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A Comparative Assesment Of Nutritional Status In Cirrhosis Of Liver By Anthropometry And RFNP-Tool In Indian Population

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ABSTRACT

Background and Aim: Patients of cirrhosis of liver are at risk of malnutrition with associated increased mortality and morbidity due to multiple reasons. But inadequate attention is given on this aspect in these patients.

Methods: 219 newly diagnosed cirrhotics of different stages were examined in a prospective observational study whose nutritional status was assessed by Royal Free Nutritional Prioritising Tool and anthropometry . Serum albumin , total lymhocytic count and total cholesterol were also assessed Students' unpaired t tests by Chi-square and ANOVA tests were used as indicated.

Results: Prevalence of malnutrition in cirrhosis in this study was 90%. 51% of alcoholic compared to 33% non-alcoholic cirrhosis had some form of malnutrition (p = 0.0015). Males and females had significant difference (p= 0.0476). Comparing the methods of assessment between RFH-NPT and anthropometric parameters, no statistical difference was found but a positive correlation existed between them and the severity of cirrhosis. Lymphocyte count, serum albumin and cholesterol differed significantly between the cases with and without malnutrition irrespective of its degree. Clinical signs of micronutrient deficiency was an additional association.

Conclusion: Severe malnutrition is common in liver cirrhosis, being significantly higher in alcoholics. It increases with the CP grade. Unlike in other Asians, fat loss is significantly less in females compared to the males who demonstrated higher protein loss. Clinical signs of micronutrient deficiency was a significant association. Both Anthropometry and RFH-NPT are equally effective in assessing nutrition. Comparatively easy to perform, RFH-NPT tool is more practical in clinical setting. This will enable us to implement a sufficient oral diet to correct protein and calorie deficits in cirrhotics to reduce morbidity, mortality and cost of treatment.

KEYWORDS : malnutrition, nutritional assessment, liver cirrhosis, nutritional deficiency

INTRODUCTION

Malnutrition is associated with poor prognosis of cirrhotic patients.[1],[2] It results in higher complication rates like ascites, encephalopathy, infections and kidney injuries. These often lead to increased mortality to the tune of 48% *vs* 18% respectively in cirrhotics with and without malnutrition. [8] The clinical features of malnutrition consist of both protein energy malnutrition (PEM) and micronutrient deficiencies. While PEM is characterized by loss of muscle mass and subcutaneous fat resulting in cachexia, micronutrient deficiencies, especially of Vitamin A and D amongst others, are very commonly seen as skin scaling, xerophthalmia, osteomalacia, neuropathies, anemia, glossitis, weakness and fatigue. The degree and prevalence of malnutrition is generally directly proportional to the disease severity, being higher in decompensated cirrhosis.[4]

Assessment of nutritional status in cirrhotics should ideally include measuring energy balance, BMI, body composition and tissue function. The methods employed include systemic dietary recall, food diary, anthropometry, 'bomb calorimetry', indirect calorimetry, liver function tests, serum/urinary creatinine and other electrolytes, bioelectrical impedence analysis etc, some of which are cumbersome and impractical. Studies assessing nutritional status by direct body-composition methods, are therefore scanty. [5]

Many of these indices are affected by the course of the disease rather than the patients' nutritional status which can confound the actual nutritional state of the patients. Hence, anthropometry combined with assessment of body mass has been recommended as a simple way to identify malnutrition in liver disease as per ESPEN guidelines.[6] The Child–Pugh (CP) and MELD classification do not include nutritional status. Several subjective global assessment (SGA) tools to screen malnutrition has also been devised like the Royal Free Hospital Global Assessment (RFH-GA)- which is the accepted gold standard for nutritional assessment of cirrhotic patients, but is also time consuming and needs a trained personnel.[7] However, although validated in transplant candidates, since SGA includes objective parameters, they are rarely practical in clinical setting. This results in placing less importance to nutritional assessment in cirrhotics, especially in outdoor settings. [8] But a validated SGA tool proposed by Arora *et al* is simpler and less time consuming.[9]

Overall, compared to the west there are few data on the nutritional status of Asian cirrhotic patients.[10] In this background this study was done to asses the prevalence and characteristics of nutritional status in cirrhosis of liver patients.

