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Evaluation of Screening for Malnutrition Risk Assessment of Chronic Liver Disease Inpatients

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ABSTRACT

Background: Malnutrition is common in all stages of chronic liver disease (CLD). Its early detection can improve liver function; and reduce the risk of nosocomial infection, morbidity and mortality. Although malnutrition has been well understood and appreciated, it is nevertheless prevalent in hospitals because nutritional status is often overlooked in favor of pressing acute complications. The present study aimed to evaluate screening for malnutrition risk assessment (MRA) in CLD inpatients of Theodor Bilharz Research Institute (TBRI). **Materials and methods:** A hospital-based cross-sectional retrospective analytical study was conducted to assess health records for MRA activities provided to CLD inpatients, men (n=61) and women (n=39). CLD severity was determined by Child–Pugh model for end-stage liver disease. The MRA checklist was created with reference to the 2002 European Society of Parenteral and Enteral Nutrition (ESPEN) standards for nutritional therapy in hospitals. **Results:** The present study revealed the absence of an organized schedule for diagnoses of MRA; in favor of managing pressing CLD complications starting with the absence of history taking and anthropometric measurements. Only apparent malnutrition signs such as ascites, lower limb edema, and Sodium retention were detected. Also, there is a lack of a lab investigation for subclinical signs and lab of protein-energy malnutrition, and macro and micronutrient deficiencies.
Conclusions: Supplementation of vitamins and micronutrients was empirically prescribed. Despite the common association of CLD patients with malnutrition, there was an absence of clearly stated MRA schemes and delegated responsibilities.

Keywords: Malnutrition Risk Assessment (MRA), Nutrition Risk Screening (NRS), chronic liver disease

1. Background

Malnutrition is common in patients with chronic liver diseases and is an important prognostic factor affecting quality of life, outcomes, and survival (Bémeur *et al.,* 2010). Malnutrition is a clinical syndrome that arises from an excess, deficiency, or imbalance of nutrients that alters body composition and impairs mental and physical abilities (Cederholm *et al.,* 2017).

One important organ where numerous protein, carbohydrate, and fat metabolic processes occur is the liver. Hence, it is in charge of preserving a healthy nutritional state. Fat malabsorption, deficiencies in fat-soluble vitamins, a drop in the amount of water-soluble vitamins, and modifications to micronutrient metabolism are all present in the advanced stage of CLD. Consequently, malnutrition

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and muscular dystrophy result from this organ's malfunction (Shergill *et al.,* 2018). For patients with liver disease, diet and nutritional status are critical therapeutic and prognostic factors (Bischoff *et al.,* 2020). Chronic liver disease is commonly associated with micronutrient deficiency. Rates of vitamin A, selenium, vitamin D and zinc deficiency have been observed to be as high as 85-95% in patients with decompensated liver disease. There is a relationship between the degree of a micronutrient deficiency and the severity of liver disease, as well as the chance of complications and death related to CLD (Ibrahim *et al.,* 2023).

Protein-calorie malnutrition (PCM) can be found in 65–90% of patients with advanced disease and is common in all stages of CLD (Campillo *et al.,* 2003). Patients with cholestatic liver disease are vulnerable to calorie deficits and run a higher risk of running low on fat-soluble vitamins (Zaina *et al.,* 2004). Although hospital malnutrition is common, nutritional care is frequently given little attention. Nutrition Risk Screening (NRS) 2002 is a validated tool to screen hospitalized patients for risk (Bischoff *et al.,* 2020; Puri *et al.,* 2021).

