Diets in Encephalopathy

George G. Abdelsayed, MD

KEYWORDS

• Diet • Encephalopathy • End-stage liver disease • Malnutrition

KEY POINTS

- As many as 80% of patients with end-stage liver disease and hepatic encephalopathy have significant protein-calorie malnutrition.
- Because of the severe hypercatabolic state of cirrhosis, the provision of liberal amounts of carbohydrate (at least 35 to 40 kcal/kg per day), and between 1.2 and 1.6 g/kg of protein is necessary.
- Protein restriction is not recommended; branched-chain amino acid supplementation and vegetable protein are associated with improved outcomes.
- Dietary supplementation with vitamins, minerals (with the notable exception of zinc) and probiotics should be decided on a case-by-case basis.

INTRODUCTION

Hepatic encephalopathy (HE) is a disorder of reversible impairment of cerebral function in patients with acute or chronic hepatic failure or when the portal circulation is bypassed by the creation of portosystemic shunts. The disorder carries a dismal prognosis with a 40% survival at 1 year.[1] Up to 80% of patients with cirrhosis may have clinically undetectable or minimal HE. This article discusses current concepts in the dietary and nutritional management of HE.

GENERAL

In order to understand the rationale behind dietary management in HE, it is important to briefly review the pathophysiology of HE. The liver plays a central role in the detoxification and neutralization of many toxic substances absorbed from the gastrointestinal tract, as well as other substances produced as byproducts of normal daily metabolism. The toxins enter the portal circulation through the low-flow hepatic sinusoids. The detoxification process occurs in the hepatocytes. Among the central toxins studied is ammonia. Ammonia is absorbed by both neurons and astrocytes. The astrocytes convert the ammonia to glutamine to minimize its toxic effects on the neurons. Ammonia is toxic to both astrocytes and neurons. It is the astrocyte (the glial cells of the central nervous system) that is pivotal in providing adequate nutrition to

Disclosure: The author has nothing to disclose.
475 Seaview Avenue, Staten Island, NY 10305, USA
E-mail address: g-abd@hotmail.com

Clin Liver Dis 19 (2015) 497–505
http://dx.doi.org/10.1016/j.cld.2015.05.001 **liver.theclinics.com**
1089-3261/15/$ – see front matter

neurons.[2] Fig. 1 shows the metabolism of ammonia and its role in HE and inflammation. Although the exact mechanisms of damage to neurons and astrocytes by toxins, including ammonia, in HE is not understood, it is known that astrocyte swelling with resultant cerebral edema is key in the pathophysiology of HE associated with acute liver failure.[3]

As many as 80% of patients with end-stage liver disease have varying degrees of protein-calorie malnutrition, caused by multiple factors (Fig. 2).[4] This degree of protein-calorie malnutrition may be as high as 25% in patients who are Child-Pugh class A.[5] Because of significant fluid retention, hypoalbuminemia, and loss of muscle mass, it is not always possible to use objective parameters to assess the degree of malnutrition in decompensated cirrhotic patients. In 2006, The European Society for Clinical Nutrition and Metabolism published guidelines on nutrition support for patients with liver disease for inpatients and outpatients.[6] Because of the aforementioned difficulties of applying objective parameters, simple bedside methods such as subjective global assessment or anthropometry are used to identify high-risk patients.[7] Wherever possible, enteral nutrition is strongly favored rather than parenteral nutrition. Parenteral nutrition may be indicated in situations of ongoing sepsis, complete bowel obstruction or persistent vomiting, diarrhea, or aspiration. Wherever possible, the benefits of parenteral nutrition must be carefully weighed against the risks, particularly septic and fluid overload complications.[8]

CARBOHYDRATE INTAKE

Because of the severe anorexia of cirrhosis, many patients inadvertently follow a hypocaloric diet. Among the contributing factors are the circulation of anorexigenic proinflammatory intermediaries, such as the interleukins and tumor necrosis factor alpha, and impaired gastric distensibility from ascites. With a resting energy expenditure 120% above baseline, and the hypermetabolic/catabolic state of cirrhosis, carbohydrate intake must necessarily be liberalized. Patients with cirrhosis are also glycogen depleted. The hyperglucagonemia and insulin resistance in these patients impairs glycogenolysis, which is combined with the already depleted glycogen stores in the liver. Gluconeogenesis therefore becomes the preferred method for the replenishment of glucose. This process consequently results in a severe depletion of amino acid stores in the liver and skeletal muscle. Although this may occur in healthy

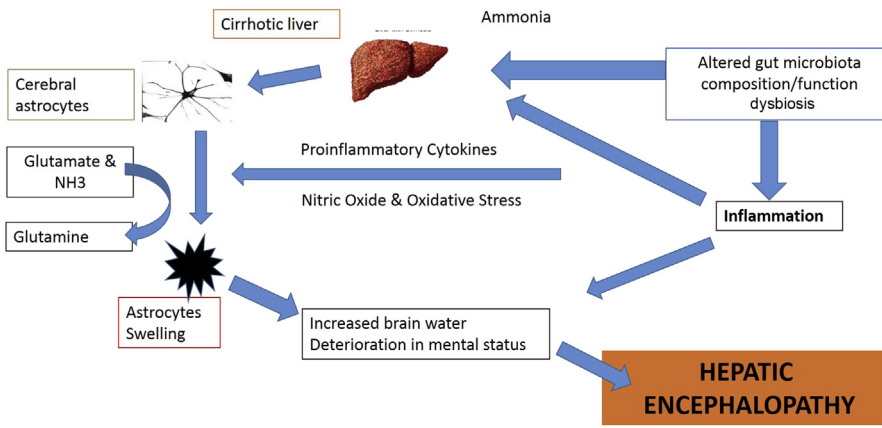

Fig. 1. Pathophysiology: ammonia and inflammation.