METHODOLOGY

This was a prospective study done over three years, in Assam Medical College, Dibrugarh, a Government tertiary care teaching hospital in North-East India. Study population consisted of 302 treatment naïve patients of cirrhosis of liver irrespective of etiology attending the OPD or indoor ward, out of which 219 patients were finally included (Male 157, Female 62, Mean age 45.7 ± 11.9 years). Exclusion criteria included associated diabetes mellitus, tuberculosis, HIV, active sepsis, malignancies, past abdominal surgeries, loose motion, vomiting, active gastro intestinal bleeds, comorbidities (like cerebrovascular accidents, cardiac failure, thyroid disorders, biliary, pancreatic, chronic obstructive airway disease, uremia) and alcoholics (>20 gm/day) with positive viral markers. Severity of liver disease was calculated by Child-Pugh (CP) score.[11]

Routine investigations included liver and renal function tests, hemogram, total lymphocyte count, hepatitis viral serology, ceruloplasmin, ferritin, ANA and trans-abdominal ultrasonogram. Detailed history and examination included dietary recall history, alcohol ingestion, past hospitalization, reason for present hospitalization, complications of cirrhosis and drug history were undertaken by direct questioning of relatives and the patients. Patients with history of regular ingestion of >20 gm alcohol/day for atleast one year were considered alcoholic cirrhosis. Micronutrient/vitamin deficiencies were first clinically assessed (skin, mucosa, hair, nail, eye changes) and when deemed necessary, with the help of a qualified dermatologist, ophthalmologist, neurologist and a dietician. Serum estimation of vitamins/minerals were not done. Nutritional assessment was done by Royal Free Nutritional Prioritising Tool (RFH-NPT tool) as described [9] and anthropometry at baseline. The RFH-NPT included 3 steps (whether a transplant candidate, presence of fluid overload, dietary history and BMI) and given a score as described. The result was noted as no malnutrition, moderated malnutrition and severe malnutrition (Score 0, 1 and 2-7 respectively).

Anthropometric measurements included midarm circumference

(MAC), triceps skinfold thickness (SFT, a measure of fat stores) and midarm muscle circumference (MAMC, a measure of muscle protein mass).[12] MAC was measured with a measuring tape at the mid point between the tip of acromian and olecranon process to the nearest centimeter in the right arm. SFT was measured to the nearest millimeter at the right arm using Harpenden skinfold caliper (*British Indicators Ltd, Bedfordshire, England*). For SFT and MAC, three measurements were taken before recording the average value. Mid-arm muscle circumference (MAMC) was calculated by the formula: MAMC = MAC - (3.1415xSFT). Severe malnutrition was defined by MAMC and/or TSF <5th percentile and moderate malnutrition by MAMC and/or TSF < 10th percentile.[2],[13] RFH-NPT, MAMC and SFT were done independently by three different observers blinded to each other's findings.

Serum albumin (representing visceral protein reserve), total lymhocytic count (representing immune depression) and total cholesterol (a calorie depletion index) were also assessed since they were found to be good indicators for nutritional assessment of all hospitalized patients.[14]

Statistical analysis: For all qualitative and quantitative datas, Students' unpaired *t* tests by Chi-square and ANOVA tests respectively were used to determine the differences. Contingency tables were used to find out the probability of being malnourished or not by the RFH-NPT, MAMC and TSF methods. The x^2 test was used to find out the association levels between these 3 methods. Statistical significance was assumed at a p value of <0.05.

RESULT

Majority belonged to CP C class (62%) followed by CP B (25%) and A (13%). Most (56%) of them were alcoholic cirrhosis and 46% were non-alcoholic (26% cryptogenic and 13% viral) (Table 1). The overall prevalence of malnutrition in cirrhosis was 90% as assessed by RFH-NPT tool. By MAMC and SFT estimation the prevalence of malnutrition were 91% and 90% respectively (Table 2). While comparing the methods of assessment between RFH-NPT and MAMC, RFH-NPT and SFT, and MAMC and SFT, it was found that there were no statistical difference (p= 0.7432, p=1.0000 and p= 0.7432 respectively). Further interpretation of these p values revealed that the chance of having a p value < 0.7432 by one or more of these three tests had a 98.307% probability, which is significant. While quantifying the interrater agreement for malnutrition among RFH-NPT, MAMC and SFT methods it was found that the number of observed agreements was 35.01% of the observations, and number of agreements expected by chance amounted to 33.33% of the observations with *Kappa*= 0.025 (SE of *kappa* = 0.023 ,95% CI: from -0.019 to 0.070).

