Nutritional Status Assessment in Children with Chronic Liver Disease

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ABSTRACT

Chronic Liver disease (CLD) is commonly seen problems in children. Children are in the stage of growth and development so the macronutrients and micronutrients requirements is more compared with adults with CLD. Disease process due to CLD on the other hand cause early satiety and anorexia. Combined effects of increased nutritional requirements and anorexia leads to various micronutrient and macronutrient deficiencies. Therefore it is important to diagnose these nutritional deficiencies at the earliest so that early nutritional interventions can be done for better management of children with CLD.

Objective: Purpose of the study was to know and assess the nutritional status of children with CLD.

Methodology: 50 children with CLD recruited from the Pediatric Gastroenterology Department. Assessment was done by taking detailed three day dietary history, weight and height as per IAP guidelines. BMI, Triceps skin fold (TSF), MAC (Mid arm circumference) calculated by respective centile charts. Whole body DEXA scan to know Bone mineral content (BMC) and fat area.

Analysis and results: Half of the children were underweight. 44% were stunted and 38% were both stunted and wasted.40% had low BMI and ascites. Smaller children (<5 years) were more severely affected. AMA (Arm muscle area) was affected in majority of children (88%). A synthetic function of liver was low in more than half of the children. DEXA scan reveals low body fat in 28% children and 12% children had lo BMC (Bone mineral content).

Conclusion: Malnutrition is common in children with CLD. Early nutritional status assessment and early nutritional intervention is key for the better outcome of CLD.

Keywords: Nutritional status, Chronic Liver Disease, Whole body DEXA Scan

INTRODUCTION

Chronic liver disease (CLD) occupies a major portion in pediatric gastrointestinal diseases. Around two third pediatric populations with CLD awaiting liver transplantation are malnourished.[1] There is a to and fro interaction between CLD and malnutrition. Majority of children with CLD are often malnutrition, and malnutrition adversely affects the course of liver disease[1]. Nutritional deficiencies are frequently noted in children with CLD, particularly in cholestatic liver disease and onset is in infancy period[2,3]. It has been demonstrated in many studies that malnutrition is an independent risk factor for poor outcome of CLD, which lead to the emergence of many severe complications in patients with cirrhosis, such as ascites, hepatic encephalopathy and various infections[4]. An imbalance between nutritional intake and nutrient requirement can adversely affect and lead to metabolic abnormalities, physiological changes, reduces organ and tissue function, and loss of body mass[5]. Protein and energy intake may be inadequate because of multiple

factors such as anorexia; early satiety (caused by impingement upon viscera by enlarged liver, spleen or ascites) recurrent infections. Malabsorption of dietary fat due to impaired bile flow is also observed in many children with CLD[6,7]. In addition, alteration in amino acid metabolism[8, 9] and increased energy requirements due to disease process and many other factors[10] may contribute to suboptimal energy and nitrogen balance. These nutritional imbalance are thought to be secondary to the interaction between factors such as reduced energy intake, lipid and fat-soluble vitamins malabsorption, increased energy expenditure, altered intermediate metabolism, hormonal dysregulation and chronic anemia related to hypersplenism and portal hypertension.[11,12,13,14]

Regarding laboratory parameters, which are markers of nutritional status, such albumin and prealbumin could be low because of low levels of synthesis, rather than because of poor nutritional status. So there levels may not reflect true nutritional status.

Weight for height is more useful as it assess weight in relation to current stature. It might be more accurate as it can assess whether wasting, stunting or both have occurred and it is also age independent parameter to assess nutritional status in children. Other important parameter is body mass index (BMI), an index of nutritional status, may also be overvalued in patients with edema and ascites because fluid informs of ascites and edema will reflect as falsely extra body weight. Intelligent and analytical interpretation of nutritional data using these techniques in the presence of these complications is therefore required. Generally accepted methods for assessing the clinical status and severity of disease in cirrhotic patients are the Child-Pugh-Turcotte classification[15]. The uses of anthropometric parameters that are not affected by the presence of ascites or peripheral edema are mid-arm muscle circumference (MAMC), mid-arm circumference (MAC), and triceps skin fold thickness (TSF). Subcutaneous fat is approximately (50%) of body fat stores, therefore measuring subcutaneous fat would reflect total body fat (TBF)[16]. Diagnosis of malnutrition is established by values of MAMC and/or TST[17]. By measuring TSF and MAC it enables arm muscle area (AMA) and arm fat area (AFA) to be calculated .AMA reflects calorie intake and muscle mass and is sensitive to changes in nutritional status[18]. Skinfold thickness measurements cannot be taken in infants less than three months because of variations in fluid compartments.

