Dietary protein intakes in patients with hepatic encephalopathy and cirrhosis: current practice in NSW and ACT

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ABSTRACT

Objective: To ascertain whether current practice in teaching hospitals in New South Wales and the Australian Capital Territory delivers adequate dietary protein in the management of malnutrition in adults with cirrhosis, in accordance with European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines for nutrition in liver disease.

Study design: Cross-sectional study of dietitians using a self-administered, mail-back survey.

Setting: Teaching hospitals in NSW and the ACT treating patients with cirrhosis.

Participants: Dietitians seeing patients with cirrhosis in the 12 months prior to completing the survey.

Main outcome measures: Current dietary protein prescription practice for patients with cirrhosis (with and without hepatic encephalopathy); use of nutritional supplements and enteral feeding for malnourished patients with cirrhosis.

Results: Dietitians following the ESPEN guidelines were in the minority: 36% of the dietitians recommended an adequate protein intake for patients with hepatic encephalopathy. Sixty-four per cent of the dietitians had received referrals from the medical team requesting inappropriate protein-restricted diets for patients without hepatic encephalopathy. Seventy-eight per cent of the dietitians requested clarification of the recommended nutritional management of patients with cirrhosis.

Conclusion: Many medical and dietetic staff inappropriately restrict protein intake of patients with cirrhosis.

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The restriction of dietary protein intake in patients with cirrhosis of the liver and hepatic encephalopathy became common practice in the 1970s and 1980s but was not clearly evidence-based.[1] During this period, the surgical creation of a portosystemic shunt was often the only successful therapy for bleeding oesophagogastric varices. After this operation, some patients were noted to develop significant hepatic encephalopathy as a result of shunting protein-rich blood from the gastrointestinal tract away from the hepatocytes. In this setting, protein restriction was shown to decrease hepatic encephalopathy, but the practice of protein restriction was expanded to include management of all patients with cirrhosis who developed hepatic encephalopathy. Hepatology textbooks at that time recommended severe dietary protein restriction of 0–40 g protein per day.[2]

Increased understanding of the pathophysiology of cirrhosis has shown that most patients with cirrhosis do not have high-level portosystemic shunting, and that protein restriction in these patients has no impact on their hepatic encephalopathy.

Guidelines published by the European Society for Clinical Nutrition and Metabolism (ESPEN) in 1997[3] recommended higher protein intakes for these patients (Box). A recent randomised controlled trial comparing high- (1.2 g protein per kg body weight per day) and low- (0.5 g protein per kg body weight per day) protein diets for inpatients with hepatic encephalopathy clearly showed that a low-protein diet increased protein catabolism and that restricting protein intake during hepatic encephalopathy had no beneficial effect.[4] Furthermore, studies by Nielsen et al showed that, with increased protein intake, there was very efficient protein retention in patients with cirrhosis, and that increased protein intake did not result in hepatic encephalopathy.[5]

A survey of British dietitians found that 58% continued to restrict dietary protein intake in patients with hepatic encephalopathy 1 year after the ESPEN guidelines had been published.[6]

We conducted a similar survey of dietitians practising in Australian hospitals to ascertain whether the dietitians in our referral area were providing dietary advice in accordance with the ESPEN guidelines for nutrition in advanced liver disease.

METHODS

We developed a questionnaire based on the one used in the British study.[6] It was piloted by dietitians at Royal Prince Alfred Hospital (RPAH) in Sydney and modified, based on their feedback, to improve its relevance to Australian dietetic practice. Our study cohort comprised dietitians in the public and private teaching hospitals ($n = 62$) in New South Wales and the Australian Capital Territory where adult patients with cirrhosis received treatment. Dietitians at RPAH were excluded from the survey because they were already following the ESPEN guidelines for liver disease after an intensive educational program.

The self-administered, retrospective questionnaire on dietary treatment of patients with cirrhosis and hepatic encephalopathy assessed patient numbers, referrals, diet therapy, and symptom management, as well as the use of vitamin/mineral supplements, oral nutritional supplements, and enteral/parenteral feeding. The covering letter sent with the questionnaire gave the background for the study and investigators' contact details, and encouraged the return of the questionnaire anonymously in the included prepaid reply envelope. A follow-up letter requesting the return of the questionnaires was sent a month after the original mailing.