Significantly, 51% of alcoholics compared to 33% non-alcoholic cirrhosis had some form of malnutrition (p = 0.0015). The severity of malnutrition was also significantly high in alcoholic cases (p = 0.0032) but not micronutrient deficiency (p= 0.5608), when compared to non-alcoholic cirrhosis (Table 2).

The lymphocyte count, serum albumin and serum cholesterol differed significantly between the cases with and without malnutrition (p< 0.0001, p=0.0429 and p< 0.0001 respectively) and the statistical difference persisted irrespective of the degree of malnutrition (Table 2).

A statistically significant difference was observed between males and females with malnutrition as assessed by MAMC and TSF (p= 0.0476 , Table 3).

Severe malnutrition was significantly higher than moderate or normal nutrition across the study population.

DISCUSSION

Malnutrition is common among cirrhotic patients. The reason is multifactorial and include anorexia, catabolism, increased requirements, restrictive diets (sometimes unnecessarily), maldigestion, malabsorption, and altered nutrient metabolism. Metabolically, a 12 hour fast of cirrhotic patient is equivalent to a 72 hour fast in normal persons.[15] It is difficult to manage nutrition in cirrhotic patients because of alterations in metabolic and storage functions of the liver. In addition to meeting macro and micronutrient requirements, the composition and timing of food supplements also affect the efficacy of nutrition support. In cirrhosis, as the liver is unable to synthesize and/or store adequate amounts of glycogen, glucose is not readily available from body carbohydrates. It leads to a relative starvation where glycerol and amino acids from the body are recruited for neoglucogenesis , leading to a progressive breakdown of body fat and muscle. This results in tissue depletion and muscle wasting.

Nutritional screening should be simple and quick so that appropriate planning can be undertaken for those at risk of malnutrition. The use of traditional assessment tools, such as anthropometric and biometric measures, is often difficult because of presence of associated complications like ascites and infection. Similarly, a calorie count is accurate but it requires a detailed record of meal portions in addition to the skill to calculate calories of food eaten. Therefore, in a hospital setting it rquires a trained nursing staff/dietician. A food diary and food frequency questionnaires require the patient to be sufficiently alert, educated and are usually time consuming to complete. The 24-hour recall is a rapid, simple, low cost method, but again the patient's literacy, memory and level of encephalopathy are confounding factors.[16]

In our cases, the total cholesterol was significantly low in malnutrition group having a positive correlation with CP grade, implying that the calorie intake significantly decreased with severity of the liver disease. In Asia, chronic Hepatitis B infection is the major cause of liver cirrhosis and it's associated complications.[17] But in our study alcohol was the commonest cause, followed by cryptogenic cirrhosis. Malnutrition was significantly more in alcoholics compared to the non-alcoholic cases. Severe malnutrition was also significantly higher in alcoholic cirrhosis. Alcoholism facilitates the development of hepatic injury in several ways. Our study suggests alcohol causes significant nutritional deficiency in cirrhosis compared to non-alcoholic cirrhosis. Similar findings were also reported by other workers from Asia and the west, [10],[18],[19],[20] although some reports found that the prevalence, characteristics, and severity of malnutrition are similar in alcoholic and viral cirrhosis.[21]

Overall, we found 62% of CP –C cirrhotics as having severe malnutrition, followed by 25% of CP-B and 13% of CP-A grade cirrhotics. The degree of malnutrition increases with increasing severity of cirrhosis of liver.[22] TSF and MAMC values therefore decrease significantly according to the CP score and a positive correlation exists between these parameters and the severity of cirrhosis, [23] which was similar to our findings.

