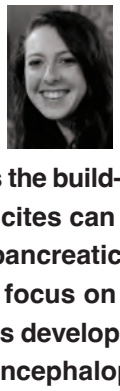
Ascites Management

A focus on patients with cirrhosis of the liver



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Ascites is the build-up of excess fluid in the abdominal cavity and is a multifactorial and complex process.[1] Whilst ascites can be a common symptom of liver cirrhosis, it can also occur due to other conditions such as pancreatic disease, heart failure, and cancers of the stomach, bowel or ovary.[1] This article will primarily focus on ascites secondary to liver cirrhosis.

Ascites develops in those with liver decompensation, among other clinical features such as jaundice, hepatic encephalopathy and variceal bleeding.[2] A grading system is used to determine the volume of ascites and appropriate treatment required, as shown in Table 1.[2]

Table 1: Ascites grading system

Grading	Severity	Treatment
Grade 1	Mild	No treatment
Grade 2	Moderate	Salt restricted diet & diuretics
Grade 3	Large/Tense	Paracentesis, salt restricted diet & diuretics

Refractory ascites

Refractory ascites refers to fluid that no longer responds to standard medical treatment or rapidly reaccumulates despite appropriate management. It is usually seen in advanced liver disease and can significantly impair quality of life.[3]

It is divided into two subtypes:

- Diuretic-resistant ascites: Fluid persists despite strict sodium restriction and optimal diuretic therapy.[3]
- Diuretic-intractable ascites: Diuretics cannot be continued due to complications, such as acute kidney injury, electrolyte imbalance or worsening hepatic encephalopathy. In these cases, alternative treatments are required.[3]

Ascites & liver cirrhosis

Pathophysiology

One of the main drivers for hepatic ascites formation in cirrhosis is portal hypertension (PHT).[3, 4] PHT causes the body to produce higher levels of substances that widen blood vessels, while also slowing their breakdown. These substances affect how tightly or loosely blood vessels are regulated and include nitric oxide and other naturally occurring vasodilators.[4]

As a result, blood vessels supplying the abdominal organs widen significantly, causing blood to pool in this area rather than circulate efficiently around the body. This leads to reduced effective blood flow to vital organs, despite the body often holding excess fluid.[3, 4]

This state of 'effective low blood volume' plays a key role in ascites formation. In response, the body activates several hormone and nervous system pathways designed to maintain blood pressure by retaining sodium and water. These include the renin–angiotensin–aldosterone system, the sympathetic nervous system and antidiuretic hormone.[3, 4]

The kidneys are particularly affected by these changes. Reduced blood flow to the kidneys leads to further sodium and water retention, worsening fluid build-up and increasing the risk of complications, such as refractory ascites and hepatorenal syndrome-chronic kidney disease (HRS-CKD).[4]

Diagnosis

Initial assessment includes a detailed history, physical examination, abdominal ultrasound, and blood tests to evaluate liver and kidney function.[5] Additional investigations include serum and urine electrolytes, and analysis of ascitic fluid.[5]

Diagnostic paracentesis is recommended for individuals presenting with new-onset ascites or clinical deterioration, including gastrointestinal bleeding, hepatic encephalopathy, infection or worsening renal function.[3] This procedure involves inserting a needle into the abdomen to remove fluid for analysis and symptom relief.[4] Patients with cirrhosis and ascites should also be assessed for spontaneous bacterial peritonitis (SBP). If suspected or confirmed, prompt antibiotic treatment is required.[3]

Management & treatment

Paracentesis

Paracentesis is indicated for new-onset or large-volume ascites and provides rapid symptom relief, improving abdominal discomfort, breathlessness and early satiety.[3] Evidence suggests that early paracentesis (within 12 hours of hospital admission) may reduce complications, shorten hospital stay and improve outcomes.[6]

Transjugular intrahepatic portosystemic shunt (TIPS)

TIPS is a well-established and effective interventional treatment for people with portal hypertension, variceal bleeding, and recurrent or refractory ascites.[7] The procedure involves placing a shunt within the liver to create a channel between the portal vein and the hepatic vein, allowing blood to bypass the liver and thereby reduce pressure within the portal circulation.[8]

