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High Calorie Protein Formulas Vs Normal Indian High Calorie High Protein Diet in Chronic Liver Disease

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Abstract

Chronic liver disease (CLD) is a result of progressive deterioration of liver functions for more than six months. Out of all the reported cases of CLD, approximately 20% with compensated cirrhosis and 65%-95% with decompensated cirrhosis have protein-calorie malnutrition (PCM). Protein-calorie malnutrition is a condition wherein cachexia occurs due to nutritional deficiencies in calories and protein. In CLD patient's malnutrition is an independent predictor of mortality. It is therefore essential to diagnose the presence of PEM and provide appropriate nutritional support in CLD patients with PEM to improve the prognosis.

The nutritional intake of chronic hepatitis C (CHC) patients is almost similar to that of healthy individuals. For patients with compensated cirrhosis without malnutrition, the ingestion of 1.2-1.5 g/kg(weight) daily protein is suggested. In malnourished patients, 1.0–1.8 g/kg(weight) is suggested, depending on the severity of malnutrition and liver disease.

Nutritional supplements provide significant increase in the protein-caloric intake with no modification of hospital diet. They have become an appropriate nutritional source when the diet is insufficient. Commercially manufactured polymeric formulas are considered as the "standard" approach to enteral nutrition. They provide complete nutrition and are made up of mostly intact nutrients, which implies a functional digestive system, and are appropriate for use both in hospitals and home care. In polymeric formulas, protein provides 15 -25% of total energy. Protein content ranges from 30 to 80 g l^{-1}, with non-protein calorie: nitrogen ratio between 75:1 and 200:1 kcal $g^{-1}N$.

Indian diets are unhealthy in all states and income categories. In addition, Indians consume too many cereals and too few

proteins, fruits, and vegetables. Importantly, unlike many other countries, India does not have a problem with excessive animal protein consumption. Indian officials must speed up efforts to make healthier, more sustainable diets more affordable, accessible, and acceptable across the entire food system. Treatment should target on maintaining adequate protein and caloric intake and rectifying nutrient deficiencies. Along with treatment nutritional support is also required to prevent malnourishment, treatment interruption, and improve the quality of life in CLD patients. Nutritional therapy should be designed to encourage consumption of frequent small meals and a late evening snack to reduce protein breakdown. In cases where oral intake is insufficient, early implementation of enteral feeding should be considered. Malnutrition is a potentially reversible condition and when identified early and treated appropriately, can lead to improved outcomes.

Keywords: Chronic Liver Disease; Protein Calorie Malnutrition; Protein; Calories; Nutritional Protein Supplements; Polymeric Formulas; Enteral Nutrition; Parenteral Nutrition

Introduction

Chronic liver disease

Chronic liver disease (CLD) is a result of progressive deterioration of liver functions for more than six months, it includes synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism and excretion of bile [1]. Out of all the reported cases of CLD, approximately 20% with compensated cirrhosis and 65%-95% with decompensated cirrhosis have protein-calorie malnutrition (PCM) [2]. Protein-calorie malnutrition is a condition wherein cachexia occurs due to nutritional deficiencies in calories and protein. This reduction in essential macronutrients level can lead to extensive body wasting as well as other related sequelae [2].

Methodology

In this review article the literature has been collected from the available data on protein calories intake in CLD. Indians have lower muscle mass and higher prevalence of sarcopenia. Given the wide consensus that nutritional status should be routinely assessed in all patients with CLD in order to recognize malnutrition and prevent nutritional depletion, the development of a simple, well validated and reproducible high protein nutritional formula for the fulfilment of nutritional requirement in these patients is very necessary. Indian diets derive almost 60% of their protein from cereals with relatively low digestibility and quality.

Protein metabolism and the liver

A crucial role is played by the liver in the metabolism of proteins along with carbohydrates and fats. Liver has four main functions in protein metabolism and they are:

- Formation of blood proteins, 80% of which are synthesized in the liver and secreted into the blood stream to perform many functions;
- Amino acid interconversion;
- Protein metabolism is amino acid deamination, or breakdown, the by-products of which can be used to produce energy (ATP);
- Liver removes this excess ammonia by producing urea which is ultimately excreted by the kidneys.