The European Society of Clinical Nutrition and Metabolism (ESPEN) has developed guidelines to improve the prescription of hospital diets to increase the quality of nutritional routines, lower the risk of malnutrition and achieve good patient safety in nutritional care, to provide advice as possible regarding the diets required in rehabilitation centers, nursing homes, hospitals, and their specific management of diet supply and the indications (Holst *et al.,* 2014; Thibault *et al.,* 2021). Early detection of micronutrient or macronutrient deficits is crucial to minimize the risk of infection and in-hospital mortality. Patients with liver disease should undergo a validated tool-based malnutrition screening (Bischoff *et al.,* 2020). An essential component of nutritional assessment is the evaluation of food energy intake (Jensen *et al.,* 2019). The main cause of hospital malnutrition is a reduction in nutritional intake along with an increase in energy requirements. Therefore, to decrease the incidence of hospital malnutrition, it has been highly advised that mandatory nutrition screening be widely introduced (Sam and Nguyen, 2009).

The current study aimed to screen the malnutrition risk assessment (MRA) of CLD inpatients of the Department of Hepatology and Gastroenterology in Theodor Bilharz Research Institute (TBRI), according to ESPEN guidelines of nutritional care in hospitals.

2. Subjects & Methods

The present work was a hospital-based cross-sectional retrospective analytical study.

Samples

A total of 100 inpatients, 61 men and 39 women, were examined for the provision of MRA screening. CLD inpatients' files were recorded by a detailed nutritional assessment checklist designed to detect metabolic, nutritional, or functional manifestations of undernutrition. The checklist was specifically designed according to hospital ESPEN guidelines of NRS 2002 (McClave *et al.,* 2016; McClave *et al.,* 2014; Warren *et al.,*2016; Silva *et al.,* 2015; Lalama and Saloum, 2016; Correia *et al.,* 2017; Gaikwad *et al.,* 2016; Fernandes *et al.,* 2016).

Ethical clearance

This study was done following the ethical standards of the responsible Committee on Human Experimentation and with the Helsinki Declaration. The study protocol was granted TBRI Ethical Committee approval number (FWA 00010609).

Data management and statistical analysis

Version 15 of the Statistical Package of Social Science was used to process the data. Simple frequencies were utilized to verify the data, and descriptive statistics were employed to summarize the data, and bi-variant relationships were displayed in cross-tabulation to perform tests of significance for all the results P value was significant <0.05, and the corrected P-value was used for values less than 5%.

3. Results

The results of this study revealed, that the mean age group among males was 51.5±12.28 whilst among females 50.9±26.25, p=0.5 revealing statistical insignificance between both sexes (Table 1).

Table 2 shows that CLD was commonest among male age group 50- years (32.8%) and >60 years among females (35.9%), with statistical insignificance between both sexes. The most frequent presenting signs were edema, either lower limb or lower abdominal wall; 32.7% among males and 38.5% among females, with statistical insignificance between both sexes (Table 3).

Table 1: Age and sex characteristics of the studied groups.

Age	Sex	
	Men	**Women**
Total	61	39
Mean ± SD	51.5±12.28	50.9±16.25
Median	52.5	52
Minim	20	20
Maximum	77	83

Table 2: Age-gender distribution.

Age	Sex				Total
	Men		**Women**		
Total	61	100%	39	100%	%
<20	1	1.6	1	2.56	2
20-	5	8.2	3	7.7	8
30-	4	6.5	3	7.7	7
40-	12	19.7	8	20.5	20
50-	20	32.8	10	25.6	30
>60	19	31.1	14	35.9	33

Table 3: Distribution of the study group according to clinical signs.

Sign	Sex				Total
	Men		**Women**		
Total	61	100%	39	100%	%
Jaundice	19	31.1	11	28.2	30
LL, LAW edema	20	32.7	15	38.5	35
GIT bleeding	15	24.5	7	17.9	22
Oriented	61	100	39	100	100
H.encephalopathy	0	0	0	0	0

Table 4 showed that chronic viral hepatitis with liver cirrhosis and ascites were 67% and 66% respectively, with no statistical significance between both sexes. Table 5 revealed the absence of Nutrition Risk Screening (NRS) 2002 as a specific tool for the assessment of malnutrition. Limiting disease severity and risk assessment to Child-Pugh score. Also detailed nutrition history was completely absent in CLD patients' files (Table 6) and the anthropometric measurements from patients' files (Table 7) were limited to the body weight.

