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Review

A practical approach to nutrition in people with cirrhosis

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► Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/flgastro-2025-103201).

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Received 14 August 2025
Accepted 21 October 2025

To cite: Carbonell MJ, Graydon M, Mahmood S, *et al*. *Frontline Gastroenterology* Epub ahead of print: [please include Day Month Year]. doi:10.1136/flgastro-2025-103201

ABSTRACT

Cirrhosis is the end-stage of chronic liver disease and clinically can manifest with a spectrum of disease; patients with compensated cirrhosis may have a normal lifespan, yet those who decompensate have greatly increased morbidity and mortality. Optimising the nutrition of patients with cirrhosis helps prevent decompensation and also addresses the complications of decompensated cirrhosis, with tangible benefit to patients. The prevalence of cirrhosis is rising worldwide, and increasingly, patients with cirrhosis come under the care of non-hepatologists. Multiple society guidelines have been published describing nutritional intervention in the patient with cirrhosis, but these can be inaccessible to a non-specialist. Here, a simple, practical framework for how nutrition can be integrated into the care of patients with cirrhosis at various stages is presented, with reference to a patient narrative.

KEY POINTS

⇒ Nutritional intervention in patients with cirrhosis is important, particularly as an increasing number are likely to be managed by non-hepatologists due to rising disease prevalence.
⇒ A quick nutritional assessment, and providing a brief explanation to the patient of the importance of nutrition in cirrhosis, can be high yield with regard to patient outcomes.
⇒ Malnutrition and sarcopenia are common in patients with cirrhosis and associated with mortality, inpatient length of stay, hepatic encephalopathy, ascites severity and variceal bleeding.
⇒ Patients with cirrhosis are at risk of a rapid shift to starvation physiology and as such should eat three to five meals per day, as well as a late evening snack containing carbohydrates and proteins.
⇒ Micronutrient deficiencies and their sequelae are common in patients with cirrhosis: these should be screened for and intervened upon (often empirically).
⇒ A high protein diet is protective against hepatic encephalopathy.
⇒ Osteoporosis is common in patients with cirrhosis and should be screened for with a low threshold for treatment with a bone-sparing agent
⇒ Where a low-salt diet is limiting nutritional intake in patients with ascites, this can safely be relaxed so long as diuretics are appropriately titrated.

INTRODUCTION

The incidence and mortality of chronic liver disease (CLD) are rising,[1] imposing an increasing burden on health services.[2] As such, all healthcare professionals should expect to see more patients with cirrhosis in clinical practice: it is not sustainable that all of these patients will be managed by hepatologists. The rising incidence of CLD also means that access to dietetic review will come under increasing strain. Therefore, it is important that all clinicians who care for patients with cirrhosis understand nutritional intervention in the different stages of cirrhosis. A better understanding of nutrition among clinicians and patients improves patient outcomes, including re-admission rate, quality of life and mortality.[3 4]

Understanding of the role of malnutrition in the natural history of cirrhosis, as well as the importance of nutritional assessment and intervention, has grown over recent years. This has been reflected by several society guidelines.[5–8] Malnutrition is common in liver disease, affecting up to 20% of patients with compensated and 60% of decompensated cirrhosis.[5] Malnutrition is associated with increased mortality, length of inpatient admission, ascites severity, encephalopathy, variceal

bleeding and transplant waiting list mortality.[9–12] The triggers for malnutrition in cirrhosis are multiple, often synergistic and worsen with time. This creates a cycle of malnutrition and disease progression unless intervened on. Similarly, the manifestations of malnutrition in cirrhosis are complex. Multiple, often concurrent, micronutrient deficiencies can drive patient morbidity. Sarcopenia—the reduction of skeletal muscle mass and function due to increased proteolysis from chronic inflammation and reduced hepatic glycogen stores—increases mortality.[11]

In this guide, a patient narrative has been used to illustrate the importance of nutrition in a variety of scenarios that will be recognisable to any physician who routinely manages patients with cirrhosis.

FIRST CLINIC VISIT

You are seeing a new patient, Ms Smith, in the clinic. She is a 55-year-old woman referred with an elevated Fibrosis-4 (FIB-4) score. She has type 2 diabetes mellitus and hypertension and an alcohol intake of 30 units per week. She has a body mass index of 34 kg/m^2. Her transient elastography liver stiffness measurement is 19 kPa, consistent with cirrhosis. A liver ultrasound demonstrates a coarse liver echotexture. On examination, she has no features of decompensation. You diagnose MetALD cirrhosis.

- **On first review of a patient with cirrhosis, clinicians should assess potential barriers to nutrition.**
- **Initial broad advice on the importance of nutrition in cirrhosis should be provided, to be personalised in future consults.**

There are many components to the initial review of a patient with cirrhosis and often limited time. Despite this, it is important that a brief assessment of nutrition is undertaken given its impact on patient outcomes and the potential benefits of early intervention. Asking a patient about barriers to effective nutrition can be high yield and can identify reversible drivers of malnutrition that, if addressed, may significantly alter the course of the patient's disease.