Research shows that TIPS can reduce the risk of recurrent or worsening ascites, lower liver-related mortality, and decrease the likelihood of further liver decompensation.[9, 10] However, TIPS is not suitable for everyone, and careful patient selection is essential, as potential risks include worsening hepatic encephalopathy, heart strain and shunt dysfunction, which means ongoing monitoring and follow-up are required.[7]

Diuretics

Diuretics are a cornerstone of treatment for moderate ascites.[3] Spironolactone is typically used as first-line therapy, with furosemide added if a faster response is needed.[3] These medications increase urine output to remove excess fluid.[3]

Close monitoring is essential, as diuretics can cause complications such as electrolyte imbalance and hypovolaemia. Treatment should always be combined with dietary sodium restriction.[11]

Nutritional complications

Ascites is a major complication of advanced liver disease, and both reflects and drives worsening nutritional status and sarcopenia.[12] Sarcopenia, defined as the loss of skeletal muscle mass, strength and function, is strongly associated with poorer clinical outcomes, including increased susceptibility to infection, higher morbidity and elevated mortality risk.[12] It also independently predicts ascites development.[12] A retrospective study of patients with chronic liver disease identified sarcopenia as an independent risk factor for ascites formation.[12]

The mechanisms of malnutrition in ascites are multifactorial and reinforce one another. Reduced oral intake and increased nutritional requirements are central contributors.[13] Due to impaired hepatic glycogen storage, patients with cirrhosis enter a 'starved state' after only 3-4 hours of fasting, accelerating fat and protein catabolism and promoting rapid muscle loss.[13] Hypermetabolism, defined as an increase in resting energy expenditure (REE), occurs in approximately 18-34% of patients with cirrhosis and ascites further increases REE due to the physical burden of excess intra-abdominal fluid.[14] Mechanical symptoms such as abdominal distension, nausea, reflux and early satiety frequently limit appetite and food tolerance.[14]

Nutritional assessment

To minimise the confounding effects of fluid overload, conventional anthropometric measures such as weight, body mass index, and weight loss are unreliable, as fluid shifts mask true body composition changes.[15] Validated screening tools that adjust for fluid status are therefore recommended for assessing nutritional risk.[16]

Tools such as the Royal Free Hospital Nutrition Prioritising Tool (RFH-NPT) and the Liver Disease Undernutrition Screening Tool (LDUST) incorporate fluid status, intake and functional measures, demonstrating improved sensitivity and specificity in cirrhosis.[17]

Functional and body composition assessments are essential for detecting sarcopenia.[15] Hand grip strength is a simple, reproducible measure of muscle function and correlates strongly with clinical outcomes.[16] Mid-arm muscle circumference (MAMC) offers a bedside assessment of muscle mass which is less affected by ascites.[16, 18] Radiological assessment of sarcopenia remains the gold standard but is limited in clinical practice by cost and radiation risk.[19]

See **Table 2** for an overview of the strengths and limitations of assessment methods to identify sarcopenia.[18]

When calculating nutritional requirements, estimated dry body weights should be used. Adjustments can vary between 5% of actual body weight for mild ascites to 15% for severe ascites.[16] Post paracentesis weight can sometimes be useful but rarely reflects complete fluid removal.[16]

Nutritional management

Sodium restriction is a core component of management of ascites alongside diuretics. Current guidelines recommend that patients follow a no added salt or sodium-restricted diet, equivalent to approximately 5.0-6.6 g of salt (87-113 mmol sodium) per day.[3] Excess sodium intake can worsen fluid retention and contribute to the further accumulation of ascitic fluid, whereas reducing salt intake can improve the effectiveness of diuretic therapy and help support better fluid balance.[13]

Table 2: Assessment methods for identification of sarcopenia[18]

Assessment	Type	Strengths	Limitations
Hand grip strength	Functional	Fast, predictive of outcomes	Effort dependent, not a direct measure of muscle mass
MAMC	Anthropometric	Less affected by ascites	Requires correct technique
Tricep skin fold	Anthropometric	Less affected by ascites	Requires correct technique
CT L3 skeletal muscle index	Imaging	Gold standard for sarcopenia	Radiation; availability