Table 4: Chronic liver disease diagnosis.

CLD	%
Chronic viral hepatitis	79
- Cirrhosis	67
- Portal hypertension	31
- Ascites	66
Hepatocellular carcinoma	8
Primary Biliary Cirrhosis	15
Sclerosing Cholangitis	3

Table 5: Nutrition risk screening (nrs) 2002.

	Nutritional status score	Done	Not
Step I	1. Is BMI < 20.5	0	100
	2. 10% unintentional weight loss in the last 3months	0	100
	3. Decreased dietary intake in the last week	0	100
Step II	Disease severity score Child-Pugh score ICU, Age >70	100	0
Total score	Nutritional status score + Disease severity score	0	100
Age adjusted total score	if age ≥ 70 years add 1 to the total score above	0	100

Table 6: Performance of detailed nutritional history.

Detailed nutritional history	Done	Not
Personal habits		
• Number of meals and snacks	0	100
• Preferred foods	0	100
• Non-preferred foods	0	100
• Smoking	0	100
Gastrointestinal assessment		
• Dentition	0	100
• Swallowing	0	100
• Bowel function, early satiety, distention, flatulence, constipation, diarrhea	0	100

Table 7: Performance of anthropometric measurements of nutrition status.

Anthropometric measurements	Done	Not
Weight (kg)	7	93
• actual, usual, ideal	0	100
• Weight changes	0	100
Height (cm)	0	100
Body mass index	0	100
Skinfold thickness	0	100
Functional consequences of under nutrition		
Hand Grip	0	100
Body composition bio-impedancemetry	0	100
DEXA scan	0	100
CT scan	0	100

Table 8 reveals that clinical examination for assessment of signs of malnutrition was limited to ascites (66%), lower limb edema (35%), and sodium retention (35%). Meanwhile absence of signs of other signs of protein and energy undernutrition, and vitamin, and micronutrient deficiencies were absent.

Table 8: Performance of clinical examination of malnutrition.

Signs of malnutrition:	Done	Not
Protein and energy undernutrition:		
• Muscle wasting of quadriceps and deltoids	0	100
• Ascites	66	34
• Lower limb edema	35	65
• Subcutaneous fat	0	100
Vitamin deficiency:		
Fat soluble		
• Vitamin A	0	100
• Vitamin D	0	100
• Vitamin K	0	100
Water soluble		
• Vitamin Thiamin B_1,	0	100
• Vitamin B_6,	0	100
• Vitamin B_{12}	0	100

• Folate	0	100
Minerals disorders		
• Sodium retention	35	65
• Zinc deficiency	0	100
• Iron deficiency anemia	0	100
• Hemochromatosis	0	100

The only laboratory investigations of nutritional assessment present among the studied files were serum albumin and sodium (Table 9). On the contrary, prealbumin, vitamins and micronutrients, glycated hemoglobin HbA1c, and triglycerides assessment were absent in all files.

Table 9: Performance of laboratory investigations.

Tests	Done	Not
Total protein	100	0
Prealbumin	0	100
Albumin	100	0
Vitamin D	0	100
Retinol	0	100
Vitamin K	0	100
Vitamin E	0	100
Thiamin	0	100
B_6	0	100
B_{12}	0	100
Folate	0	100
Zinc	0	100
Selenium	0	100
Magnesium	7	93
Sodium	100	100
Potassium	0	100
Iron		
Hemoglobin	79	21
Ferritin	0	100
Transferrin	0	100
Serum glucose		
Fasting blood glucose	42	58
Random blood glucose	80	20
Hb $A1_C$		
Triglycerides	0	100
Total Cholesterol	1	99
High density lipoprotein (HDL)	0	100
Law density lipoprotein (LDL)	2	98

Table 10 showed that the CLD hospital diet was more frequently modified by nurses than by physicians; 71% and 37% respectively. Supplementation among CLD Patients was 60% vitamins and 12% micronutrients.

