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Original Article

Sarcopenia and Sarcopenic Obesity among Non-alcoholic Fatty Liver Disease Patients in North East India

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Abstract

Introduction: Non-alcoholic fatty liver disease (NAFLD) is an epidemic of new millennium. NAFLD and its complications are increasing with the prevalence of metabolic syndrome. Sarcopenia is considered to be complication of chronic liver disease. Chronic liver disease patients may be of a normal body mass index, or overweight or obese and yet be with sarcopenia. Sarcopenic obesity is defined as simultaneous presence of both sarcopenia and obesity.

Result: In this study, frequency and severity of sarcopenia and sarcopenic obesity in NAFLD was evaluated. NAFLD patients were categorized based on ultrasonography abdomen into grade 1, 2, and 3 and their skeletal muscle index was calculated after getting psoas muscle area by CT scan of abdomen. Out of total 110 patients, 17.3% patients were of grade 1 NAFLD, 39.1% patients were having grade 2 NAFLD, and 43.6% patients were having grade 3 NAFLD. In the correlation between sarcopenic obesity and NAFLD showed that 5.5% with grade 1 fatty liver, 25.5% with grade 2 fatty liver, and 43.6% of grade 3 NAFLD patients were having sarcopenic obesity.

Discussion: It was observed that sarcopenia and sarcopenic obesity is seen even in early stages of NAFLD. These patients can develop complications of chronic liver disease at an early stage.

Key words: Metabolic syndrome, Non-alcoholic fatty liver disease, Sarcopenia, Sarcopenic obesity

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is defined as presence of more than 5% fat in the liver (hepatic steatosis) either on imaging or on liver histology after exclusion of secondary causes of fat accumulation in the liver.

Sarcopenic obesity is defined as simultaneous presence of both sarcopenia and obesity. There is low muscle mass, instead of over-weight, or obesity as per body mass index (BMI). It is highly related with metabolism related disease, chronic disease, and functional disabilities and described as thin outside, fat inside.

Due to multisystem involvement, NAFLD is now renamed as metabolic (dysfunction) associated fatty liver disease.

NAFLD, non-alcoholic steatohepatitis (NASH), and sarcopenia share several features, such as chronic inflammation, oxidative stress, hormonal alterations, and decreased physical activity. Most of the cytokines found increased in patients with NAFLD and NASH are known to favor protein catabolism.

Reducing excess adiposity remains the fundamental pathogenic treatment for obese individuals, but it may however also compromise the ability to preserve muscle function and mass. Liver steatosis, cirrhosis, and liver cancer become a raising challenge for patients with long-standing obesity.

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In particular, in the pre-model for end-stage liver disease (MELD) score, when less strict criteria were adopted for patients, 8% increased risk of death per unit decrease in transverse psoas muscle thickness per height was observed. The ability of MELD score in predicting mortality was significantly increased when combined with muscle thickness measured [Table 1].

Earlier the causes of chronic liver disease and HCC were alcoholic hepatitis followed by viral hepatitis followed by NASH, but in recent years, the chronology has been changed and now the causes are, respectively, NASH followed by alcoholic hepatitis followed by viral hepatitis.

METHODOLOGY

In this study, 110 patients who were included who had minor gastrointestinal symptoms like dyspepsia was not having any clinical feature of chronic liver disease except mild to moderate hepatomegaly over a period of 2 years (2019–2021), after obtaining informed consent, sociodemographic data, personal history, and excluding significant alcohol history as per criteria Asia pacific association of study of liver. After enrolment vitals, waist circumference, fasting lipid profile, thyroid profile, LFT, FBS, PPBS, HBA1c, blood pressure, BMI, hepatitis B, and hepatitis C (for exclusion if positive) were studied. The study group patients were subjected to ultrasonography of abdomen to obtain the status and grading of NAFLD. Then, they underwent non-contrast computed tomography (NCCT) abdomen to see the cross-sectional area of psoas muscle at the level of L3 vertebra to calculate skeletal muscle index (SMI) as per designed and standard guideline.