Excessive sodium restriction can impair nutritional status by reducing palatability and lowering energy and protein intake. Although sodium restriction may reduce paracentesis frequency, it has also been linked to higher sarcopenia prevalence and increased 6-month mortality.[20]

Nutrition education is essential, as hidden sodium sources are common, and overly strict restriction can reduce caloric intake by up to 20%. Few patients achieve energy, protein and sodium targets simultaneously.[21, 22] Structured dietary counselling and nutrition education can improve adherence, resulting in better ascites control and improvements in quality of life.[13] This highlights the importance of a personalised approach that integrates symptom control with nutritional needs.[21]

Practical strategies for a no added salt diet include basing meals around high-protein, naturally low-sodium foods such as fresh or frozen meat, poultry, fish, eggs, plain legumes, tofu and plain dairy.[23] Processed meats, tinned fish in brine, ready-meals, takeaways and most hard cheeses are major contributors.[23] Replacing processed to fresh protein sources alone can reduce sodium by 1,000-2,000 mg/day without impacting protein targets.[23] Tastelessness is the main driver of reduced intake, and reduced palatability can be combatted by utilising herbs, spices, garlic and lemon juice.[23] See **Table 3** for practical tips on reducing sodium intake.

Energy & protein requirements

Patients with ascites often experience early satiety, abdominal discomfort and reduced appetite, leading to insufficient energy intake.[19] Alongside frequent meals and snacks to reduce fasting intervals, and a late evening carbohydrate snack, patients may benefit from energy dense, low-volume nutrition strategies.[19] Modular supplements – for example, isolated protein powders and carbohydrate modules – allow for targeted macronutrient delivery without increasing meal volume.[19] These are particularly useful for patients with significant ascites who cannot tolerate large meals but require concentrated sources of protein and energy.

The energy and protein requirements for liver disease patients with ascites are:[18, 19]

- **Energy:** 35-40 kcals/kg dry body weight
- **Protein:** 1.2-1.5 g/kg/day dry body weight.

Enteral feeding

When oral intake remains inadequate despite dietary optimisation, enteral feeding becomes an essential component of nutritional management. Evidence supports nasogastric (NG) tube feeding in decompensated cirrhosis for improving nitrogen balance, muscle mass and clinical outcomes.[19] NG tube feeding is generally safe in patients with ascites, including those with varices, provided there is no active gastrointestinal bleeding.[19] Energy dense (1.5-2 kcal/ml), high protein formulas are typically used to meet nutritional requirements while minimising volume burden.[19]

Enteral feeding has been associated with reduced ascites and paracentesis requirements, as well as improved nutritional requirements, and should be considered in patients who fail to respond to oral nutritional intervention.[24] Gastrostomy placement is a contraindication in this cohort due to the risk of peritonitis, stomal leakage and infection, and is associated with higher in-hospital mortality.[19, 25]

Conclusion

Overall, the evidence supports nutrition support as a therapeutic intervention, not merely supportive care. By improving protein status and functional muscle mass, nutrition support contributes directly to reduced ascites severity, improved tolerance to medical therapy, and better overall prognosis in advanced liver disease. For a dietitian managing these patients day-to-day, the core challenge is simultaneously achieving adequate energy and protein in the context of early satiety, dysgeusia, unpalatable low-sodium food and variable adherence.

Table 3: Practical tips and swaps for reducing sodium intake[23]

High salt food/practice	Alternative
Adding salt during cooking or at the table	No added salt; use herbs, spices
Processed meats (ham, bacon, sausages)	Fresh poultry, fish, lean meats
Tinned soups, sauces, ready-meals	Fresh or low salt labelled versions
Salted butter/spreads	Unsalted butter or plant-based spreads
Salted snacks (crisps, crackers)	Unsalted nuts, seeds, low salt crackers
Cheese (e.g. halloumi, processed slices)	Lower salt cheeses (e.g. mozzarella, ricotta)

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