

Dispelling dietary myths of liver disease

Julie Leaper and Susie Hamlin dispel some of the myths that are commonly held about dietary advice for people with liver disease

Nutrition in patients with liver disease has changed beyond recognition over the past 20 years. Historically low-fat and low-protein diets were advised which have now proven to be unnecessary and lead to malnutrition (Plauth et al, 2006). Emphasis is now on treating malnutrition and feeding to elevated nutritional requirements and there are very few dietary restrictions necessary. Therapeutic dietary intervention is still appropriate for symptoms such as ascites, jaundice and diabetes, with a new epidemic of non-alcoholic fatty liver disease (NAFLD) patients being seen.

Information available through the internet and media can often be conflicting and confusing so advice should be sought from a registered dietitian, nurse or GP with relevant experience. The aim of this article is to dispel some of the myths commonly held about nutrition in liver disease.

Are low protein diets required for people with liver disease?

Patients with established liver disease have a high incidence of protein calorie malnutrition. This occurs where there is insufficient energy and protein consumed to meet requirements and/or they are unable to absorb essential nutrients to meet demand. Protein calorie malnutrition can be found in 80–100% of patients with decompensated cirrhosis and is associated with worsening ascites, encephalopathy, increased infection rates and hospital re-admissions (Cabre et al, 1990; Mullen and Weber, 1991; McCullough and Bugionesie, 1997; Plauth et al, 1997; Runyon, 1998).

Liver cirrhosis significantly affects protein metabolism and the ability of the liver to store glycogen, which is required for energy. Patients experience severe muscle wasting, lethargy and weight loss. For many years patients with encephalopathy were advised to follow low protein diets in order to reduce the level of aromatic amino acids circulating which may have contributed to neurotoxicity (Saunders et al, 2010). Low protein diets are not recommended owing to a lack of evidence that they contribute to improvement in encephalopathy; they may

cause harm as a result of worsening nutritional status (Plauth et al, 1997).

Instead, patients should be encouraged to continue with high energy and protein diets especially with grade 1 or 2 encephalopathy. Therapy should be optimized to treat the cause of the encephalopathy. This may involve increased laxative dose for constipation to encourage bowels opening 2–3 times a day, antibiotics for sepsis or correction of an electrolyte disturbance. Restricting protein and therefore energy will only serve to hasten a decline in nutritional status. It is beneficial to avoid large protein boluses; therefore meals should be spaced throughout the day with snacks in between and a late evening 50g carbohydrate snack encouraged.

Should patients with liver disease eat every 2–3 hours?

The simple answer is yes. Cirrhotic patients develop starvation-type metabolism earlier than healthy patients. By encouraging the use of frequent snacks and small meals there is a reduced need for the liver to produce fuel from amino acids. Providing a 50g carbohydrate late evening snack can improve nitrogen balance and even quality of life (Plank et al, 2008; Saunders et al, 2010). This can be a simple change that many patients will be able to comply with. Examples of late evening snacks are shown below:

- 2 crumpets with 200 ml of milk
- 2½ thick slices of bread
- ¾ carton of juice-based 1.5 kcal/ml supplement
- Breakfast cereal with milk
- 1 slice fruit cake
- Scone with jam and 300 ml of milk
- 5 dried dates and 200 ml of milk
- Small square of flapjack and 300 ml of milk
- 1 milk-based 1.5 kcal/ml supplement and 2 plain biscuits

Will 'healthy eating' diets help patients with liver disease and diabetes?

The benefits of healthy eating depend on the patient. Currently, diabetes and obesity are

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endemic and as a consequence there is an increase of patients with NAFLD which is now the commonest cause of liver disease in the postindustrial world (de Alwis and Day, 2008). NAFLD is classed as the hepatic consequence of metabolic syndrome. Type 2 diabetes, insulin resistance and central obesity are key factors in developing NAFLD and in the more serious cirrhotic outcome, non-alcoholic steatohepatitis (NASH).

