



Research articles

Nutritional approaches towards prevention and treatment of DCLD

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ABSTRACT

Decompensated chronic liver disease (DCLD) is also known as decompensating cirrhosis. Cirrhosis is a chronic liver disease that is commonly resulting of hepatitis or alcohol use disorder. It is the severe scarring of liver seen at the terminal stages of chronic liver disease. The diet of patients with chronic liver disease is based on a standard diet with supplements addition as necessary. Restrictions may be harmful and should be individualized. In this study we detailed a patient having decompensated chronic liver disease and observed all require parameter in dietary management. The patient undertook a dietary counselling for 16 days and dietary modification was done according to the patient condition. The HB level was 9.1g/dl, so beetroot juice in the mid-morning and soybean and 2 egg whites were suggested to increase the protein level. Later it was seen that Hb level was increased to 9.9g/dl and protein level was increased to 6g/dl. Also, the potassium level was below normal, so coconut water suggested. what to avoid and what to include and a sample menu and a diet chat was given to the patient at the time of discharge. Malnutrition is a potentially reversible condition that, when identified and treated appropriately, can lead to improvement of the outcomes of patients with DCLD.

Keywords: DCLD, decompensate cirrhosis, potassium, haemoglobin, prevention and treatment.

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INTRODUCTION

Numerous patients with DCLD have a diminished food admission, frequently because of HE and digestive symptoms such as anorexia, nausea, early satiety (once in a while identified with micronutrients lacks, like zinc or magnesium, that may cause dysgeusia), gastroparesis, ascites or expanded leptin levels. ^[1,2] DCLD patient require more energy and protein in their eating regimen. A fatty, high protein, low fat eating regimen is recommended.

The eating regimen of patients with DCLD depends on a standard eating routine with supplements expansion as essential ^[3] truth be told, as a rule it is feasible to give a for all intents and purposes ordinary eating regimen. ^[4] Limitations might be hurtful and ought to be individualized. The treatment objectives are to improve the degree of PCM, to guarantee a satisfactory measure of supplements, to accomplish a positive nitrogen balance and to keep away from hepatotoxic agents. ^[5-7] Early amendment of dietary insufficiencies improves long-term prognosis. ^[8]

Food ought to be well cooked, given the patient's expanded weakness to diseases, and ought to be conveyed in 5–7 small daily meals to forestall protein over-burden and nausea/vomiting. Timetable might be a higher priority than the measure of food

ingested, in light of the fact that during the postprandial period there is a suppression of protein corruption for blend incitement. Expanding postprandial period can improve the patient's condition. ^[9-12]

A late evening food positively affects the nitrogen balance, builds bulk by turning around sarcopenia, can improve quality of life, decrease the seriousness and recurrence of HE and increment endurance ^[13]. Accordingly, it is prescribed to limit for the time being fasting period, to try not to quick periods longer than 6 h and decrease the catabolism rate. In spite of the fact that information on this subject is scant, patient's consistence might be troublesome in light of the fact that this delayed dinner can deteriorate reflux complains, debilitate sleep quality and cause glucose intolerance. ^[14] In this investigation, we clarified subtleties sustenance and diet like what to be kept away from, and food varieties to be incorporated relying upon various biochemical boundary in a DCLD patient.

MATERIAL AND METHOD

This is an observational analysis carried out at Institute of Medical Sciences and Sum hospital, Bhubaneswar. The patient history was given below

Prognosis

The patient undertook a dietary counselling for 16 days and

dietary modification was done according to the patient condition. The patient was taking food from the central therapeutic kitchen. The patient had improvement in the biochemical parameters and also in his behavior, so the prognosis is good.

Age	:69yrs
Sex	: Male
Date of admission	:4/2/21
Height	: 169 cm
Weight (at the time of admission)	: 58kg
Weight (at the time of discharge)	: 60kg
BMI (at the time of admission)	: 24.1kg/m ²
BMI (at the time of discharge)	: 24.9kg/m ²
Chief complaints	: Abdominal distress
Diagnosis	: DCLD, mild ascites
Past disease	: T2DM, HTN
Bad habits	: Alcoholic
Previous operation	: None
Family history	: Nil
Any allergy	: Nil

Observation and results

Diet High calorie, High Protein, Low sodium, low fat, moderate CHO, Normal Diabetic diet.

