

Abstract - Muscle function is already impaired in non-cirrhotic patients with steatotic liver diseases compared to healthy controls

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Introduction

Muscle function decay and frailty are prevalent in cirrhosis whatever the cause. However, muscle function evaluation in non-cirrhotic patients is lacking, as well as data by etiology, including steatotic liver diseases (SLD). Our aim is to determine if muscle function already decays in non-cirrhotic SLD and if the SLD subtype impacts muscle function.

Aim

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Methods

SLD patients were prospectively recruited and classified according to the recent SLD subtype categories. Liver disease was assessed by transient elastography (Fibroscan®). Cirrhotic patients were excluded. Controls were defined by the absence of liver steatosis on controlled attenuation parameter (< 215 dB/m). Muscle function was assessed by isokinetic dynamometer (Cybex®). All patients and controls also underwent the three tests used to calculate the liver frailty index (LFI): handgrip strength, sit-to-stand test and balance test. SLD patients and controls were classified based on the LFI as robust (LFI < 3) or pre-frail/frail (LFI ≥ 3). Results are expressed as means ± SD.

Results

One hundred and thirty-seven patients with SLD were included: 69 with alcohol-related liver disease (ALD) and 66 with metabolic dysfunction-associated steatotic liver disease (MASLD). Thirty healthy participants matched for sex and age (controls: 47.8 years ± 14.1, ALD: 50.2 ± 10.8, MASLD: 52.5 ± 10.4; $p = 0.16$) were used as controls. However, the groups differed for several parameters, such as alanine aminotransferase (controls: 18.4 IU/L ± 10.2, ALD: 74.8 ± 53.2, MASLD: 48.3 ± 29.5; $p < 0.0001$), liver stiffness (controls: 4.4 kPa ± 1, ALD: 8.9 ± 3.5, MASLD: 9 ± 3.9; $p < 0.0001$) and body mass index (controls: 22.1 kg/m² ± 2, ALD: 26.3 ± 5, MASLD: 33.3 ± 6.4; $p < 0.0001$). LFI was higher in all SLD subgroups compared to controls (controls: 2.2 ± 0.8, ALD: 3.2 ± 0.8, MASLD 3.1 ± 0.7; $p < 0.0001$) without any difference between MASLD and ALD patients ($p = 0.67$). 56.5 % of ALD patients and 58.5 % of MASLD patients were considered pre-frail or frail ($p = 0.81$) compared to only 10 % of control patients ($p < 0.0001$). LFI negatively correlated with right knee extension evaluated by isokinetic dynamometer in all SLD patients ($N = 137$, $r = -0.53$; $p < 0.0001$).

Conclusions

LFI is an accurate method to assess muscle function in non-cirrhotic SLD patients. Muscle function assessed by the LFI decays compared to age-matched non-SLD controls. Frailty is not influenced by the SLD subtype. This reinforces the concept of a muscle-liver axis already in the early stages of SLD.