Patients with viral hepatitis, alcohol intake history, and liver diseases of other known causes such as ATT, estrogen, or other drug-induced hepatitis were excluded from the study.

In NCCT abdomen scans, skeletal muscle tissue is separated according to different density thresholds. Density value of + 35 HU was used to separate fat from muscle tissue and + 150 HU to separate muscle from bone tissue. The L-3 SMI was expressed as cross-sectional mass area/m^2 [Figure 1].

- Non-contrast CT scans of abdomen, to measure the area of psoas muscle at the level of L3 vertebra is used
  - Skeletal muscle area: Males – 144.3 cm^2, Females – 92.2 cm^2
  - SMI: Males – 45.4cm^2/m^2, Females – 34.4 cm^2/m^2

L3 SMI = cross-sectional area of muscles at L 3 (cm^2)/ height^2 (m^2).

BMI was computed as body weight (kg)/height (m^2) was calculated by Quetelet index formula. In this study, Asia – Pacific BMI chart was used.

BMI with <18.5 considered underweight, 18.5–24.9 considered normal, 25–29.9 considered overweight, and >30 are considered as obese according to Asia – Pacific classification.

RESULTS

Male and female ratio was almost same. M: F: (48.2%: 51.8%).

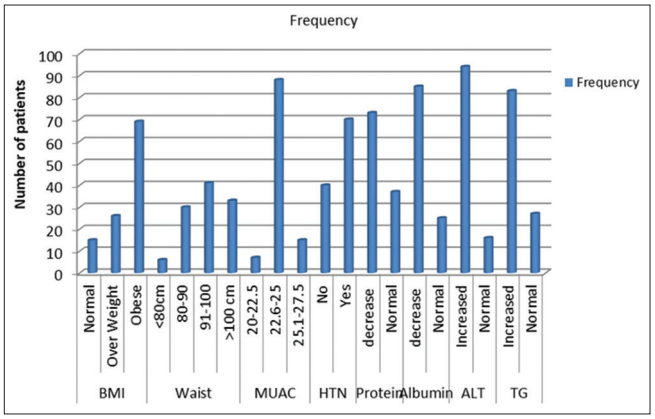

Figure 1: Bar diagram showing distribution of body mass index, waist circumference, hypertension, and triglyceride

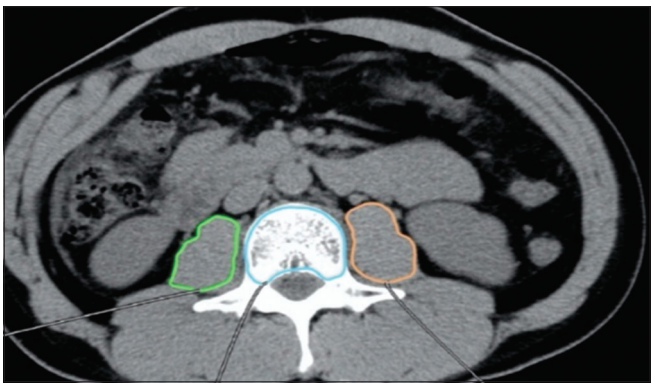

Figure 2: Non-contrast computed tomography abdomen showing psoas muscle cross-sectional area at the level of L3 vertebra

Table 1: Association between skeletal muscle index and USG abdomen

SMI	USG grade 1 (%)	USG grade 2 (%)	USG grade 3 (%)
15–20	0	0	13 (11.8)
21–25	0	0	27 (24.5)
26–30	0	16 (14.5)	8 (7.3)
31–35	2 (1.8)	10 (9.1)	0
36–40	6 (5.5)	11 (10)	0
41–45	0	4 (9.3)	0
46–50	11 (10)	2 (1.8)	0
Total	17.3	39.1	43.6

As per age group: (a) 16.4%: 21–30 years of age, (b) 24.5%: 31–40 years, (c) 34.5% 41–50 years, and (d) 24.5% 51–60 years of age. Comparatively more in 41–50 years of age group. In this study, 13.6% patients had normal BMI, 23.6% were overweight, and 62.7% patients were obese [Figure 2].