Symptoms that can prevent effective nutrition are common in patients with cirrhosis, including bloating (40%), anorexia (34%), nausea (16%), depression (16%) and anxiety (11%).[13] Symptoms may worsen with time: nausea may be present in 58% of patients with end-stage liver disease,[14] while taste abnormalities occur in 59% of decompensated patients (compared with 7% of compensated patients).[15] Oral disease, including xerostomia, periodontal disease, caries and edentulism, is common and may impair oral intake, warranting dental review.[16] Oral candidiasis is also more common in patients with cirrhosis[16] and should be treated with an appropriate antifungal agent, such as topical miconazole/nystatin or oral fluconazole. However, anti-fungals need selecting with care in CLD—due to factors including the risk of antifungal toxicity in liver disease, and rising rates of azole resistance[16]—and input from Pharmacy and Microbiology may be helpful. In the UK, the prevalence of liver disease in the most deprived areas is approximately fivefold compared with the most affluent; patients may struggle to make nutritious food choices due to poverty. Key barriers to nutrition that may respond to intervention are detailed in table 1. These symptoms and circumstances should be re-evaluated regularly.

It is important to give easy to follow nutritional advice. This supports the patient in making healthy dietary choices and introduces the concept of dietary modifications as an intervention in CLD. Recommendations are illustrated in figure 1. The British Liver Trust has an online guide for patients that contains clear recommendations for diet, to which patients can be signposted.[17]

Table 1 Barriers to effective nutrition and potential interventions to consider

Barrier to effective nutrition	Potential considerations
Nausea	*Consider:* constipation; medications (both prescribed and non-prescribed); lifestyle factors (alcohol/hydration); gut motility
Early satiety	*Consider:* diet; size/number of daily meals; gut motility; constipation; medication; mood (including possible depression)
Taste abnormality (dysgeusia)	*Consider:* micronutrient deficiency
Poverty	*Intervene with:* support services (alcohol/ tobacco/street drugs); signposting; community prescribing where available; liaison with housing officers or social workers may be appropriate, for example, to support appropriate accommodation
Low mood	*Intervene with:* support services/charities; potential role for pharmacotherapy if depressed, which is generally safe in cirrhosis
Bloating	*Consider:* constipation; diet (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols 'FODMAP' diet); medication (lactulose); ensure 'bloating' does not in fact refer to symptomatic ascites
Anorexia	*Consider:* taste abnormalities; food availability; snacks; this may also be a manifestation of low mood/depression
Oral health	*Consider:* micronutrient deficiencies *Intervene with:* dentist/hygienist review
Alcohol use disorder	*Intervene with:* opportunistic brief intervention; signposting to alcohol liaison services; pharmacotherapy