Several other hormones in the body such as insulin, glucagon, epinephrine, and steroids also alter protein metabolism, the effects of which can be amplified even more in the setting of liver disease. The loss of hepatic regulation of protein metabolism leads to a rapid death in acute liver failure, and that changes in protein metabolism play a role in complications of chronic liver failure such as the development of hepatic encephalopathy (HE), ascites and PCM [3].

In liver disease the hallmarks of protein and amino acid metabolism are lowered concentrations of circulating branched-chain and increased concentrations of circulating aromatic amino acids with concomitantly altered amino acid kinetics. The alteration in amino acid kinetics in liver disease are characterized by increased endogenous leucine flux, an indicator of protein breakdown, and leucine oxidation in the post-absorptive state (when calculated using a reciprocal-pool model and normalized for body cell mass). Additionally, the rise in whole-body protein synthesis in response to an amino acid infusion may be diminished in patients with cirrhosis. These changes are often accompanied by clinically apparent muscle wasting, manifested as PCM, and associated low levels of hepatically synthesized plasma proteins [4].

Protein-calorie malnutrition

Protein-calorie malnutrition may be present in 65–90% of patients with advanced disease. Malnutrition occurs at a very initial stage of liver disease and there is, almost, a direct association between the severity of liver disease and the degree of malnutrition. The PCM can lead to several complications such as esophageal varices, hepatic encephalopathy (HE), hepatorenal syndrome, impaired liver function and regeneration capacity and increased surgical morbidity and mortality. In CLD patient's malnutrition is an independent predictor of mortality. Patients with hepatocellular disease are more likely to have protein deficiency [5]. It is therefore essential to diagnose the presence of PEM and provide appropriate nutritional support in CLD patients with PEM to improve the prognosis [6].

Protein-calorie malnutrition occurs in CLD patients as a result of a deficit in calorie and protein intake due to [7]:

- Poor nutrition intake in liver disease;
- Metabolic changes in liver diseases;
- Hyperactivity of β-adrenergic system; and
- Malabsorption of fats.

As liver is incapable of producing sufficient amounts of bile, and because of decreased micelle formation, fatty acid malabsorption occurs which contributes to PCM by reducing the amount of calories available for the body's use [7].

Several studies have reported that patients with cirrhosis and hepatic encephalopathy may benefit themselves from the use of modified normocaloric (30kcal/kg of body weight/day) or hyperproteic (1.2g protein/kg of body weight/day) diets, with increased vegetable and dairy proteins intake; and with significant reduction of plasma ammonia [8].

Factors That Contribute to Malnutrition in Chronic Liver Disease

Inadequate nutrient intake [9]
• Early satiety (ascites)
• Anorexia (nausea, vomiting, abdominal distention)
• Dysgeusia (zinc deficiency)
• Hepatic encephalopathy
• Restricted diet (low protein, low sodium, fluid restriction)
• Alcohol intake
• Socioeconomic barriers
Metabolic changes
• Hypermetabolic state
• Increased gluconeogenesis
• Insulin resistance
Malabsorption
• Portosystemic shunting (bowel wall edema, portal venous stasis)
• Bile acid deficiency
• Small-bowel bacterial overgrowth

Results

Nutritional requirement in chronic liver diseases

The nutritional intake of chronic hepatitis C (CHC) patients is almost similar to that of healthy individuals. During interferon (IFN)-based antiviral treatment, reduction in weight is seen in 11–29% of patients because of decreased appetite and malnutrition [10].

For patients with compensated cirrhosis without malnutrition, the ingestion of 1.2-1.5 g/kg(weight) daily protein is suggested. In malnourished patients, 1.0–1.8 g/kg(weight) is suggested, depending on the severity of malnutrition and liver disease. Protein requirement are higher in malnourished patients and in stress situations (such as bleeding, infection or surgery), provided that there is no renal dysfunction (in which may be necessary a protein restriction) [5].