Patients with type 2 diabetes and NAFLD who have a body mass index (BMI) over 25 kg/m² and central obesity should be following a heart healthy, diabetic diet and aiming for the tightest glycaemic control possible. They should also be advised to lose weight aiming for a BMI of 25 kg/m². Weight loss should not exceed 1.5 kg a week, as rapid weight loss can be detrimental to liver function (Targher et al, 2007).

The use of oral hypoglycaemic drugs and insulin should be maximized and reviewed regularly. In diabetes patients with liver disease high cholesterol and hypertension are common. Giving heart healthy diabetic advice is appropriate for these conditions too. Drug treatment may be necessary. All patients should be encouraged to exercise for a minimum of 30 minutes a day as this improves all aspects of the metabolic syndrome.

Some patients with diabetes will develop end-stage liver failure with NASH and other liver diseases. Nutritional support is often needed in these patients and diets rich in calories and protein are advised. Diabetic advice is simplified and, depending on symptoms, food such as fruit, vegetables and high fibre wholegrain foods may be discouraged. This may be advised in patients with a poor appetite and early satiety owing to ascites. Oral nutritional support products can be prescribed in patients with diabetes and diabetic medication adjusted, if necessary, to avoid hyperglycaemia. Weight loss should not be advised in end-stage liver disease unless closely supervised by a dietitian (Alvares-da-Silva and Reverbel, 2005; Plank et al, 2008).

Should patients with liver disease be on a low-salt diet?

Historically all patients with liver disease were advised to follow a salt restricted diet and sometimes as low as 40 mmol of sodium a day (2.3 g of salt). Fortunately for the patients, this is no longer recommended as it was practically difficult and made diets highly unpalatable. Only in patients with ascites should a no-added salt diet be recommended if appropriate.

Ascites is the most common complication of cirrhosis and only occurs once portal hypertension has developed. The development of ascites in cirrhosis indicates a poor prognosis with mortality; approximately 40% at 1 year and 50% at 2 years (Guevara, 2005).

A no-added salt diet is 80–100 mmol of sodium a day which equates to 4.6–5.75 g of

salt. There is no evidence to restrict sodium or salt intake below this level (European Association for the Study of the Liver, 2010).

If patients are eating well, basic salt reducing advice includes not adding salt at the table and minimizing convenience foods and salty snacks. Some patients may find it helpful to count salt intakes. However, any food can be included in the diet as long as 5.75g of salt a day is not exceeded.

How much is too much salt?

Patients can be advised to read food labels to help judge whether a food is high in salt or not. The label will state how much salt is in the food per 100g. It is possible to work out if the food is high, medium or low in salt (Table 1).

It is important to consider that once ascites has developed prognosis is poor and patients have end-stage liver disease. In patients with ascites, protein-calorie malnutrition is common and patients’ intakes can be severely limited owing to early satiety, secondary to the ascites volume (Plauth et al, 1997).

On assessment it may become apparent that patients’ intakes are well below 80mmol of sodium and restriction is not necessary and may even hinder their intake and worsen protein-calorie malnutrition and outcome. Nutritional support is often necessary and most oral sip feeds can be fitted into a no-added salt diet. Oral fluid restriction may be recommended if the patient is hyponatraemic or drinking excessive amounts with rapid accumulation of ascites. Individual assessment is necessary and fluid always restricted under medical guidance.

Do people with liver disease need extra vitamins and minerals?

Some patients with liver disease do need extra vitamins with the following considerations.

Bone health in liver disease

An important complication of chronic liver disease is osteodystrophy which includes osteoporosis and osteomalacia. The reasons for this are multifactorial including poor intake and absorption of calcium, poor vitamin D synthesis, malnutrition and steroid use in cer-

tain liver conditions. The recommended intake for patients with cirrhosis is calcium 1000mg and vitamin D 800iu daily (Collier et al, 2002).