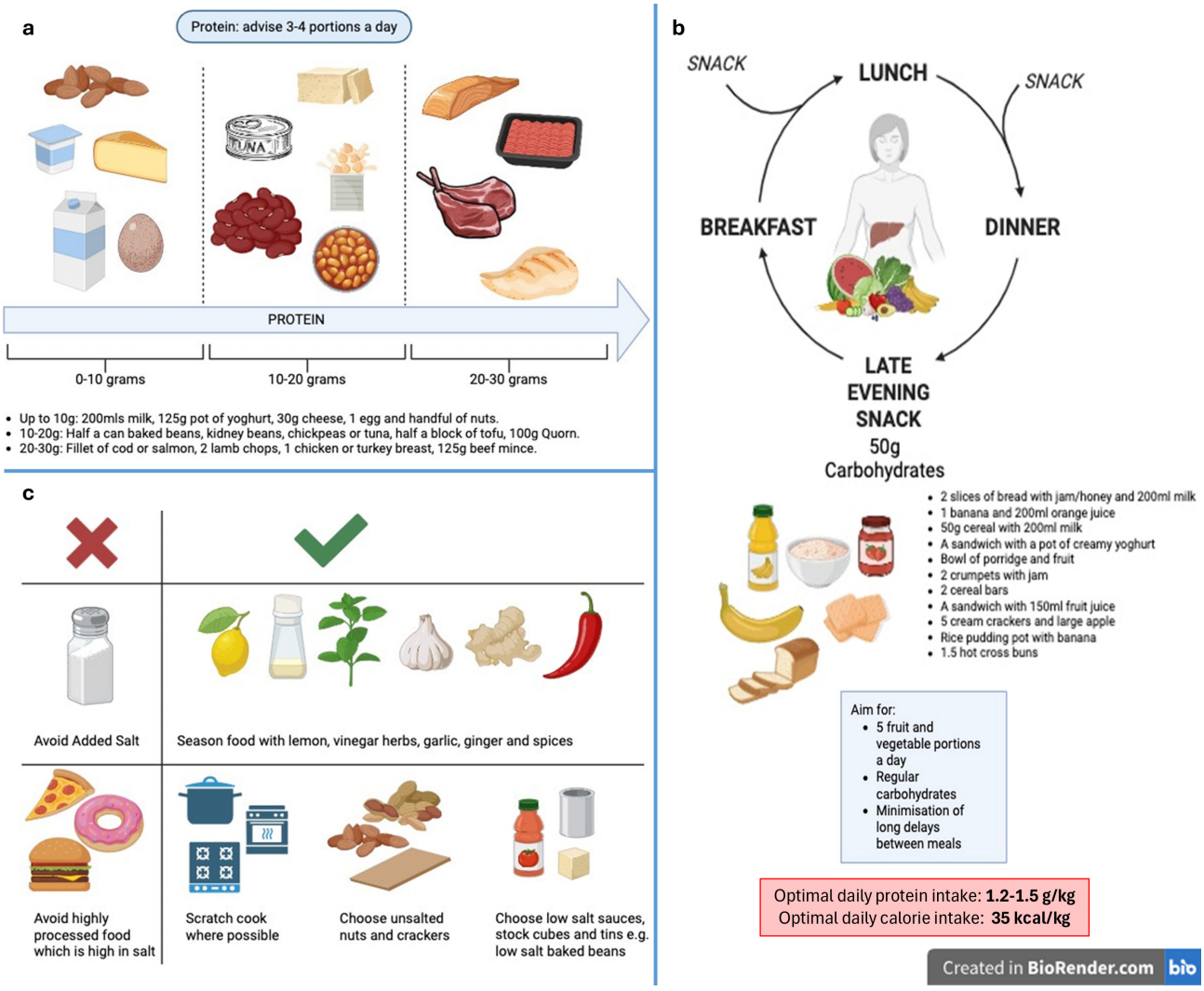

Figure 1 Simple nutritional recommendations for patients with cirrhosis. (a) Patients with cirrhosis should adhere to a high protein diet, choosing three to four protein-rich portions per day. (b) It is important that patients with cirrhosis consume regular meals throughout the day, including a late evening snack containing protein and carbohydrate. (c) Suggestions for improving adherence to a low-salt diet.

SECOND CLINIC VISIT: LIVER SUPPLEMENTS?

Ms Smith attends a routine follow-up appointment 6 months later. She has stopped drinking alcohol, although her diet is otherwise unchanged. Her liver disease remains stable. Since her last appointment, she has been researching liver disease on the Internet. Ms Smith has read that certain food supplements may benefit patients with cirrhosis.

- **There is insufficient evidence to recommend particular food supplements in cirrhosis.**
- **Coffee consumption has been linked to reduced hepatocellular carcinoma (HCC) risk and may be recommended.**
- **The role of probiotics is uncertain despite promise in preclinical settings.**

Questions regarding 'liver health' supplements are common. Herbal and complementary medicines are popular with patients, with almost $10 billion spent annually by US consumers on herbal products alone.[18] Patients may access these products freely, without medical assessment. Herbal and complementary medicines are associated with drug-induced liver injury (DILI), with more than 100 such preparations described as potentially toxic to the liver, accounting for approximately 16% of all DILI.[19] Table 2 summarises common supplements used by patients with liver disease.

Probiotics are a potential therapeutic approach with clinical appeal given the importance of the gut microbiome in CLD.[20] However, despite promise in preclinical settings, there is limited evidence that their use provides clinical benefit. A recent network analysis noted considerable uncertainty for the potential role of probiotics (and, indeed, any nutritional supplementation) in improving clinical outcomes in metabolic dysfunction-associated steatotic liver disease (MASLD).[21] As such, despite no adverse safety signal with their use, there is insufficient evidence to recommend routine probiotic use.

One recommendation that has been shown to be of potential benefit in patients with liver disease is coffee consumption. Moderate coffee consumption may slow

Table 2 Supplements commonly used in chronic liver disease (CLD)