Table 10: Dietary modification and supplementation.

Dietary modifications	Done	Not
By nurses	71	29
By physicians	37	63
Micronutrients supplementation		
Vitamins	60	40
Minerals	12	88

4. Discussion

Choosing NRS 2002 in the present study goes with; Kondrup *et al.* (2003) who stated that the NRS-2002 system's goal is to identify undernutrition and the possibility of it developing in a hospital

setting (Kondrup, *et al.,* 2003). Additionally, it is intended to cover all potential patient categories in a hospital as a patient with a given diagnosis does not always fall under the same category. It includes the nutritional components of MUST, a grading of disease severity as a reflection of increased nutritional requirements (Kondrup, *et al.,* 2003). Moreover, through the use of an ESPEN ad hoc working group overseen by the ESPEN Educational and Clinical Practise Committee, its content validity was increased (Kondrup *et al.,* 2003). In a two-year implementation study conducted at three hospitals in Denmark (local, regional, and university hospitals), it was also utilized by nurses and dietitians (Rasmussen *et al.,* 2010).

Table 2 shows that age was stratified into ten-year interval groups from <20, 20-, up to >60. This revealed that the age of the study group of CLD patients was mainly50- years among males (32.8%) and >60 years among females (35.9%). This goes in line with Henkel & Buchman who mentioned that malnutrition is not typically a complication of acute liver injury, but manifests with progression to liver failure (Henkel and Buchman, 2006).

In the present study, table 3 showed that the most frequent clinical signs in CLD patients were edema, either in the lower limb or a lower abdominal wall (32.7% and 38.5% among males and females respectively) and GIT bleeding (24.5 and 17.5 among males and females respectively). Past research has shown that liver disease causes a progressive alteration in whole-body bioelectrical parameters. The region most impacted by the liver disease's progression was the lower extremities. Males also showed lower resistance and reactance values than females in measurements of the arm, leg, and trunk in addition to the entire body (Panella *et al.,* 1995).

Prolonged prothrombin time or partial thromboplastin time is an example of coagulopathy, and poor hepatic synthetic function and hypersplenism are the causes of thrombocytopenia in many patients with chronic liver disease (Jensen, 1996).

The dominant clinical findings associated with chronic viral hepatitis were cirrhosis and ascites in both sexes (Table 4). According to reports, CLD is characterized by an ongoing process of liver parenchymal inflammation, destruction, and regeneration that results in cirrhosis and fibrosis (Mandato *et al.,* 2017). Protein-energy malnutrition is linked to chronic liver disease at all stages, so patients with the condition should follow a normal diet and takes supplements as needed (Silva *et al.,* 2015).

The primary reasons for insufficient nutritional care were a lack of guidance on how to handle these issues, a lack of fundamental understanding of dietary requirements, and practical aspects of the hospital's food service (Kondrup *et al*., 2002). also stated that patients with cirrhosis who are malnourished are twice as likely to have refractory ascites (Serón-Arbeloa *et al*., 2022).

The results in table 5 revealed the lack of use of Nutrition Risk Screening (NRS) 2002 as a specific tool for the assessment of malnutrition. Hence, disease severity and risk assessment were limited to Child-Pugh score, which is considered to be incomplete; as noted by Rasmussen *et al.* (2010) that The NRS 2002 system was created under the presumption that the severity of undernutrition and the rise in nutritional needs brought on by the disease serve as indicators for nutritional support, i.e., It may be necessary to provide nutritional support if there is severe undernutrition, severe illness, or any combination of the two (Serón-Arbeloa *et al*., 2022). This will also include patients who are not undernourished at the time but are at risk of becoming so because of disease and/ or its treatment (Kondrup *et al.,* 2003; Rasmussen *et al.,* 2010).