In the evaluation of association between sarcopenic obesity and NAFLD (diagnosed by USG abdomen) showed that 5.5% with grade 1 fatty liver, 25.5% with grade 2 fatty liver, and 43.6% of grade 3 NAFLD patients were having sarcopenic obesity.

It is important to know that normal Skeletal muscle index in males is 45.4 cm^2/m^2 and in females is 34.4 cm^2/m^2.

Out of 110 patients (n = 110), 62.7% were diabetic and 37.3% were non diabetic and 63.6% had hypertension (HTN). Triglyceride (TG) levels were increased in 83 (75.5%) patients, 67 (60.9%) of them had metabolic syndrome.

It was found that 57.3% of hypertensive patients, 54.5% of diabetic patients and 90.8% of patients with hypertriglyceridemia were having sarcopenic obesity.

DISCUSSION

The result of this study showed that maximum number of patients was having NAFLD and sarcopenic obesity in their late 40. Aging is accompanied by changes in body composition, like decrease in muscle mass, increase in abdominal adiposity, and ectopic fat deposition and insulin resistance.[1]

In the present study, 51.8% were female and 48.2% were male. The prevalence of NAFLD and sarcopenia tends to increase in post-menopausal women.[2]

In this study, 13.6% patients had normal BMI, 23.6 were overweight, and 62.7% patients were obese. Higher amounts of visceral relative to peripheral and subcutaneous adipose tissue are associated with greater risk of metabolic syndrome and are directly linked to liver inflammation and fibrosis, independent of insulin resistance, and hepatic steatosis.[3] In this group, normal weight individuals can be classified as metabolically obese normal weight and they demonstrate an increased risk for cardiometabolic risk.[4]

In the study group, 62.7% were diabetic and 37.3% were non diabetic, 63.6% had HTN. TG levels were increased in 75.5% and 60.9% of them had metabolic syndrome.

In this study, 57.3% of hypertensive patients, 54.5% of diabetic patients, and 90.8% of patients with hypertriglyceridemia were having sarcopenic obesity. Few studies showed that skeletal muscle is a major insulin responsive organ, loss of skeletal muscle, and myosteatosis can lead to a decrease in insulin response and energy expenditure, leading to increased hepatic gluconeogenesis, increased free fatty acid uptake and FFA oxidation.[5]

In recent studies, two different subtypes of NAFLD have been proposed based on lipid deposition. In subtype 1 based on insulin resistance, patients tend to have mono unsaturated TAGs and free fatty acids enriched with ceramides in liver. Subtype 2 based on carrying the PNPLA3 risk genotype have polyunsaturated TAGs.[6]

The prevalence of NAFLD was elevated among patients with T2DM, higher levels of waist circumference, BMI, TG.[7,8]

CONCLUSION

Sarcopenia begins to manifest in the early stages of NAFLD. Sarcopenia is preventable and disease modifying variable. The main intervention for preventing Sarcopenic obesity is physical activity which can cause certain weight loss and improve insulin sensitivity. But only physical activity can cause muscle mass loss if it is not supported with proper nutrition. Few studies have shown that Mediterranean diet, intermittent fasting can help with maintaining weight loss and insulin sensitivity. Adding resistance exercise to weight loss program can help prevent the reduction in muscle and bone mass.[9-11]

Screening for sarcopenia in early stages of NAFLD and treating it can improve the outcome as few studies have shown that higher skeletal muscle mass is associated with lower incidence of NAFLD and resolution of existing NAFLD.[12-14]

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