



Correspondence

Malnutrition, sarcopenia, and refractory ascites in end stage liver diseases: is there a way to climb back up?



The recently published guidelines on nutrition in chronic liver disease, proposed by the European Association for the Study of the Liver (EASL), offer a wide panorama of the most important recommendations on malnutrition in cirrhotic patients [1]. Nevertheless, as stated by the authors, it is still controversial whether malnutrition in decompensated liver disease can be reversed by the proposed nutritional strategies (late evening oral nutritional supplementation and breakfast in a dietary regimen, branched chain amino acid supplements, leucine-enriched amino acid supplements, improvement of diet palatability and enteral or parenteral nutrition) [1].

In the course of liver cirrhosis, a reduction of protein synthesis and an increase of gluconeogenesis from amino acids are common metabolic alterations that contribute to sarcopenia. In patients with end-stage liver disease, sarcopenia can be aggravated by malnutrition, accelerating the progression to decompensation and ascites [2]. Therefore, malnutrition combined to severe sarcopenia is detrimental in these patients, especially if they are included in a waiting list for liver transplant since this condition significantly increases the risk of morbidity/mortality, exposing these patients to the possibility of being withdrawn from the list [3].

In the last decade, an upward trend has been reported in the age of liver transplant recipients [4]. Since older age increases the risk of frailty [5], we can reasonably expect that, in the future, we will deal with a higher proportion of sarcopenic malnourished patients on the waiting list.

In this clinical setting, a particularly challenging condition is represented by refractory ascites [6]. Every liter of ascites counts about 21 g of protein (predominantly albumin); therefore, since large volume paracentesis can be repeated within a few days, it can lead to a genuine “protein losing condition”, subtracting a large amount of proteins and other micro- and macro-nutrients [7]. Such a precious loss determines a further progressive deterioration in the nutritional status that cannot be reversed either by mere albumin infusions or by one of the nutritional strategies reported in EASL guidelines [1].

A recently published pilot study suggested a possible rescue therapy in patients with decompensated liver disease who do not respond to standard nutritional interventions. It consists in continuous (24 h) administration of low-volume/low-calorie enteral nutrition by nasogastric tube. In this study, tube feeding reduced the severity of ascites, the requirement for paracentesis, and improved muscle strength [2]. However, these results need to be confirmed by more extensive studies since at follow up (1.5–12

weeks) there was no change in the nutritional status assessed by the mid-arm muscular area.

Another aspect to consider is the possibility of treating refractory ascites by implanting a transjugular intrahepatic portosystemic shunt (TIPS), once all contraindications are excluded [6]. As reported in a systematic review evaluating eight studies on cirrhotic patients undergoing TIPS, the resolution of the ascites led to an improvement in both the nutritional status (both fat free mass and fluid/ascites-free body weight, in all studies) and muscle strength (only in one study) [8]. However, in none of these studies, a direct measurement of skeletal muscle mass was performed. More recently, another study demonstrated that TIPS can reverse sarcopenia in cirrhotic patients [9]. Finally, it has been reported that this interventional procedure can contribute to improving the nutritional status not only through the disappearance of ascites (which is beneficial towards anorexia, nausea, vomiting, and early satiety), but also by increasing adiponectin production and consequently ameliorating the anabolic state [10].

In conclusion, the occurrence of refractory ascites in end-stage liver disease represents a protein-losing condition that requires rapid and specific interventions capable of arresting/ameliorating the progression of sarcopenic malnutrition. Nutritional support consisting of a continuous enteral feeding by nasogastric tube could represent a possible approach to be used alone or in combination with TIPS. This therapeutic intervention should be adopted as soon as possible in all patients experiencing refractory ascites and especially in liver transplant candidates since it could be a bridge solution to transplantation.

Conflict of interest

None declared.

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