Also, detailed nutrition history (Table 6) was completely absent in CLD patients' files and the anthropometric measurements from patients' files (table 7) were limited to the body weight. Previous studies recorded that CLD was associated with a common picture of malnutrition and differences in body composition (Traub *et al.,* 2021). Abdominal bloating and abdominal pain were recorded as common symptoms (80% of patients) in liver cirrhosis (Sam and Nguyen, 2009) [12] and associated with weight loss (Yasutake *et al.,* 2018).

The results showed the absence of some important Anthropometric parameters for the evaluation of malnutrition in CLD patients (Tables 5 &7). Malnutrition is associated with poor survival rates in cirrhotic patients (Alberino *et al.,* 2001). Early detection of malnutrition in CLD patients is of great importance for patient recovery (Barbosa-Silva and Barros, 2005, Barbosa-Silva *et al.,* 2005). Two approaches are suggested for the diagnosis of malnutrition by the most recent European Society of Clinical Nutrition and Metabolism (ESPEN) consensus: A body mass index (BMI) of less than 18.5 kg/m2 is the first indicator of malnutrition. Second, the combination of low

BMI or low fat-free mass index (FFMI) (less than 15 or 17 kg/m2 in females or males, respectively) and unintended weight loss (more than 10%) (Cederholm *et al.,* 2015). However, a variety of additional instruments, including laboratory tests and anthropometric and non-anthropometric approaches, may be employed to categorize the severity of malnutrition as mild, moderate, or severe in various ways. Some nutritional anthropometry metrics include total lymphocyte count, serum albumin level, triceps skinfold thickness (TSF), hand grip, and mid-arm circumference (Barbosa-Silva *et al.,* 2005; Cederholm *et al.,* 2015; Periyalwar and Dasarathy, 2012). It was found that electrical bioimpedance (BIA) has been proposed as a body composition tool that has demonstrated good results regarding the nutritional state. In both healthy subjects and patients with cirrhosis, BIA shows total body water as well as fat mass, lean mass, and basal metabolic rate (Kyle *et al.,* 2004; Ellis 2000).

The clinical examination to assess signs of malnutrition (Table 8) was limited to ascites, lower limb edema, and sodium retention. Also, the laboratory investigation of those patients (Table 9) showed that serum albumin and sodium were the only performed tests among nutritional assessments in the studied files. This explains that clinical examination is directed towards staging CLD for prognosis or to reveal decompensating liver functions. This is opposed to McCullough and Tavill, 1991 who cited that malnutrition is an independent risk factor for predicting clinical outcomes in patients with liver disease (McCullough and Tavill, 1991).

Vitamins are vital micronutrients with a variety of biological applications. It is reasonable to assume that chronic liver diseases would affect these processes as the liver is where many crucial phases of their metabolism, storage, and activation take place. It appears that many liver diseases are associated with decreased vitamin levels. Due to their significance, it's critical to evaluate the consequences of vitamin deficiencies and determine whether vitamin supplements may be therapeutically useful in the treatment of certain hepatic conditions (Bjelakovic *et al.,* 2021).

Vitamin A metabolism is regulated by the liver which produces retinol-binding protein 4 (RBP4) essential for its distribution in peripheral tissues (Licata *et al.,* 2021) [43]. Vitamin A deficiency was highly correlated with higher rates of decompensated liver disease, high Child-Pugh score, and portal hypertension. (Simbrunner *et al.,* 2020). Vitamin D was reported as an anti-inflammatory mediator (Koop *et al.,* 2018; Gad *et al.,* 2020; Kubesch *et al.,* 2018; Stokes *et al.,* 2014). Its deficiency has been correlated with liver disease complications, liver scoring methods including Childs-Pugh score, portal hypertension, and increased overall mortality (Kubesch *et al.,* 2018; Stokes *et al.,* 2014; Finkelmeier *et al.,* 2015; Paternostro *et al.,* 2017; Zhang *et al.,* 2021).

Children with chronic cholestasis can have their neurological abnormalities reversed by vitamin E supplementation, but severe vitamin E deficiency makes it difficult to treat, especially in adults with advanced liver disease who need liver transplantation (Jeffre *et al.,* 1987).