Supplement	Significance of use in CLD
Milk thistle (*Silybum marianum*)	► Main constituent (silymarin) proposed to be hepatoprotective via anti-inflammatory, immunomodulating and anti-fibrotic effects.[71] ► Generally reported to be safe and well tolerated[72 73] and deemed by Liver Tox as unlikely to cause acute liver injury.[74] ► However, systematic reviews have yielded discrepant results with respect to clinical benefits:Some report a reduction in liver enzymes relative to placebo,[71 75 76] though the clinical relevance is unclear.[77]One review found silymarin to be associated with a reduction in liver-related mortality in patients with cirrhosis.[74] However, a separate Cochrane review reported a reduction in liver-related mortality that was no longer significant when analysis was restricted to high-quality trials.[73]Others found no reduction in mortality and no histological or biochemical improvement in patients with CLD taking silymarin.[72]Existing trials considered to be limited by low methodological quality.[73 77] ► Currently insufficient evidence to recommend use in CLD.
Chicory root	► Linked to significant decreases in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) in patients with metabolic dysfunction-associated steatotic liver disease in one 2023 meta-analysis.[78] ► Further study required to assess whether this translates to any clinical benefit.
Turmeric and curcumin	► Touted as having hepatoprotective properties, purportedly via anti-inflammatory effects and modulation of the metabolic syndrome. ► Recently linked to cases of clinically significant liver injury and should therefore be avoided.[79 80] ► Hepatotoxicity may be exacerbated by concurrent ingestion of black pepper.[81]
Green tea extract	► Used as weight loss supplement with additional claims of benefit in liver disease. ► Has been associated with clinically significant liver injury.[82 83]

the progression of liver disease, reducing the risk of liver fibrosis, cirrhosis and HCC, regardless of coffee preparation.[22] Guidelines support encouraging coffee consumption in patients with CLD.[23 24] ESPEN guidelines[5] suggest three–four cups of coffee a day is associated with the largest risk reduction, although this recommendation is not unanimous.[23 24]

There is also an established role for vitamin supplementation in liver cirrhosis, discussed later in this guide.

ADMITTED FOLLOWING A FALL: BONE HEALTH?

Four months later, Ms Smith falls in her garden onto an outstretched arm and sustains a Colles' fracture. This is managed non-operatively in her local Emergency Department. Could this have been avoided?

- **Osteoporosis is common in cirrhosis.**
- **All newly diagnosed people with cirrhosis should have a baseline DEXA scan.**
- **Patients with osteopenia or osteoporosis should receive calcium and vitamin D supplementation, as well as considering bisphosphonates.**
- **Vitamin D levels should be routinely checked and supplemented as required.**
- **Bisphosphonates are generally safe for use in liver disease.**
- **Consider pancreatic exocrine insufficiency (PEI) as a cause of fat-soluble vitamin deficiency.**

Osteoporosis is present in around 30% of patients with cirrhosis.[6] This translates to a higher incidence of fractures,[25–28] especially among patients with concomitant alcohol use disorder (AUD).[29] Other common risk factors include physical inactivity, post-menopausal status and cholestasis.[30 31] Patients with primary cholestatic disorders appear to be at higher risk than patients with other aetiologies of liver disease, with an incidence of up to 80%. This is likely related to reduced absorption of fat-soluble vitamin D.[32]

While the prevalence of vitamin D deficiency in patients with cirrhosis shows wide variation (36.9%–77.6%), vitamin D deficiency is consistently higher than the general population and correlates with liver disease progression.[33–35] Vitamin D is important for maintaining bone mineral density through promoting intestinal calcium absorption and regulating genes associated with bone metabolism. Deficiency can occur due to poor dietary intake, cholestasis, reduced sun exposure and reduced '25-hydroxylation by the liver. Therefore, all patients with cirrhosis should have a '25-OH vitamin D serum level measured regularly; pragmatically, this can be checked with their routine outpatient bloods every 6-12 months, taking into account expected seasonal differences. Oral supplementation should be started depending on the severity of deficiency, in line with local guidelines. If vitamin D remains in the deficient range despite a course of

oral replacement, consider factors such as malabsorption or poor compliance; in these cases, intramuscular supplementation may be considered. Patients should be instructed on foods that are good sources of dietary vitamin D, such as oily fish, mushrooms, egg yolk and fortified cereals.[34]

Reduced intestinal absorption of vitamin D and other fat-soluble vitamins may occur due to PEI. While typically associated with alcohol use, PEI is also associated with cirrhosis of any cause. A recent study found a prevalence of 14.6%, excluding patients with alcohol-related liver disease.[36] If suspected, due to fat-soluble vitamin deficiencies and/or steatorrhoea, a faecal elastase should be sent and pancreatic enzyme replacement therapy considered.

Given the risk of bone disease in cirrhosis, EASL guidelines recommend a DEXA scan at diagnosis; however, in practice, this may depend on local resources.[6] The surveillance interval will depend on the initial result, local nuclear medicine availability and the presence of other risk factors. However, patients with osteopenia (T-score between −1.5 and −2.5) or osteoporosis (T-score <-2.5) should have a repeat DEXA scan in 1 year to assess response to intervention. All patients identified to have osteopenia or osteoporosis should be started on calcium and vitamin D supplementation regardless of serum vitamin D level. Oral bisphosphonates are also recommended in all patients with osteoporosis and selected patients with osteopenia (although it is not fully established which patients with osteopenia are best suited for bisphosphonate treatment).[6] Bisphosphonates are not contraindicated in liver disease[37] and are safe in patients with varices without high-risk stigmata.[38] However, poor dental health is more prevalent among patients with CLD,[39] and this may theoretically increase the risk of osteonecrosis of the jaw. Further, patients with hepatic encephalopathy (HE) may be less compliant with sitting upright for 30 min after oral bisphosphonate administration, potentially increasing the risk of oesophageal injury. This risk is also likely to be increased after variceal sclerotherapy or band ligation and therefore should be avoided in the post-intervention period.[6]