- Diet was given to him from the therapeutic kitchen.
- On 6/2/21 the menu of diabetic diet of calorie - 1800 kcal, Protein-75g, and fat - 23g was provided.
- As the HB level was very low, iron rich foods like beetroot, turnip, raw banana, carrot, beans were provided
- On 9/2/21 with the regular therapeutic diet two boiled egg was suggested to complement the protein deficiency. In the report of 10/2/21 it was seen that the albumin level was increased to 3.3g/dl
- On 11/2/21 the patient was diagnosed with mild ascites so a low sodium diet was suggested.
- On 12/2/21 the HB level was 9.1g/dl, so beetroot juice in the mid-morning and soybean and 2 egg whites were suggested to increase the protein level. In the report of 14/2/21 it was seen that Hb level was increased to 9.9g/dl and protein level was increased to 6g/dl.
- On 16/2/21 raw banana fry was suggested to increase the Hb level.
- In comparison to other days the patient was more active.
- In the report of 17/2/21 it was seen that the potassium level was below normal so was provided
- At the time of discharge, dt 20/2/21 all his report was up to the mark. He was much active and happy. He and his family members were made understood about the diet, what to avoid

and what to include and a sample menu and a diet chat was given to the patient at the time of discharge.

Table 1: Biological Parameters

Parameters	Value at the time of admission	Value at the time of discharge	Normal value
HB	8.1	13.9	13-17g/dl
Albumin	2.1	4.1	3.3-5.2g/dl
Total protein	5.1	6.5	6-8.3g/dl
Sodium	141	139	135-145mEq/L
Potassium	3.6	4.1	3.5-5.0 mEq/L
Bilirubin direct	0.5	0.31	0.1-0.4 mg/dl
FBS	286	210	70-100 mg/dl
ALP	99	89	40-129mg/dl

SAMPLE MENU

Calorie: 1800kcal, CHO: 230 G, Protein: 75g, Fat: 23g

Table 2: Sample menu given at discharge

MEAL	MENU
Early morning	Tea (skimmed milk) +Biscuit
Breakfast (9am)	Veg Suji upama -1 cup, matar curry -1 small katori
Mid-morning (11am)	Egg white (2) badam fist 1 cup
Lunch (1pm)	Parboiled rice (1/2 bowl), Roti (2), Dal (1cup), Soyabeen curry/Santula/ bhaji veg, / stewed chicken curry-2-3npsc
Snacks (5pm)	Boiled chana with kakudi and tomato-1 small katori besanchila-2 nos with pudina chutney.1-2 tsf
Dinner (8pm)	Roti(3),rajma curry/santula/dalma/tadaka/papaya buta curry/-1 middle katori

DISCUSSION AND CONCLUSION

Decompensated cirrhosis is a continuous reason for admission to the acute medical unit, and such patients regularly have complex clinical necessities that can prompt a drawn-out emergency clinic stay and a significant risk of an in-hospital death (10–20%). Decompensated cirrhosis is characterized as an intense weakening in liver capacity in a patient with cirrhosis and is described by jaundice, ascites, hepatic encephalopathy, hepatorenal disorder or variceal drain. Basic precipitants of hepatic decompensation incorporate contaminations, gastrointestinal (GI) dying, high liquor admission/liquor related hepatitis or medication initiated liver injury albeit no particular reason is found in around half of cases ^[15] So, It is essential to attempt to decide the hidden reason for hepatic decompensation through a cautious history, assessment and examinations so suitable treatment can be given. Diet, as we see assumes an essential part for our investigation.

Addressing to the patient's dietary requirements is vital in decompensated liver disease, as sarcopenia is exceptionally predominant. All patients ought to have a wholesome appraisal, food diagram and, whenever required, oral/nasogastric nutritional supplements expecting to give an all-out energy admission of around 35–40 kcal/kg daily.^[16] Refeeding disorder is a typical entanglement, so phosphate, potassium and magnesium ought to be observed day by day, and electrolytes supplanted orally or intravenously as proper. Pabrinex (intravenous thiamine) ought to be endorsed if there is proof of inadequate nutrition or in patients who devour unnecessary liquor, to decrease the danger of Wernick's

encephalopathy [17].