It has been recommended by the researchers that the simple addition of a carbohydrate and protein-rich evening snack may also help nitrogen balance, improve muscle cramps and prevent muscle breakdown by supplying the body with overnight carbohydrate energy, and preventing gluconeogenesis. One of the most common limiting amino acids in vegetable proteins is methionine. These intestinal by-products of methionine are crucial for the pathogenesis of Hepatic Encephalopathy (HE). Vegetable proteins have low methionine; hence, it is therefore thought that they may be better protein sources for patients with HE or those at high risk of developing hepatic encephalopathy [11].

Presently, the recommended protein intake should be1.2 to 1.5 g/kg/day. Patients who do not meet their protein intake goals by dietary protein may benefit from branched-chain amino acid (BCAA) supplementation. In patients with HE, BCAA supplementation may also have a benefit [9].

Energy and Protein Recommendations in Chronic Liver Disease: ESPEN/ASPEN Guideline [9]

	ESPEN Guideline	ASPEN Guideline
Energy requirement	35-40 kcal/kg/day	With acute encephalopathy: 35 kcal/kg/day Without encephalopathy: 25-35 kcal/kg/day Stable and malnourished: 30-40 kcal/kg/day
Protein requirement	1.0-1.5 g/kg/day	With acute encephalopathy: 0.6-0.8 g/kg/day Without encephalopathy: 1.0-1.5 g/kg/day Stable and malnourished: 30-40 kcal/kg/day

Indian high calorie/high protein diet

India stands at a very vulnerable position with one of the highest prevalence of undernutrition in the world in spite of improvement in food availability and poverty alleviation [12].

Daily protein requirements for Indians based on food habits

It has been stated in the Indian Market Research Bureau's 2017 report that protein deficiency among Indians stands at more than 80%, measured against the recommended 60g/ day. To fulfil the daily requirement of 60g protein/ day, it is necessary to eat eight bowls of lentils or drink 7-8 glasses of milk. A mixed diet can meet the protein requirements of children, provided that the protein content of the foods is such that it contributes to around 10% of the total calories [13].

Recommended Dietary Allowances for Indians [14]

Group	Particulars	Body weight (kg)	Net Energy (Kcal/d)	Protein (g/d)
Male	Sedentary work	60	2320	60
	Moderate work		2730	
	Heavy work		3490	
Female	Sedentary work	55	1900	55
	Moderate work		2230	
	Heavy work		2850	
	Pregnant female		+350	+23
	Lactation 0-6 months		+600	+19
	6-12 months		+520	+13
Infants	0-6 months	5.4	92 Kcal/kg/d	1.16 g/kg/d
	6-12 months	8.4	80 Kcal/kg/d	1.69 g/kg/d
Children	1-3 years	12.9	1060	16.7
	4-6 years	18	1350	20.1
	7-9 years	25.1	1690	29.5
Boys	10-12 years	34.3	2190	39.9
Girls	10-12 years	35.0	2010	40.4
Boys	13-15 years	47.6	2750	54.3
Girls	13-15 years	46.6	2330	51.9
Boys	16-17 years	55.4	3020	61.5
Girls	16-17 years	52.1	2440	55.5

Some protein rich foods [14]			
Nutrients	**Food groups**	**Foods**	**Nutrient content for 100gm edible portion**
Protein	Pulses and legumes	Bengal gram, black gram, green gram, lentil and red gram	22 gm
	Nuts and oilseeds	Groundnuts, cashew nuts and almonds	23 gm
	Fish		20 gm
	Meat and poultry	Meat Egg white	22 gm 11 gm
	Milk products	Cheese, *khoa,* skimmed milk powder (cow) and whole milk powder (cow)	30 gm

High calorie protein formulas: Recommendation criteria

Nutritional supplements are judiciously used in most of the cases. They provide significant increase in the protein-caloric intake with no modification of hospital diet. They have become an appropriate nutritional source when the diet is insufficient [15].

Commercially manufactured polymeric formulas are considered as the "standard" approach to enteral nutrition. They provide complete nutrition and are made up of mostly intact nutrients, which implies a functional digestive system, and are appropriate for use both in hospitals and home care [16].