CLINIC VISIT: NEW ASCITES

At her next follow-up appointment, Ms Smith discloses that since her wrist fracture, she has become more sedentary. Further, she has returned to drinking alcohol. She reports recent abdominal swelling. Clinical examination reveals moderate ascites, confirmed on subsequent ultrasound and diagnostic paracentesis (serum-ascites albumin gradient $>11 g/L$).

- **A reduced salt diet is recommended for patients with ascites.**
- **Fluid restriction does not play a role in ascites management and may be dangerous.**
- **Multivitamin status should be assessed routinely in decompensated patients; empirical oral multivitamin supplementation may be considered where this is not pragmatic.**
- **Oral thiamine should be given to patients with ongoing alcohol intake.**
- **All patients with decompensated cirrhosis should be referred for dietetics review.**

Restricted salt intake is universally recommended for patients with ascites. However, the nature of this restriction and how it is explained can be complex. General consensus is to restrict patients to around 80 mmol sodium/day (equivalent to 2 g sodium in food and 5 g added salt). Restriction to below <60 mmol sodium/day is often unpalatable, resulting in restricted calorie intake, which increases the risk of sarcopenia.[6 40] However, explaining this restriction to patients can be difficult, resulting in non-adherence: up to two-thirds of non-adherent patients may falsely believe they are following recommendations.[41] Advising a 'no added salt' diet and discussing which foods should be avoided (eg, ready meals) is easier for patients to follow. It is important to explain *why* salt restriction is necessary, as this may improve adherence.[42]

It is salt restriction and not fluid restriction that is beneficial in ascites. Ascites arises from portal hypertension driven activation of the renin-angiotensin-aldosterone system, which leads to renal sodium retention and consequent passive water retention. Therefore, the primary issue relates to salt, rather than water. Fluid restriction of patients with cirrhosis is difficult in practice. Patients with ascites are often oliguric; therefore, achieving a negative fluid balance can often require significant fluid restriction (<1 L per day), which may have subsequent effects on nutritional intake.[43] Patients with cirrhosis are particularly susceptible to pre-renal acute kidney injury (AKI), which is associated with increased mortality.[44 45] The risk of developing a pre-renal AKI may be increased by fluid restriction.[44]

Micronutrient deficiencies are common in patients with decompensated cirrhosis. Contributory factors include low oral intake, reduced hepatic storage and malabsorption.[6] Deficiencies may be in fat-soluble vitamins (A, D, E, K), water-soluble vitamins (including thiamine, B12 and folate), electrolytes or trace elements. The American Association for the Study of Liver Diseases (AASLD) guidelines recommend routine assessment of micronutrient status in patients with cirrhosis.[7] However, appreciating that micronutrient testing can be expensive and difficult to access, and given the relatively low cost and side effect profile of multivitamins, guidelines support commencing empirical oral multivitamins in patients with decompensated cirrhosis, where routine assessment is not pragmatic.[6 7 40] The British Society of Gastroenterology (BSG) guidelines also advise empirical supplementation of oral thiamine (100 mg twice daily) in all patients with decompensated cirrhosis with ongoing alcohol intake.[40] Nevertheless, if a

patient demonstrates signs or symptoms suggestive of a specific micronutrient deficiency, this should be tested for and therapeutically repleted as per local protocols.[5 6 40] AASLD guidelines provide a useful reference table outlining clinical manifestations of and treatment options for deficient micronutrients in cirrhosis.[7]

If a patient with compensated cirrhosis decompensates, for example, by developing ascites, they should ideally be referred for formal dietetic assessment. Decompensation may provide further challenges to nutrition. For example, HE may affect food acquisition, or variceal banding may result in dysphagia or dyspepsia, with resultant food avoidance. Ascites poses a particular challenge. These patients have increased energy requirement due to the energy cost of keeping ascites at body temperature and ascitic protein loss during recurrent large volume paracentesis (LVP).[5]

There is some evidence that transjugular intrahepatic portosystemic shunt (TIPS) placement may improve sarcopenia in patients requiring recurrent LVP.[5 7 46] However, sarcopenia prior to TIPS is associated with an increased rate of complications and mortality.[47 48] The presence of sarcopenia is therefore an important factor to consider in the work-up for TIPS insertion.