Dual-energy X-ray absorptiometry (DEXA) is an indirect, low radiation exposure of bone mineral content or bone mineral density. It uses the same assumptions as the body compartment approach of assessment, that is,

Soft tissue = bodyweight − skeletal mass and soft tissue = fat + water equivalent tissue.

It also assumes soft tissue-overlying bone cannot be sampled and its composition has to be extrapolated from the composition of adjacent tissue. Bone mass, FFM and fat body mass (FBM) can be determined with a 2–3% precision in adults (5% in newborns). The ease of use, low radiation exposure and ability to obtain bone mineral content makes this method very useful[19].

Detailed nutritional assessment should be done at the time of diagnosis of CLD by a trained dietician/nutritionist and subsequently while on nutritional rehabilitation[20]. Serial measurement of nutritional status can guide and confirm the effects of successful nutritional therapy. Till date there are scarcities of literature on nutritional assessment of children with CLD. We have prospectively evaluated the usefulness of various nutritional assessment techniques to find out magnitude and type of malnutrition present in children with CLD. We also correlated the anthropometric, biochemical and whole body DEXA scan parameters.

SUBJECTS AND METHODS

Subjects

This was a prospective, cross sectional cohort study done over two years. The ethics committee approval was obtained from Institutional ethics committee before starting the study. Patients recruited from children attending to outpatient and inpatient services of Department of Pediatric Gastroenterology, Sanjay Gandhi Post Graduate Institute of medical Sciences, Lucknow, India. Out of 50 children recruited, the mean age of the children 9.2 (range 1-22) years. 40 were males and 10 females with various forms of CLD with onset from infancy to older age distribution included in the study. Etiological distribution of children with CLD is shown in table-1

Table-1

S No	Etiology	No.(%)
1.	Wilsons disease	12(24)
2.	Autoimmune Liver disease	09(18)
3.	CLD underwork up	13(26)
4.	Chronic hepatitis B infection	07(14)
5.	Cryptogenic	03(06)
6.	Biliary atresia	02(04)
7.	Progressive familial intrahepatic cholestasis (PFIC)	03(06)
8.	Paucity of intrahepatic biliary duct (PILBD) Alagille syndrome	01(02)

20(40%) patients detected mild to moderate ascites on clinical or on ultrasonographic evaluation.

Dietary history

As dietary patterns in children vary compared to that in adults, a three-day dietary recall was taken and average of three-day dietary intake taken as calorie/protein intake per day.

Body mass index

Body mass index (BMI) was calculated as: Weight (Kilogram)/Height (Meter)2

Nutritional assessment

All anthropometric evaluations were done by only one dietician to avoid inter observer error. Length or height measured to the nearest 1.0 cm on SECA make stadiometer. Weight measured to the nearest 5.0 gm., triceps skinfold (TSF) and subscapular skin fold (SSF) measured to the nearest 0.01 mm with a skinfold caliper (Harpenden type, SECA Make, SFCG 45) on the back of the arm, mid-way between the acromion and olecranon process with the arm relaxed, and mid circumference (MAC), to the nearest 1.0 mm (13). Arm muscle area (AMA) and arm fat area (AFA) were calculated with the help of TSF and MAC. AMA and AFA were calculated through the following equations:

$$AMA \ (cm2) = (MAC \ [cm] - [TSF \ X \ \pi]) \ 2 \ /(4 \ x \ \pi)$$

AFA (cm2) = upper arm area − AMA,
Where upper arm area = MAC2 (cm) / (4 X)
Where $\pi = 3.14$

Laboratory parameters

Basic laboratory parameters like hemoglobin, liver function tests done in all cases.