Decompensated cirrhosis or DCLD is a typical problem introducing to intense clinical units and has high mortality. A systematic approach, with the guide of a consideration group, can guarantee patients receive appropriate management of their complications. Most patients with CLD can have an essentially ordinary eating routine with supplements expansion as vital. Limitations might be hurtful and ought to be individualized. Treatment management ought to be founded on keeping up sufficient protein and caloric intake and right supplement deficiencies. The intaking of frequent little dinners and a late-night food can decrease protein breakdown. A maintained negative nitrogen balance through protein restriction leads to an increase in PCM and should be prevented. Protein restriction can aggravate malnutrition and is not necessary except in cases of HE unresponsive to optimized therapy.

CONFLICT OF INTEREST

No conflict

REFERENCE

1. Thuluvath P, Triger D, 1989. Autonomic neuropathy and chronic liver disease, *Q J Med*, 72, 737-747.
2. Quigley E, 1996. Gastrointestinal dysfunction in liver disease and portal hypertension, *Dig Dis Sci*, 41, 557-561.
3. O'Brien A, Williams R, 2008. Nutrition in end-stage liver disease principles and practice, *Gastroenterol*, 134, 1729-1740.
4. Mesejo A, Juan M, Serrano A, 2008. Cirrosis encefalopatía hepáticas consecuencias clínico-metabólicas y soporte nutricional *Nutr Hosp*, 23, 8-18.
5. Amodio P, Bemeur C, Butterworth R, Cordoba J, Kato A, Montagnese S, 2013. The nutritional management of hepatic encephalopathy in patients with cirrhosis International Society for Hepatic Encephalopathy and Nitrogen Metabolism Consensus *Hepato*, 58, 325-336.
6. McGhee A, Henderson J, Millikan W, Bleier J, Vogel R, Kassouny M, 1983. Comparison of the effects of hepatic-aid and a casein modular diet on encephalopathy, plasma amino acids, and nitrogen balance in cirrhotic patients *Ann Surg*, 97, 288-293.
7. Diehl A, Boitnott J, Herlong H, Potter J, Van Duyn M, Chandler E, 1985. Effect of parenteral amino acid supplementation in alcoholic hepatitis *Hepato*, 5, 57-63.

8. Cabre E, Gonzalez-Huix F, Abad-Lacruz A, Esteve M, Acero D, Fernandez-Bañares F, 1990. Effect of total enteral nutrition on the short-term outcome of severely malnourished cirrhotics, A randomized controlled trial, *Gastroenterol*, 98, 715-720.
9. Plank L, Gane E, Peng S, Muthu C, Mathur S, Gillanders L, 2008. Nocturnal nutritional supplementation improves total body protein status of patients with liver cirrhosis a randomized 12-month trial *Hepato*, 48, 557-566.
10. Fukushima H, Miwa Y, Ida E, Kuriyama S, Toda K, Shimomura Y, 2003. Nocturnal branched-chain amino acid administration improves protein metabolism in patients with liver cirrhosis comparison with daytime administration *J Parenter Enteral Nutr*, 27, 315-322.
11. Miwa Y., Shiraki M., Kato M., Tajika M., Mohri H., Murakami N., 2000. Improvement of fuel metabolism by nocturnal energy supplementation in patients with liver cirrhosis *Hepato Res*, 18, 184-189.
12. Swart G, Zillikens M, Van Vuure J, van den Berg J, 1989. Effect of a late evening meal on nitrogen balance in patients with cirrhosis of the liver *BMJ*, 299, 1202-1203.
13. Zillikens M, van den Berg J W, Wattimena J, Rietveld T, Swart G, 1993. Nocturnal oral glucose supplementation, The effects on protein metabolism in cirrhotic patients and in healthy controls, *J Hepato*, 17, 377-383.
14. Tsien C, McCullough A, Dasarathy S, 2012. Late evening snack, exploiting a period of anabolic opportunity in cirrhosis, *J Gastroenterol Hepato*, 27:430-441.
15. Moreau R, Jalan R, Gines P, 2013. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis, *Gastroenterology*, 144, 1426-37.
16. Runyon BA, 2009. AASLD Practice Guidelines Committee Management of adult patients with ascites due to cirrhosis an update, *Hepatology*, 49, 2087-107.
17. Rossi RE, Conte D, Massironi S, 2015. Diagnosis and treatment of nutritional deficiencies in alcoholic liver disease overview of available evidence and open issues *Dig Liver Dism*, 47, 819-25.

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