ESPEN Guideline on Enteral Nutrition: CLD

(steatohepatitis and liver cirrhosis) [17]

Recommended energy intake: 30-40 kcal/kgBW/d

Recommended protein intake: 1.2-1.5 kcal/kgBW/d

In polymeric formulas, protein provides 15 -25% of total energy. Protein content ranges from 30 to 80 g l^{-1}, with non-protein calorie: nitrogen ratio between 75:1 and200:1 kcal g^{-1}N. Sources contain proteins in their original natural forms (e.g., milk, egg protein) and protein isolates, separated from the various original foods : Casein, delactozed lactalbumin and whey protein – Cow's milk Soy protein isolate – Soy Egg white, egg albumin – Eggs. Because of their size proteins have little influence on the formula's osmolality; however normal levels of pancreatic enzymes are required for adequate digestion. Protein modules are capable of increasing nitrogen intake. Commonly used sources include: casein or calcium caseinate, lactalbumin, egg albumin, whey, soy protein etc. The disadvantage of using caseinate is that it doesn't mix easily due to its high viscosity. Unlike maltodextrins, some protein modules maybe indigestible to patients. The specialized formulas for hepatic failure and HE contain higher proportion of branched-chain amino acids (BCAA) and a reduction in aromatic amino acids (AAA) and methionine, in order to rectify the abnormal plasma ratio of these amino acids and increase the Fischer ratio (BCAA/AAA). Most diets are low in protein and electrolytes, and slightly calorically dense (>1 kcal ml^{-1}) due to find restrictions. These diets should be considered for patients with normal gut function, exhibiting HE, and not responding tstandard formul[16].

ESPEN guideline on clinical nutrition in chronic liver disease [17]

- Cirrhotic patients in conditions of increased energy expenditure (i.e. acute complications, refractory ascites) or malnutrition, should take an increased amount of energy
- Non-malnourished patients with compensated cirrhosis should take 1.2 $g \cdot kg^{-1} \cdot d^{-1}$protein
- To replenish malnourished and/or sarcopenic cirrhotic patients the amount of 1.5 $g \cdot kg^{-1} \cdot d^{-1}$ protein should be consumed
- In cirrhotic patients with HE, protein intake should not be restricted as it increases protein catabolism
- Cirrhotic patients with malnutrition and muscle depletion should consume oral diet that provides 30-35 kcal x kg^{-1} x $d^{-1}$and 1.5g protein x kg^{-1} x d^{-1}
- In cirrhotic patients who are protein "intolerant", vegetable proteins or BCAA (0.25 g x kg^{-1} x d^{-1}) should be used by oral route to compensate for adequate protein intake
- In patients with advanced cirrhosis long-term oral BCAA supplements (0.25 g x kg^{-1} x d^{-1}) should be prescribed in order to improve event-free survival or quality of life
- After the acute postoperative phase an energy intake of 30-35 kcal x kg^{-1} x d^{-1} (126-147 kJx kg^{-1} x d^{-1}) and a protein intake of 1.2-1.5 g x kg^{-1} x d^{-1} should be considered
- After transplantation, enteral formula together with selected probiotics should be given to lower the rate of infection
- BCAA-enriched formulas can be used in patients with HE requiring EN.

Disease's condition: Indian high calorie/high protein diet vs. Protein supplements

Liver cirrhosis

Nutritional support in liver cirrhosis[17]	
Compensated cirrhosis	25-35 kcal/kg/d 1.0-1.2 g/kg/d
Inadequate intake or malnutrition	35-40 kcal/kg/d 1.5 g/kg/d
Encephalopathy I-II	35-40 kcal/kg/d 0.5-1 g/kg/d if protein intolerant: vegetable protein or BCCA
Encephalopathy III-IV	35-40 kcal/kg/d 0.5 g/kg/d BCCA-enriched amino acid solution is recommended

Chronic liver disease patient's diet is based on a standard diet with supplements addition as necessary. In fact, in most cases it is possible to give a standard normal diet. Restrictions can be hazardous and should depend on individual case. The treatment goals are to achieve the level of PCM, to ensure an adequate amount of nutrients, to achieve a positive nitrogen balance and to avoid hepatotoxic agents. Long-term prognosis can be improved with rarely rectification of nutritional deficiencies [5].