Whole body DEXA scan

Dual energy X-ray absorptiometry (DEXA) is an indirect, low radiation exposure of bone mineral content or bone mineral density. Fat free mass (FFM) and fat body mass (FBM) determined by whole body DEXA scan in all patients.

Statistical analysis

Z score was calculated for weight, height and BMI with the help of charts. TSF, SSF and AMA data analysed using standard centile charts. Standard fat and bone mineral content percentile chart (USA CDC0 was used to analyse whole body DEXA scan data.

RESULTS

In this cohort half 50% (25/50) children were underweight (z score < 2 SD). 44% (22/50) children were stunted

suggestive of chronic malnutrition. 38 % (19/50) were both stunted and wasted. 40% (20/50) had low BMI (z score < - 2 SD). Ascites was present in 40 %(20/50) patients. There were 11 children in 1-5 years age group.7 children out of 11 in these group had MAC <12 cm area, indicating significant malnutrition.72%(36/50) having caloric intake between 50-80% of their required value for their age. 42% (21/50) patients having TSF < 5th centile using standard centile charts. Arm circumference and AMA falls below 5th centile in 88% (44/50) children using standard centile charts. AMA was low in 95%(19/20) in children with ascites verses 83%(25/30) in non-ascetic group patients, although statistically there was no significant difference (P= 0.25). 58%(29/50) children having low serum protein value and 72%(36/50) patients having low serum albumin. Over all 22%(11/50) patients having both low value of both serum protein and albumin.

Whole body DEXA scan done in all children revealed low body fat % in 28% (14/50) children and 12%(6/50) children had also low bone mineral content. Bone mineral content was affect mainly in patients with severe disease.

The summary of results are shown in Table-2

Table-2

S No	Nutritional parameter	Numbers/total (percent value)
1	WSDS (z score > -2 SD)	25/50(50%)
2	HSDS > -2SD	22/50(44%)
3	WSDS + HSDS > - 2SD	19/50(38%)
4	BMI z score >-2SD	20/50(40%)
5	Total body fat % DEXA scan	14/50(28%)
6	Bone mineral content(BMC) by DEXA scan	6/50(12%)
7	Arm circumference (AC) < 5th centile	44/50(88%)
8	AC < 5th centile (Non Ascetic form)	25/30(83%)
9	AC < 5th centile (Ascetic)	19/20(95%)
10	Low serum protein(SP)	29/50(58%)
11	Low serum albumin(SA)	36/50(72%)
12	Low SP+ SA)	11/50(22%)

WSDS= Weight standard deviation score
HSDS= Height standard deviation score

DISCUSSION

We found that nearly half of the patients had chronic malnutrition, and in many of them it was compounded by superadded acute malnutrition. Under nutrition and stunting is explainable on the basis of primary disease process and reduced oral intake leading to inadequate calories. More than two third of children had low caloric intake, which may be due to various factors, i.e. chronic disease process leading to anorexia, easy satiety due to extrinsic compression of stomach by organomegaly and ascites. Muscle wasting was more prominent in younger age group (1-5 years) children indicating that this subset children is more vulnerable to morbidity and complications. As expected children with ascites have more protein depleting parameters. This also indicates that there is more severe catabolic state in ascitic form of CLD patients. Both low serum albumin and total proteins values reflect poor synthetic functions of the liver in the majority of disease process are advanced.

The whole body DEXA scan reveals that total body fat was less affected as compared to muscle mass, because CLD patients have higher catabolic state that leads to protein utilization as energy source leading to protein depletion and sparing of fat. Only few children have low bone mineral density content might suggesting that Vitamin D3 metabolism is least affected in children with chronic liver disease.

CONCLUSION

Majority of children with CLD were malnourished. Children below 5 years were more affected compared to older children. Bone metabolism was less affected in children with CLD. Early detection of malnutrition and early specific and therapeutic nutritional intervention is the key factor in the nutritional management of CLD.

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