The development of decompensation in patients with cirrhosis should also prompt consideration of referral for liver transplant assessment. Both malnutrition and severe obesity are associated with worse post-transplant outcome,[49] and nutritional assessment by a dietician is mandatory for all patients prior to referral to a transplant centre.[50] Detailed guidance on preliver transplant nutritional optimisation is provided by ESPEN and EASL.[5 6]

TENSE ASCITES, STABLE WEIGHT

You next review Ms Smith as an outpatient 4 weeks later. Her measured weight is the same as at her previous appointment, but her ascites is now tense. She tells you that she does not often feel like eating and has been struggling to comply with her low salt diet, as she finds food to be bland and unpalatable.

- **Weight and body mass index can be misleading in patients with ascites.**
- **The benefits of salt restriction must be weighed against consequent reduced nutritional intake.**

Clinical progression of ascites with a stable weight reflects the pitfalls of using weight or BMI as a marker for nutritional status in decompensated cirrhosis.

Sarcopenia is common in cirrhosis, particularly in patients with decompensated cirrhosis, and is associated with a higher complication rate and increased mortality[11] but can be difficult to diagnose. Physical examination can be used to screen for sarcopenia, noting loss of muscle mass in the calves, deltoids, temples and the interosseous muscles; however, interobserver variability is high. As patients with cirrhosis often have cross-sectional imaging for various reasons, using these images to evaluate sarcopenia may be possible. Cross-sectional abdominal muscle mass at the level of the L3 vertebra below 50cm^2/m^2 for men and 39cm^2/m^2 for women is associated with mortality.[6 7 51] It is important to note that obesity does not preclude sarcopenia; with the rising incidence of MASLD, comorbid obesity and cirrhosis will be increasingly seen. Sarcopenic obesity represents a specific management challenge, as nutritional interventions for obesity may worsen sarcopenia.[52] Any patient with cirrhosis with suspected sarcopenia should be prioritised for dietetic consultation. Physiotherapy can be helpful, if accessible: aerobic and resistance exercises can improve muscle function and mass in patients with cirrhosis.[53] This is of particular importance in sarcopenic obesity.

In patients decompensated with ascites, adherence to a low salt diet is often poor, reported as low as 31%.[41] Adherent patients only consume 80% of the calories consumed by non-adherent patients.[41] Hence, if there are concerns that nutritional intake may be insufficient in a salt-restricted patient, targets should be relaxed, although there is limited guidance as to what the new target should be.[6 7] Interestingly, different levels of salt restriction do not significantly affect ascites management when patients are on diuretics. This suggests that if nutritional intake is impaired, relaxation of salt restriction is probably safe if diuretics are titrated appropriately.[54 55]

Dysgeusia is common and often under-recognised in patients with cirrhosis. In addition to salt restriction, deficiencies in vitamin A, zinc and magnesium may be contributory[5 56] and should be considered in those reporting altered taste sensation.

Oral nutritional supplements may be useful in helping decompensated patients meet the nutritional requirements outlined in figure 1. The BNF provides a useful breakdown of the nutritional content of commonly used supplements.[57] For decompensated patients who are unable to achieve adequate oral nutrition despite supplements, a short-term course of enteral feeding may be considered.[40] One case series of 14 patients with cirrhosis, decompensated with ascites, who were treated with a 7-week course of continuous nasogastric feeding, found significant improvement in nutritional status as well as reduction in ascites and paracentesis requirements across these patients.[58]

ADMISSION: VARICEAL BLEED

Ms Smith is brought in by ambulance to the Emergency Department with haematemesis. Her international normalised ratio (INR) is 2.0.

- **There is minimal evidence to support the use of vitamin K in variceal bleeding.**
- **The presence of oesophageal varices is not an absolute contraindication to nasogastric tube insertion.**

The association between prolonged INR and coagulopathy in cirrhosis has been discussed extensively.[59–61] INR is not a reliable marker of coagulation in this context.[60] Regardless, vitamin K supplementation is

frequently given to patients who present with variceal bleeding to correct a potential underlying vitamin K deficiency. An average decrease of INR between 0.08 and 0.31 has been found with vitamin K supplementation.[62 63] However, no studies have assessed whether vitamin K supplementation decreases bleeding risk in patients with CLD. Vitamin K supplementation is unlikely to cause harm, with an anaphylaxis rate of 0.03%, comparable to most other medications.[64]

Expert opinion is that enteral nutrition should be withheld for 48–72 hours after acute bleeding due to the risk associated with increased splanchnic blood flow.[6] However, one small randomised controlled trial of 22 patients did not find an increased risk of rebleeding with nasogastric feeding during the first 4 days after variceal bleed.[65] The theoretical risk of rebleeding with enteral nutrition must be balanced against the significant risk of prolonged starvation in patients with cirrhosis.

It is important to note that the presence of oesophageal varices, in itself, should not be considered a contraindication to nasogastric tube insertion.[5] There is no current evidence that fine-bore nasogastric tube insertion represents a substantial risk in patients with oesophageal varices[5] in contrast to the established risks of inadequate nutrition.