In case of liver cirrhosis, the high protein diet helps regenerate liver cells. Also, it maintains the nitrogen balance but low enough to prevent hepatic coma. In cirrhosis there is increased protein breakdown and inadequate resynthesis, which results in depletion of visceral protein stores and muscle wasting. It has been recommended that 0.8-1g of protein/kg/day is the mean protein requirement to achieve nitrogen balance. Therefore, in uncomplicated hepatitis or cirrhosis without HE, protein requirements range from 0.8 to I g/kg of dry weight per day to attain nitrogen balance. To promote nitrogen accumulation or positive balance, at least 1.2 to 1.3g/kg daily is required [17].

In patients with compensated cirrhosis without malnutrition, the ingestion of 1.2–1.5g/kg(weight) daily protein is recommended. In malnourished patients, 1.0–1.8g/kg(weight) is recommended, depending on the severity of malnutrition and liver disease. Protein needs are higher in malnourished patients and in stress situations (such as bleeding, infection or surgery), provided that there is no renal dysfunction (in which may be necessary a protein restriction) [5].

In case of liver cirrhosis, protein restrictions can have a devastating effect on nutritional status as it changes the protein requirements and energy metabolism. It leads to negative nitrogen balance which in turn can result in worsening HE [18]. In case of alcoholic hepatitis or decompensate disease (sepsis, infection, gastrointestinal bleeding, severe ascites), at least 1.5g of protein per kg/ day is recommended. In case it is required to provide additional protein, enteral or parenteral formulas that are low aromatic amino acids and high in branched chain amino acids can be given. These patients are able to tolerate vegetable and dairy protein better compared to animal protein because they contain fewer ammonia containing substances and aromatic amino acids (AAA) and more BCAA. Also, they provide more fibre which accelerates intestinal transit, thereby reducing the time available for production and absorption of ammonia in the body. Vegetable protein contains more valine and is also higher in arginine, an amino acid that reduces blood ammonia levels thereby increasing urea synthesis. Also, they have low methionine and tryptophan, amino acids that exacerbate encephalopathy through gut conversion to neurotoxin metabolites [17].

Alcoholic hepatitis

Nutritional status should be assessed in patients with alcoholic hepatitis. When malnutrition is present, vigorous nutrition therapy should be provided. Hepatology. In patients with alcoholic hepatitis with severe malnutrition, inadequate caloric intake was associated with 51% mortality compared with 19% mortality in patients who received adequate oral nutrition (>2,500 kcal/day) [19]. In patients with alcoholic cirrhosis receiving a daily supplement of 1,000 kcal and 34 g of protein (given as a casein-based enteral nutrition product) had better outcomes [20].

Hepatic encephalopathy

The recommended protein content is 1–1.5g/kg(weight)/day. The International Society for Hepatic Encephalopathy and Nitrogen metabolism (ISHEN) recommends the same energy (35–40kcal/kg(weight)/day) and protein contents (1.2–1.5g/kg(weight)/day) as those that are recommended for patients without, HE [5].

The ISHEN recommends that in patients with recurrent or persistent HE a diet richer in vegetable and dairy protein than meat and fish protein should be encouraged. The ASPEN recommends an increase in the content of milk or vegetable protein origin and BCAA-enriched supplements in patients with protein intolerance. Several studies have revealed that dairy protein is better tolerated than protein from mixed sources and that vegetable protein is better tolerated than meat protein. The proteins of vegetable and dairy origin improve the nitrogen balance and, if well tolerated by the patients, they may be provided without constraints. The vegetable proteins may influence intestinal transit. They have a higher content of fibre and BCAA and a lower content of AAA [5].

Liver transplantation

Post liver transplantation, normal food and/or enteral nutrition should be initiated within 12-24 hours postoperatively in order to attain lower rates of morbidity and complications and cost than during parenteral nutrition. Whole protein formulae with or without pre- and probiotics or peptide-based formulae via catheter jejunostomy are been used for early enteral nutrition

of adult liver transplant recipients. Post transplantation there is a considerable loss of nitrogen and patients typically remain in negative nitrogen balance for up to 28 days because of which it is necessary to have an increase in the provision of protein or amino acids. Protein or amino acid intakes of 1.0–1.5 gkgBW^{-1}d^{-1} have been recommended. There is no need to use a BCAA-enriched amino acid solution post liver transplantation [21].