Vitamin considerations

Water soluble vitamins

People who are malnourished owing to alcoholic liver disease may particularly lack the vitamin B group. Vitamin B1 thiamine supplements are necessary in alcoholism as alcohol metabolism is dependent on thiamine as a cofactor. Patients should be prescribed thiamine and additional B vitamins at a treatment level if they are drinking alcohol at harmful levels, or are alcohol-dependent. Pabrinex, an intravenous vitamin B and C preparation, is prescribed for rapid therapy of severe depletion during hospital admissions. This is indicated in acute alcohol withdrawal where a severe depletion of thiamine can lead to Wernicke’s encephalopathy.

Smaller doses of B vitamins should be prescribed as outlined by the National Institute for Health and Clinical Excellence (NICE) (2006) guidelines and may be indicated in malnourished patients with decompensated liver disease patients or those at risk of refeeding syndrome.

Fat soluble vitamins (A, D, E and K)

Dietary deficiency of fat soluble vitamins can be found in severe cholestatic liver conditions such as primary sclerosing cholangitis and primary biliary cirrhosis. Stores of fat-soluble vitamins can last up to 3 months so people with severe jaundice for longer than this, or those who have malabsorption, should have levels checked and supplemented if indicated.

Vitamin A

This family of fat-soluble compounds, retinoids, have vitamin A activity. Retinol is the predominant form and is important for vision particularly at night. Deficiency is shown by xerophthalmia (night blindness).

Vitamin D deficiency

Osteomalacia is caused by the dietary deficiency of vitamin D or lack of sunlight exposure. Vitamin D undergoes 25-hydroxylation in the liver which is impaired in the presence of severe chronic liver disease. Additionally, cutaneous synthesis of vitamin D in the presence of jaundice is impaired and common in patients with cholestatic conditions such as primary biliary cirrhosis or primary sclerosing cholangitis.

Table 1. Measurement of salt and sodium levels			
	High	Medium	Low
Salt (per 100g)	>1.5g	0.3–1.5g	<0.3g
Sodium (per 100g)	>0.6g	0.1–0.6g	<0.1g

Vitamin E

Deficiency of vitamin E is rare but can be observed in chronic cholestatic liver disease. Symptoms include muscle weakness, ataxia and haemolysis. In advanced primary sclerosing cholangitis this can be seen in 40–50% of patients (Lee, 2002).

Vitamin K

Vitamin K is essential for normal clotting and bone metabolism. Deficiency results in clotting disorders which are frequently found in patients with liver disease. Supplementation can be given via intramuscular or intravenous injection.

Antioxidants

There is no evidence for the use of antioxidants in liver disease (Bjelakovic, 2011).

Should patients with liver disease take herbal remedies?

There is a lot of information available on diet, complementary and alternative medicines in the media and on the internet, with many people and organizations offering advice.

Traditional herbal medicines are not regulated through large trials in comparison to the process for prescribable medication. Many complementary and alternative medicines are promoted to benefit the liver or treat symptoms of liver disease. However, many of these are processed by the liver, so can be toxic to people with liver disease and can cause liver disease.

Health professionals are not clear on the role and place of some therapies in managing liver disease and while more research is awaited the products should not be recommended. If patients are thinking of using alternative or complementary medicines they should be advised to discuss this with a doctor or dietitian.

Conclusions

Protein calorie malnutrition is extremely common in liver disease, irrespective of the aetiology. Meeting nutritional requirements for both macronutrients and micronutrients is essential. Restriction of specific nutrients to treat symptoms must be carefully considered to prevent deterioration of nutritional status. NAFLD should be treated with weight loss advice and promotion of tight glycaemic control in patients with diabetes. Herbal preparations are not recommended.

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KEY POINTS

- Historically low-fat and low-protein diets were advised for patients with liver disease, which can lead to malnutrition
- It is important to treat malnutrition and elevate nutritional requirements
- Only in patients with ascites should a no-added salt diet be recommended
- Extra vitamins and minerals should be given to patients with particular complications of liver disease

Further information

British Dietetic Association

www.bda.uk.com/foodfacts

British Liver Trust

www.britishlivertrust.org.uk

Diabetes UK

www.diabetes.org.uk