DELAYED GASTROSCOPY, PROLONGED NBM

Ms Smith is made nil-by-mouth (NBM) pending gastroscopy. Unfortunately, this does not occur, and she is kept NBM throughout her first night in hospital and into the next day, when she is once again listed for gastroscopy.

- **Patients with cirrhosis enter a starvation physiology after short periods of NBM**
- **They are at high risk for refeeding syndrome and Wernicke's when nutrition is restarted**
- **A bedtime snack can prevent overnight starvation**
- **NBM periods should be limited and, when unavoidable, consideration should be given to administration of intravenous glucose.**

A patient with cirrhosis does not respond to starvation in the same way as a healthy individual. Depleted hepatic glycogen stores mean that an overnight fast is sufficient to switch to protein catabolism as a source of substrates for gluconeogenesis, something that is only seen in healthy individuals after prolonged periods of starvation. As a result of this 'accelerated starvation', short periods of fasting can lead to muscle breakdown and worsening sarcopenia.[5] As such, it is of particular importance that the timing of a patient's oral intake throughout the day minimises periods of fasting. Patients with cirrhosis should have three to five meals throughout the day, and a late evening snack before going to sleep.[5 6] A late evening snack can improve numerous metabolic parameters, including muscle breakdown, to an extent not seen with isocaloric snacks given during the day. The nature of this snack does not appear to be important, as it is the energy content that prevents the switch to starvation physiology.[66] It is recommended that patients choose something that they enjoy, containing a mix of protein and carbohydrate.[7]

Specific consideration must be given to periods of NBM around procedures, with an effort made to provide a time for any such procedure and avoid moving this—prolonging a NBM period in a patient with cirrhosis is not the same as doing so in a patient without cirrhosis. Similarly, once NBM status is no longer required, patients with cirrhosis should recommence oral nutrition without delay. If a prolonged period of NBM is unavoidable, then an intravenous infusion of 5% or 10% glucose to provide 2–3 g/kg/day should be considered.[5] This is insufficient to meet calorie requirements but may prevent a switch to protein catabolism. The benefits of a glucose infusion should be weighed against the risk of third-space fluid accumulation and hyperglycaemia secondary to concomitant insulin resistance.

Patients with cirrhosis are sensitive to developing refeeding syndrome and Wernicke's encephalopathy when nutrition is recommenced after a period of fasting. Refeeding syndrome occurs due to the rapid mobilisation of potassium, magnesium and phosphates in response to insulin release, with potential multisystem complications. One study identified refeeding hypophosphataemia in 24% of patients with cirrhosis on restarting nutrition in hospital.[67] Wernicke's encephalopathy occurs due to rapid utilisation of an already reduced thiamine reserve in response to restarting nutrition. Although classically associated with AUD, thiamine deficiency has been identified at similar rates in cirrhosis secondary to hepatitis C, suggesting that it ought to be considered regardless of aetiology.[68]

HEPATIC ENCEPHALOPATHY

One week into her admission, Ms Smith becomes acutely confused. On review, she is disorientated and has asterixis. She is diagnosed as having developed HE.

- **Malnutrition and sarcopenia are risk factors for developing HE.**
- **Protein restriction is deleterious in HE.**
- **Bedside swallow assessment should be performed.**

HE is closely related to nutritional status. HE occurs due to impaired hepatic clearance of ammonia and other toxins, in combination with increased systemic inflammation. Besides the liver, muscle provides an alternative site for ammonia clearance through the synthesis of glutamine. Consequently, sarcopenia is a risk factor for developing HE.[11 48] It has previously been thought that a low protein diet in HE would be beneficial through reduction in ammonia generation; however, the opposite has now been demonstrated: low protein intake has been linked with worsening HE.[69]

Patients with HE may develop an unsafe swallow, through suppression of the gag and cough reflexes.