In non-hospitalized cirrhotic patients nutritional assessment is rarely done. But 2/3rd of all cirrhotics have some degree of malnutrition; being moderate or severe in 38.3%, and a higher prevalence in CP-C grade compared to CP-A grade.[24]

Previous western studies have documented the malnutrition rates from 20% in compensated cirrhosis to 60% in decompensated cirrhosis,[25] but recent investigators reported rates of ~50% to 90% depending on liver dysfunction.[26],[27] In our study the prevalence of malnutrition was 90% by RHG-NPT, 91% by MAMC and 90% by TSF method. All these three tests are comparable to identify malnutrition, showing a very high probability of correlation. The RFH-NPT is simple, quick and an already validated method. It includes proper dietary history, BMI, presence of edema and clinical examination that can be undertaken easily within a short time as opposed to SGA (subjective global assessment) which is time consuming. We believe, for identifying malnutrition in cirrhotics, this tool may be easier, compared to MAMC and TSF which may be affected by presence of edema and observer variation.

In Asian patients, protein malnutrition of 50% (MAMC < 5thpercentile) and fat mass depletions of 30% (TST <5th percentile) has been reported.[10] But in our series, the loss of muscle and fat did not differ significantly both in malnourished and non-malnourished groups tested by MAMC and TSF, signifying that in our region, the fat and muscle loss occur concurrently in malnutrition in cirrhosis irrespective of the etiology and CP grade.

The difference between the presence, prevalence and degree of malnutrition was significantly more pronounced in alcoholic cirrhosis compared to non-alcoholic cirrhosis in our study. This was also reported in a previous Indian study from Rajasthan.[28] But the same

workers reported a year later that the degree of malnutrition was similar among alcoholic and non-alcoholic liver disease patients.[29] In contrast, a North Indian study found that PEM was more pronounced in nonalcoholic compared to alcoholic cirrhosis, although the degree and profile of malnutrition in chronic alcoholics and in alcoholic cirrhotics were comparable.[30] Malnutrition alone is insufficient to explain the pathogenesis of alcoholic cirrhosis. Other factors, like nutritional deficiencies and genetic susceptibility, may modulate the risk of alcohol induced liver damage, although the daily amount of alcohol consumed and the duration of excessive consumption are additional important factors. Our patients have distinctly different ethnic, food and cultural habits compared to the regions where these Indian studies were performed and hence may explain the differences in our findings. Our population takes a rice based high carbohydrate diet and low protein derived mainly from fish and eggs; while fruits and vegetables are consumed in small amount.

The lymphocyte count differed significantly between malnutrition *vs* no malnutrition groups suggesting the risk of acquiring infections/sepsis and impaired immunity in malnourished cirrhotics. The same was true for serum albumin and serum cholesterol. The differences of these three parameters were statistically significant irrespective of the degree of malnutrition. The serum cholesterol and lymphocyte counts showed a stronger statistical difference compared to serum albumin between the malnutrition group.

Serum albumin concentration is a reliable laboratory marker of nutritional status, especially for visceral protein, and is used to assess changes in nutritional status and the risk of malnutrition. [31] Approximately 80% of visceral protein are diminished in malnourished cirrhotic patients.[32] Malnutrition in cirrhosis increases protein catabolism with overall cellular protein breakdown, mainly in skeletal muscles, and a positive correlation between low serum albumin and skeletal muscle mass is reported even in compensated cirrhosis.[33] Hypo-albuminemia in the absence of other causes represents liver damage, making it a component of the liver function tests. Although pre-albumin is better than albumin in reflecting protein reserve, it is not routinely used in clinical practice. In 1996, low serum albumin (81%) and lymphocyte dysfunction(59%) were reported to be more common than energy malnutrition (34%) in hospitalized cirrhotics by Caregaro L *et al.* [21] Subsequent reports also found hypoalbuminemia and and lymphocyte anomalies more frequently (45% and 22%) than energy malnutrition in cirrhosis, and a low serum cholesterol value in advanced disease.[20]

The micronutrient deficiencies, assessed clinically, did not differ between alcoholic and non-alcoholic cirrhosis in our study. Although their serum levels were not done, we assume that irrespective of etiology, micronutrient deficiencies is also common in our patients and needs to be corrected in all cases of cirrhosis when present. The micronutrients are essential for cell functions, tissue integrity, metabolism, fluid balance and enzymatic activities- all of which usually become abnormal in cirrhotics. In hospitalized patients, there is no definite guidelines for micronutrient requirements and is an area which needs further studies.

