International Journal of Advances in Medicine
Lestari IDAMD et al. Int J Adv Med. 2026 Jan;13(1):56-61
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20254146

Review Article

Management of intractable ascites in cirrhosis: a review article

I. Dewa Ayu Made Dian Lestari[1]*, Ketut Suryana[2]

[1]Departement of Internal Medicine, Wangaya Regional Hospital, Denpasar, Bali
[2]Department of Internal Medicine, Allergy-Clinical Immunology Services Unit, Wangaya General Hospital, Denpasar, Bali, Indonesia

Received: 12 September 2025
Revised: 15 October 2025
Accepted: 17 October 2025

***Correspondence:**
Dr. I. Dewa Ayu Made Dian Lestari,
E-mail: dwayudianlestari26@gmail.com

ABSTRACT

Intractable ascites (IA) also referred to as refractory ascites, is a severe complication of cirrhosis marked by resistance to standard diuretic therapy. Its occurrence reflects advanced portal hypertension and is associated with poor prognosis. This literature review summarizes current evidence and management strategies for IA in cirrhotic patients. Sodium restriction remains a core component of management, even in diuretic-resistant cases. Large-volume paracentesis (LVP) with albumin infusion is considered first-line therapy, though it carries the risk of post-paracentesis circulatory dysfunction. Long-term albumin administration shows potential benefits but requires further confirmation. In selected patients, transjugular intrahepatic portosystemic shunt (TIPS) offers sustained ascites control. For non-TIPS candidates, the alfapump system provides continuous ascites drainage, improving appetite, mobility, and quality of life, though long-term antibiotic prophylaxis is required to prevent device-related infection. Liver transplantation remains the only curative option, and combined liver–kidney transplantation should be considered in patients with concurrent chronic kidney disease. Management of IA in cirrhosis requires a multidisciplinary approach. While LVP and TIPS remain the therapeutic mainstays, emerging strategies such as long-term albumin therapy and alfapump devices are promising. Given its poor prognosis, timely evaluation for liver transplantation is crucial.

Keywords: Management of intractable ascites, Cirrhosis, Diagnostic examination

INTRODUCTION

Hepatic decompensation, characterized by ascites, hepatic encephalopathy, and gastrointestinal bleeding due to portal hypertension, is an important phase in the natural course of cirrhosis.[1] Ascites describes the condition of pathological fluid accumulation within the peritoneal cavity.[2] Ascites often serves as an early manifestation of decompensation, with an annual incidence of 5–10% in patients with compensated cirrhosis. Ascites is associated with a decrease in 5-year survival from 80% to 30%, due to increased risks of complications such as bacterial infections, electrolyte imbalances, hepatorenal syndrome (HRS), and nutritional imbalances, which collectively worsen the patient's clinical condition.[1] Therefore, the management of ascites is a primary focus in the clinical management of cirrhosis patients.[2]

Patients with cirrhosis who have clinically significant ascites and related complications should be considered for referral for liver transplantation evaluation and palliative care.[3] Therapeutic strategies continue to evolve as understanding of the pathophysiology and haemodynamics involved in the development and worsening of ascites improves.[2]

LITERATURE REVIEW

The research design used in this study is a literature review method, utilizing the keywords ‘management of intractable ascites’, ‘cirrhosis’, and 'diagnostic

examination,' to identify studies related to management of intractable ascites. The literature sources include PubMed, Google Scholar, Science Direct and EBSCO. The authors then reviewed each journal that met the criteria, engaged in discussions, and conducted cross-checks with other primary sources.

CLINICAL SYMPTOMS

Common clinical symptoms found in patients with ascites include increased abdominal girth, a feeling of fullness or discomfort in the abdomen, early satiety, limited mobility, and shortness of breath. Ascites due to cirrhosis typically develops relatively quickly, within a few weeks.[2] The onset of ascites is important to inquire about, as the etiology of rapidly developing ascites can be significantly different from that of slowly developing ascites, such as in peritoneal tuberculosis, malignancy, hepatocellular carcinoma (HCC), or hepatic insufficiency.[4]