Recommended protein nutritional supplement intake in chronic liver diseases

For enteral feeding in CHD a whole protein formula providing 35–40 kcal · kg^{-1} · day^{-1} energy and 1.2–1.5 g · kg^{-1} · day^{-1} protein is recommended. Standard nutritional supplements should contain approximately 100 kcal energy, 4 g protein, and 3.5 mmol of sodium and potassium per 100 mL. Concentrated high energy (1.5 kcal/mL) and protein commercial supplements are available worldwide and should be advised for patients with hyponatremia and ascites to regulate fluid balance. In patients who experience steatorrhea, it is advisable to limit long-chain fatty acids and increase short-chain and medium-chain fatty acids in the formula. It is advisable to initiate pancreatic enzymes supplement, especially in individuals with alcohol-related cirrhosis in whom pancreatic insufficiency is common. These enzymes get inactivated by gastric acid, and hence, proton pump inhibitors (PPIs) are necessary [22].

Liquid oral nutritional supplements

In some patient's liquid oral nutritional supplements are useful adjuncts to enhance calorie, protein and vitamin-mineral intake. Patients who experience early satiety or in any case experience issues with eating full meals can often fulfill the demand with oral liquid supplements. Although long term compliance and cost may limit the effectiveness of oral supplements for certain patients, yet, they can be an effective component of nutrition therapy for many patients diagnosed with cirrhosis [23].

Role of nutritional supplements in reversing disease or helping compensate a decompensated liver

In patients with decompensated liver disease the incidence of PCM is up to 90% which is irrespective of whether the disease is cholestatic or non-cholestatic in nature. Malnourishment is a common occurrence in almost 100% of liver transplant candidates [24].

It is recommended for patients with decompensated liver disease to eat foods which are high in calorie and contain protein. Patients should be advised to eat regular intervals throughout the day and limit the protein portions so that there is not a large protein load for the liver to breakdown at any one time [24].

Nutritional supplements are very useful for patients who cannot maintain their nutritional status. Milk based nutritional supplements provide higher protein contents and should be initiated first. They should be sipped slowly between meals in order to prevent them feeling too full. Diabetes medication should be optimised in order to allow supplements to be included in order to meet requirements instead of reducing them to control blood glucose levels [24].

Nutritional supplementation can improve nutritional status in CLD. There are data reporting that outpatient nutritional supplementation reduces hospitalization and improves immune function. In patients with cirrhosis having a late-night snack can help maintain lean muscle mass. Also, there are reports documenting that in patients with severe alcoholic hepatitis nutritional supplementation improved short-term mortality to a degree similar to corticosteroids, and had a better effect on long-term survival compared to corticosteroids [25].

Cirrhotic patients require to be compensated with high protein which is generally well tolerated by majority of patients. The adding adequate protein in the diets of malnourished patients significantly improves the mental status, level of the serum ammonia and the body weight. The daily eating pattern comprising 4 meals and l late evening carbohydrate snack contributes to liver cirrhosis improvement, avoiding protein loading in a period of day, but maintaining the protein positive balance. Protein restriction should not be advised and it is recommended to maintain 1.2-1.5 g proteins/kg/day [18].

Correcting nutrient deficiencies

In patients with CLD, nutritional therapy should not only target treating PCM, but should also focus to correct specific nutrient deficiencies.

Vitamin A

Fat malabsorption, as well as defective mobilization of vitamin A from the liver results in reduced serum vitamin A levels. Common complication of vitamin A deficiency is nyctalopia

(night blindness), which can be improved with vitamin A supplementation, generally at a dose of 25,000 units/day for 4–12 weeks. Generally, vitamin A deficiency resolves within 2 weeks of liver transplantation [26].