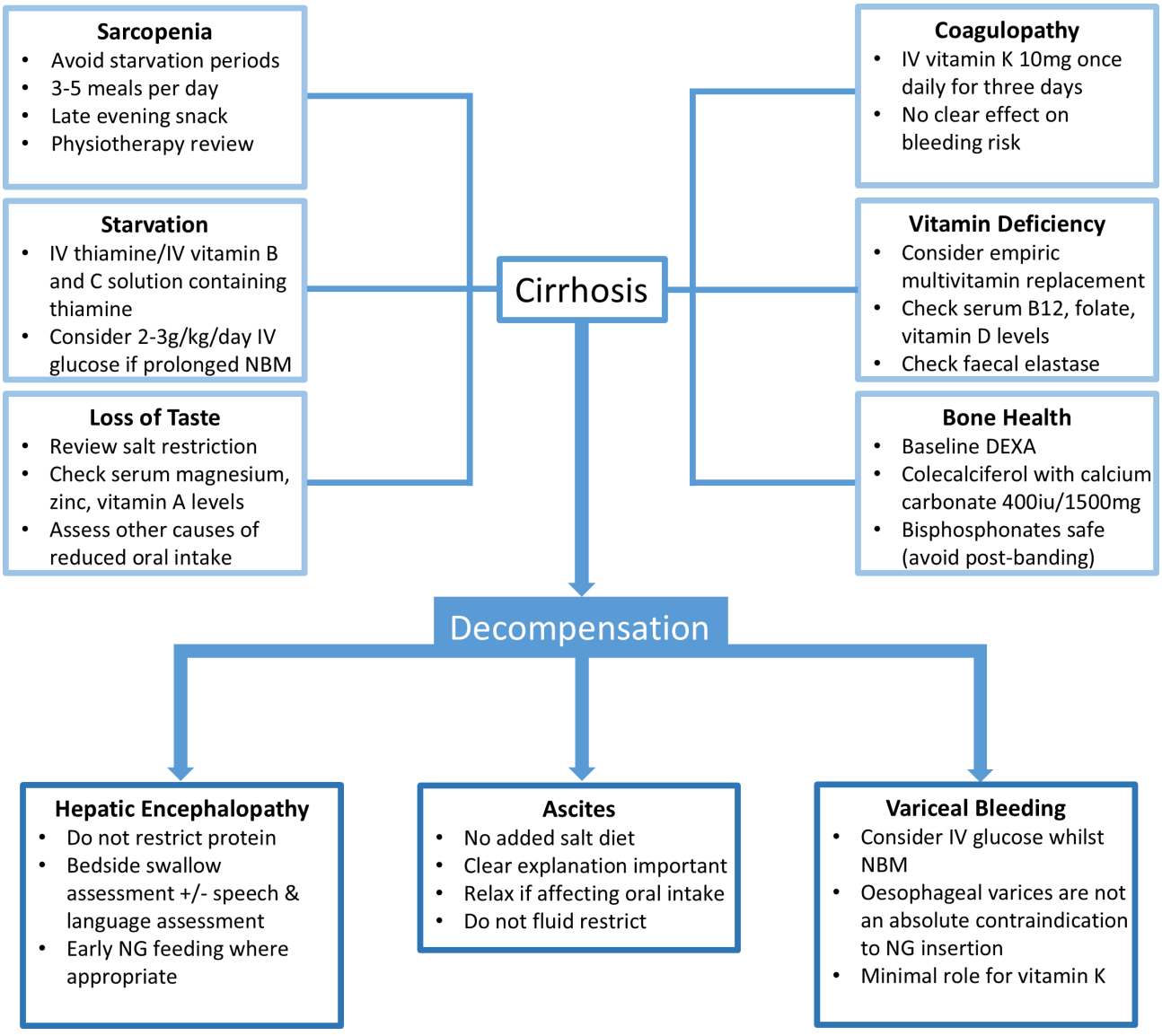

Figure 2 Summary of recommendations. IV, intravenous; NBM, nil-by-mouth; NG, nasogastric.

In a study of 294 patients with cirrhosis who were admitted with HE, 31.9% of patients had a tracheobronchial aspiration event. This was associated with a significantly increased mortality.[70] As such, patients diagnosed with HE should have a bedside swallow assessment performed, with onward referral for formal assessment by the speech and language team as appropriate.

Decisions regarding nutritional support should be guided by the degree of encephalopathy and ability to tolerate oral feeding. Careful consideration is required before placement of a nasogastric tube because of the potential for feeding tube non-tolerance and self-withdrawal in encephalopathic patients, which risks complications such as aspiration. For patients with HE who are able to eat, oral nutrition is preferable.[6] The use of enteral or parenteral feeding is particularly recommended for patients with grade III–IV encephalopathy, who are incapable of oral intake.[6]

CONCLUSION

The prevalence of liver cirrhosis is rising; hence, the care of patients with cirrhosis will increasingly fall under non-specialist clinicians. Moreover, access to dietetic input for these patients is often limited by resource, even within specialist centres. Alongside a call for increased funding for liver dietetic services, it is important that all clinicians managing patients with cirrhosis understand the integral role that nutrition plays in their management. Recommendations are summarised in figure 2. We hope that this guide will improve clinicians' familiarity with key nutritional concepts when managing patients with cirrhosis and will help clinicians feel more confident incorporating these into their practice.

Contributors MJC wrote the first draft of the manuscript with contributions and critical review from all authors. BHM and LDT are joint corresponding authors. LDT guarantees the manuscript.

Funding The Division of Digestive Diseases at Imperial College London receives financial support from the National Institute of Health Research (NIHR) Imperial Biomedical Research Centre (BRC) based at the Imperial College London and Imperial College Healthcare NHS Trust. BHM is the recipient of a Medical Research Council (MRC) Clinician Scientist award (MR/Z504002/1).

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Commissioned; externally peer-reviewed.

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