Low vitamin K levels are common in liver disease patients for two reasons: reduced absorption in cirrhosis patients and dietary deficiencies. Furthermore, there is a lack of functional interaction between the liver cells and vitamin K in cirrhosis or acute liver failure (Gish *et al.,* 2021).

Data is currently sparse regarding the role of thiamine (B1), B9 and vitamin K nutritional status in patients with liver disease. Eight water-soluble components make up the B-vitamin group. These constituents function as co-enzymes in a wide range of catabolic and anabolic enzymatic reactions, fulfilling vital and closely related roles (Licata *et al.,* 2021), additionally, the group serves as a bridge in the biosynthesis of several important substances, such as pyrimidines, fatty acids, and amino acids. The eight components are cobalamin (B12), biotin (B7), biotin (B6), pyridoxine (B6), pantothenic acid (B5), thiamine (B1), riboflavin (B2), niacin (B3), and folate (B9) (Lindschinger *et al.,* 2019). The main source of these vital nutrients in the diet is food (Kennedy *et al.,* 2016). Many vitamin B coenzymes are also involved in cellular functions, including the vital "methionine cycle" and the "folate cycle," which are both necessary for cell viability (Stanger and Wonisch, 2012).

According to reports, zinc is necessary for healthy tissue growth and repair. Moreover, it is known to have immune-regulating, anti-inflammatory, and antioxidant properties (Bloom *et al.,* 2021; Grüngreiff *et al.,* 2016). Zinc homeostasis and metabolism are primarily carried out by the liver, so a zinc deficit is linked to liver disease.

Numerous guidelines, such as those from the American Association for the Study of Liver Diseases (AASLD) and the European Society for Clinical Nutrition and Metabolism (ESPEN), established the guidelines for micronutrient replacement therapy in patients with clinically suspected

deficiencies and confirmed the high prevalence of micronutrient deficiency in patients with chronic liver disease (Jensen *et al.,* 2019; McClave *et al.,* 2009).

Data in table 10 showed that the CLD hospital diet was more frequently modified by nurses than by physicians; 71% and 37% respectively. Only, 60% and 12% of cases were supplied by vitamins and minerals respectively. Malnutrition is a significant clinical problem that affects 20–50% of hospitalized patients globally (Mandato *et al.,* 2017, Silva *et al.,* 2015). Malnutrition lengthens hospital stays and raises the risk of complications and death (Smart *et al.,* 2011; Purnak and Yilmaz, 2013). Patients' dietary intake is influenced by a complex interplay of factors, including those related to the patient, the hospital setting, and the nutrition care they receive (Mendenhall *et al.,* 1984). Patients should receive special attention and special nutrition programs. In this sense, by offering nutrition programs with the proper follow-up, nurses can significantly contribute to improving the quality of nutrition received by patients.

4. Conclusion & Recommendations

Nutritional goals for patients with chronic liver disease (CLD) should be to prevent or treat related comorbidities, improve liver function, and prevent malnutrition from inadequate nutrition. They need to eat a normal diet plus supplements as needed, as all stages of chronic liver disease are linked to the condition of protein-energy malnutrition. By preventing malnutrition, promoting a catabolic state, increasing the amount of branched-chain amino acids in the diet, controlling or preventing ascites and edema, and limiting sodium, potassium, and fluid intake, nutrition therapy is used to enhance the quality of life. Every patient with CLD undergoes screening to determine whether they are susceptible to preventable complications. Finally, though, malnutrition is a curable condition that can help patients with chronic liver disease if it is identified and treated appropriately.

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Conflicts of interest
None

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Author Contributions

WW put the design and write of the paper. All authors discussed the methodology and conclusion and contributed to the final paper. MA did the practical section. AS supervise the work, ZAS manage and correct the paper, ER corrected and submit the article. All authors have read the paper and approved it.

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