Vitamin D

Vitamin D deficiency is another complication of CLD. It can occur due malabsorption; decreased UV light exposure and inadequate dietary intake [26]. For patients with vitamin D deficiency (as defined by a 25-hydroxyvitamin D level <20 ng/mL) it is recommended to take 50,000 IU of vitamin D weekly for 8 to 12 weeks with a target 25-hydroxyvitamin D level of at least 30 ng/mL [27].

Calcium

Calcium deficiency, and eventually osteomalacia or osteoporosis, results from reduced intestinal calcium absorption. It is recommended to supplement all patients who have CLD with calcium (1 g/day) and vitamin D3 (800 IU/day) [26].

Zinc

Zinc deficiency is commonly seen in patients with cirrhosis and has been implicated in the pathogenesis of HE. Zinc supplementation at doses of 600 mg/ day for 3 months has been shown to improve mental status of patients with HE [26].

Recommendations for choosing the best nutritional supplement [28]	
Energy/Calories	Meal supplement drinks range from 200-500 calories per drink- ➢If a patient has temendous weight loss and muscle loss , a drink with 400-500 calories should be recommended ➢If patient's weight is stable or obese, a drink with 200 to250 calories is recommended
Protein	Most drinks range in protein from 9g-20 g Recommend a drink that have 10 g protein or more in each bottle
Sodium	After choosing the best options for calories and protein, the drink with lowest amount of sodium should be the ideal

Discussion

There has been an unprecedented evolution of commercial high protein diets, which have become a very strong means of nutritional intervention. The large spectrum of high protein diets formulas has diversified the prevention and treatment benefits of nutritional therapy. The objectives and formulation of high protein formulas should be patient driven , i.e. adjusted to the specific patient and disease requirements. Health care institutions should identify their patient's needs, and recommend a commercially available high protein formulas similar to a diet manual, adapting the number and variety of products to the disease type and frequency.

Poor nutritional status is associated with worse prognosis and it increases the mortality rates in CLD. India diet lacks adequate protein requirement and it is often overlooked. Indian diets are primarily cereal-based, with cereals accounting for 60% of protein and having poor digestion and quality. According to the Indian Dietetic Association (IDA), approximately 84% of Indians are protein deficient. This is due to a lack of information about the significance of eating enough protein and a largely vegetarian diet. What's more concerning is that roughly 65% of non-vegetarian Indian diets are protein insufficient.

Nutritional support via supplements reduces nutrition-associated complications. The dietary intake of patients should comprise of high levels of carbohydrate, fat, protein and cholesterol. Hence, thorough investigation of dietary habits can lead to better nutrition therapy in CLD. The CLD patients are malnourished due to presence of anorexia, vomiting and other gastrointestinal disorders. Hence, along with treatment nutritional support is also required to prevent malnourishment, treatment interruption, and improve the quality of life. Some patients with CLD have reduced dietary energy and protein intake. It has also been seen that the number of CLD patients with overeating and obesity is on a rise, indicating that the nutritional state of CLD patients has a broad spectrum. Therefore, nutritional supplement for liver cirrhotic patients should be based upon their complications, nutritional state, and dietary intake. Late evening snacks, BCAA, zinc, vita-

min and mineral supplementation, medium chain triglycerides, vegetable protein and probiotics are considered for effective nutritional supplement. Supplementation of vitamins and minerals should be considered.

Conclusion

Indian diets are diverse however lack the proper nutritional requirements due the imbalance of consuming high carbohydrates and low proteins. This pattern is consistent in all states and income categories. Indians consume too many cereals and too few proteins, fruits, and vegetables. Importantly, unlike many other countries, India does not have a problem with excessive animal protein consumption. Indian officials must speed up efforts to make healthier, more sustainable diets more affordable, accessible, and acceptable across the entire food system. Treatment should target on maintaining adequate protein and caloric intake and rectifying nutrient deficiencies.

Nutritional therapy should be designed to encourage consumption of frequent small meals and a late evening snack to reduce protein breakdown. In cases where oral intake is insufficient, early implementation of enteral feeding should be considered. Malnutrition is a potentially reversible condition and when identified early and treated appropriately, can lead to improved outcomes. Nutritional protein supplements should be advised to achieve the daily recommended intake of protein as Indian diet whether vegetarian or non-vegetarian provides inadequate protein.

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