie, high protein, high carbohydrates, moderate or restricted fat, restricted sodium (during ascites), high vitamins and minerals (help in regeneration of liver and prevent ascites), restricted fiber (during portal hypertension and esophageal varices).

General Recommendations For Cirrhotic Patients (Bémeur, C et.al.2014) (14)

Nutriment	Recommendation
Energy	30–50 kcal/kg body weight, Sufficient to restore/maintain nutritional status and enhance liver regeneration (adjust for obese patients)
Protein	1.0–1.8 g/kg body weight depending on the severity of malnutrition (adjust if renal disease present)
Carbohydrates	45–75% of caloric intake or 4–6 meals rich in carbohydrates per day
Lipids	20–30% of caloric intake (adjust if steatorrhea present)
Vitamins	B group vitamin supplements, Particular attention to lipid-soluble vitamins to Correct specific deficiencies (especially vitamin A)
Minerals	Zinc, magnesium, copper and selenium supplements to Correct specific deficiencies

Role of Probiotics in Cirrhotic Patients

The role of probiotics, live bacteria that provide a health benefit to the host, is not well documented in humans with cirrhosis. There is a wealth of experimental data in animals suggesting that probiotics are beneficial, especially certain types of probiotics. A study was done by Dr. McClain C. J. and his colleagues on *Lactobacillus rhamnosus* GG and have found that it provides many benefits for liver disease, including stabilizing the gut barrier function, improving the gut flora, decreasing endotoxin levels, and improving liver enzymes. Whether all of these benefits will translate into humans is not known, but many studies are currently investigating this issue. (McClain C. J. et.al.(2016) (4)

Diet rich in trans/saturated cholesterol and fat, fructose-sweetened beverages enhances visceral adiposity and promote hepatic lipid accumulation and passage into non-alcoholic steatohepatitis, minimizing caloric intake, elevating

soy protein consumption, supplements of monounsaturated fatty acids and omega- 3 fatty acids and whey proteins intake and probiotics have therapeutic and preventive influences. (Zahra SM, et al.2018) (2)

Conclusion

Many advances have took place within side the scientific care of sufferers with cirrhosis and the headaches of give up the level to liver disease. The majority are those who have centered on remedy of the underlying motive of cirrhosis and controlled the headaches of portal hypertension. Alcohol abuse, diabetes and hepatitis C had been the maximum large contributing elements in greater than 50 percentage of the cirrhosis cases. Health is a circumstance of whole mental, social and bodily wellbeing and now no longer simply the absence of any disease. There are many parameters which have influence on the health state of a person, however nutrients plays a key role in selling health, prevention from numerous sicknesses and consequently enhancing high-satisfactory of life. Nutritional health assessment plays a role in evaluating nutrients associated risks that might involve in person's future or current health. Liver cirrhosis patients often have PEM and poor physical activity. Nutritional fame in cirrhotic sufferers ought to be exactly and appropriately assessed as a way to layout a dietary intervention tailored to the wishes of the man or woman patient.

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