Comparatively, males cirrhotics show a higher loss of muscle mass, and females a higher loss of body fat.[18] Peng *et al* in their study found that protein depletion was significantly more prevalent in men (63%) than in women (28%) (P<0.0001), while the overall protein deficiency was 51%, in a series of 236 cirrhotics irrespective of etiology.[5] Other workers[34] also found that fat reserves were more depleted in females(48.6%) than in males (26.6%) irrespective of the etiology, while muscle mass was significantly lower in males (43.4%) compared to females (13.4%). In our study, fat reserves evaluated by TSF were significantly depleted in females (66%) than in males (56%). On the other hand, muscle reserves were more depleted in males (64%) compared to females (42%) as evaluated by MAMC. These results are similar to those observed in a multicentric Italian study performed in hospitalized cirrhotics. [34] Hence, our results reconfirm the differences in the mode of presentation of PEM between male and female cirrhotics. It is possible that, as suggested by Lolli *et al*,[35] these differences are due to a larger body fat mass in females that can be used over time to meet the catabolic demands in cirrhosis; and thereby spare the muscles for future stages when the fat reserves are exhausted with disease progression[34]

None of our subjects had overnutrition as opposed to in western literature where upto 26% of hospitalized cirrhotics may be obese.[2]

Conclusion

Malnutrition is widely prevalent in liver cirrhosis, but significantly more in alcoholic cirrhosis. It increases with the CP grade. They are at risk of immune depression, acquiring infections and have significantly low visceral protein. The muscle and fat loss occur equally, unlike in other Asians, but the fat loss is significantly less in females compared to the males who show more protein loss. Clinical signs of micronutrient deficiency is a significant association while overnutrition is uncommon. Compared to anthropometry, the RFH-NPT tool is equally effective in assessing malnutrition; and since it is easy to perform, it should be a good tool in our clinical setting and should be routinely used to assess all cases of cirrhosis whether hospitalized or not. This will enable us to institute appropriate care plan in cirrhotics to reduce morbidity, mortality and cost of treatment. The major aim of a nutritional plan should be a sufficient oral diet which includes enough proteins and calories.

TABLE 1 Showing the etiology and Child Pughes grades in study population

ETIOLOGY	Number of Cases (%)	Child Pugh grade A	Child Pugh grade B	Child Pugh grade C
Alcohol	123 (56)	17	35	71
HBV	21 (10)	6	5	10
HCV	8 (4)	2	1	5
Autoimmune	7 (3))	0	1	6
Wilson's disease	2 (1)	0	1	1
Cryptogenic	58 (26)	3	12	43
TOTAL	219 (100)	28 (13)	55 (25)	136 (62)

TABLE 2 Showing the presence and absence of malnutrition by three assessment methods, laboratory indices in alcoholic and non-alcoholic cases and micro-nutrient deficiencies

Parameter	Normal	Moderate	Severe
RFH-NPT	22	51	146
MAMC c.m.	19	46	154
SFT c.m.	22	35	162
Serum Albumin (gm/dl) Mean & SD	3±0.7	2.1±0.32	1.8±1.0
Total Lymphocyte Count (cells/ml) Mean & SD	1121±101.8	904±143	589±98.9
Total Serum Cholesterol (mg/dl) Mean & SD	162±18.3	136±18.8	109±28.2
Alcoholic cirrhosis	11	33	79
Non alcoholic cirrhosis	24	37	35
Micronutrient deficiencies ***n: 202***	13	38	151
Alcoholics	7	22	97
Non-alcoholics	6	16	54

TABLE 3 Showing the gender differences of PEM in study population in regards muscle mass and fat loss

Anthropometry	MALES *n:157*	FEMALES *n:62*
MAMC c.m.	101 (64%)	26 (42%)
TSF c.m.	89 (56%)	41 (66%)

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