The returned questionnaires were tabulated using Microsoft Excel, version 2000 (Microsoft Corporation, Redmond, Wash, USA) and analysed using SPSS software, version 10 (SPSS Inc, Chicago, Ill, USA).

Ethical approval to conduct this survey was granted by the Ethics Review Committee (RPAH Zone) of the Central Sydney Area Health Service.

RESULTS

Of the 62 questionnaires mailed, 28 (45%) were completed, returned and analysed: 57% of the respondents (16/28) were dietitians with more than 10 years' experience, and 93% of the respondents (26/28) supervised dietetics students in their hospitals.

Requests to restrict dietary protein prophylactically in patients with cirrhosis without hepatic encephalopathy were received by 64% of the respondents (18/28); 39% of respondents (7/18) said this occurred only occasionally or rarely. Most of these requests were received from medical staff.

All of the respondents agreed that it was inappropriate to severely restrict dietary protein in patients with cirrhosis without hepatic encephalopathy, but 21% (6/28) did not recommend a high-protein diet (> 1 g protein per kg body weight per day).

Just over a third of respondents (36%; 10/28) recommended a high-protein diet (> 1 g protein per kg body weight per day) for patients with hepatic encephalopathy. Sixty-four per cent (18/28) thought it was appropriate to provide a diet with either moderate protein restriction (0.8 g protein per kg body weight per day) or severe protein restriction (< 0.5 g protein per kg body weight per day) for patients with hepatic encephalopathy. In 48% of the episodes when protein restriction was implemented (13/27), the medical staff and dietitian in consultation decided the level of protein restriction. Protein restriction in patients with hepatic encephalopathy was maintained until resolution of encephalopathy by 61% of the survey respondents (17/28). However, 14% of the respondents (4/28) restricted the protein intake of patients with hepatic encephalopathy for an undefined or indefinite period.

Seventy-eight per cent of the respondents (21/27) requested clarification of the recommended dietary treatment for patients with advanced liver disease. Best practice guidelines were requested by a third of the respondents.

Nutrition in chronic liver disease — recommendations of the 1997 ESPEN consensus group

Clinical condition	Non-protein energy (kcal·kg^{-1}·d^{-1})*	Protein or amino acids (g·kg^{-1}·d^{-1})*
Compensated cirrhosis	25–35	1.0–1.2
Complications		
Inadequate intake Malnutrition	35–40	1.5
Encephalopathy I–II	25–35	Transiently 0.5, then 1.0–1.5 If protein intolerant: vegetable protein or BCAA supplement
Encephalopathy III–IV	25–35	0.5–1.2 BCAA-enriched amino acid solution

Generally, the oral or enteral routes are preferred.

Parenteral nutrition should only be used when enteral feeding is not possible or impracticable. For parenteral nutrition, energy should be provided by glucose and fat, with fat constituting 35%–50% of non-protein calories. Nitrogen should be provided using conventional amino acid solutions unless indicated otherwise. For calculations, ideal body weight should be used.

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* kg refers to body weight. ESPEN = European Society for Clinical Nutrition and Metabolism. BCAA = branched chain amino acids. ◆

DISCUSSION

With published guidelines[3] advocating high protein intakes for patients with cirrhosis, our findings that current practice is not following these guidelines is of concern. Malnutrition, an extremely common complication of advancing liver disease, adversely affects patient outcomes. Inadequate dietary protein intake has a very deleterious effect on hepatic encephalopathy,[4] nutritional status[1] and clinical outcomes[1,3,4,7] in these patients.

Our study has some limitations. Our response rate of 45% was lower than anticipated and, as our questionnaire was anonymous, we have no data on non-respondents. However, our results were quite similar to those of the British survey,[6] which suggests that our respondents are a representative sample of dietitians. On the other hand, our small sample size may affect the significance of our results.

It is crucial that protein intake is not restricted ad hoc, as unnecessary protein restriction in these critically ill patients with catabolism will further diminish lean body mass.[3,4] The dominant metabolic complication of cirrhosis is the impairment of hepatic glycogen storage capacity, resulting in a state of "accelerated starvation" in which there is early recruitment of fuel stores, other than glucose, during periods of fasting.[7] It is essential that the current ESPEN liver disease guidelines[3] are translated into clinical practice in Australia. Producing Australian dietary guidelines for dietary treatment of cirrhosis and hepatic encephalopathy would assist in the future management of malnourished patients with cirrhosis.

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COMPETING INTERESTS

None identified.

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