Patients with cirrhosis may experience symptoms of hepatic decompensation, such as altered mental status (confusion) or signs of gastrointestinal bleeding. Symptoms such as fever, abdominal pain, and changes in mental status may be present in patients with spontaneous bacterial peritonitis (SBP). Patients with severe cirrhosis leading to ascites typically exhibit signs of chronic liver disease, such as palmar erythema and spider nevi. Splenomegaly and abdominal collaterals indicate portal hypertension. In advanced stages, other symptoms such as jaundice, muscle wasting, and leukonychia may also be present.[2] Ascites must be distinguished from other causes of abdominal distension, such as obesity, gas distension, intestinal obstruction, or abdominal cysts.[2] Clinical evaluation should include a history of exposure to cirrhosis risk factors and a physical examination to identify evidence supporting chronic liver disease or alternative diagnoses.[5] Flank dullness is the most sensitive physical sign and can be found in up to 90% of patients. Shifting dullness is more specific than flank dullness but has lower sensitivity.[2] Shifting dullness can be detected when approximately one and a half liters of free fluid accumulates in the abdominal cavity. This physical sign has a sensitivity of 83% and specificity of 56% in detecting ascites.[5]

Fluid wave or fluid thrill may be present in patients with massive ascites, although this sign has low accuracy.[2] In cases of obesity or smaller fluid volumes, imaging such as ultrasound or computed tomography (CT) is required to confirm the presence of ascites.[5] In patients with cirrhosis, ascites can be classified based on the amount of fluid accumulated in the abdominal cavity and categorized according to response to therapy (Table 1).[1]

DIAGNOSTIC EXAMINATION

Abdominal ultrasound is the first-line imaging modality for detecting ascites.[4] Ultrasound has been shown to have significantly higher sensitivity and specificity than physical examination in detecting free intraperitoneal fluid. Ultrasound can detect fluid volumes as small as 100 ml, and diagnostic accuracy increases with increasing fluid volume.[6]

Table 1: Ascites classification.

According to amount of fluid accumulation		According to the response to treatment	
Grade 1: mild ascites	Only detected by ultrasound	**Responsive ascites**	Ascites that can be fully mobilized or limited to grade 1 with diuretic therapy associated or not to moderate dietary sodium restriction
Grade 2: moderate ascites	Moderate symmetric distension of abdomen	**Recurrent ascites**	Ascites that recurs on at least 3 occasions within. A 12-month period despite dietary sodium restriction and adequate diuretic dosage
Grade 3: large or gross ascites	Marked distension of the abdomen	**Refractory ascites**	Ascites that cannot be mobilized or the early recurrence of which (i.e. after LVP) cannot be satisfactorily prevented by medical therapy

Diagnostic of ascites builded through a combination of clinical examination and abdominal ultrasound, that perform abdominal paracentesis to evaluate the ascites fluid. Abdominal ultrasound and diagnostic paracentesis are recommended by the British Society of Gastroenterology, the European Association for the Study of the Liver (EASL), and the American Association for the Study of Liver Diseases (AASLD) for all patients with newly diagnosed ascites. This procedure should also be performed whenever there is clinical deterioration in patients with previously diagnosed ascites. Paracentesis, accompanied by ascites fluid analysis, is the single most important procedure and should be the first step in evaluating patients with ascites.[2]

In cirrhosis, hepatic sinusoids become less permeable due to fibrotic tissue deposition, resulting in ascites with low protein levels, making it important to measure total protein levels in ascites fluid. Concentrations below 1.5 g/dl (or 15 g/l) are a risk factor for spontaneous bacterial peritonitis. Additionally, the serum-ascites albumin gradient (SAAG) should be routinely measured. A threshold value of 1.1 g/dl (or 11 g/l) distinguishes various causes of ascites with high sensitivity, although alternative causes should still be considered based on clinical conditions (Table 2).[5]

Table 2: Classification of ascites etiology based on SAAG.

SAAG >11/l	SAAG <11/l
Portal hypertension	Peritoneal carcinomatosis
Cardiac failure	Peritoneal tuberculosis
Portal vein thrombosis	Bowel perforation
Hypohyoidism	Nephrotic syndrome

DISCUSSION

Management of uncomplicated ascites

The term uncomplicated ascites refers to any ascites that is not intractable, infected, or associated with renal failure (hepatorenal syndrome). This condition includes mild ascites (grade 1) without symptoms or moderate ascites (grade 2). Management in these cases is based on a low-salt diet, diuretic therapy, paracentesis therapy, and even liver transplantation.[7]

Reducing sodium intake and increasing renal sodium excretion are the two main pillars of moderate ascites management. This is based on the role of positive sodium balance as a determining factor in peritoneal fluid accumulation. Current guidelines recommend sodium intake restriction of 80–120 mmol per day to prevent excessive sodium retention while minimizing the risk of hyponatraemia and renal dysfunction. With this restriction, ascites volume can decrease without causing significant malnutrition or calorie deficiency. Regular monitoring of nutritional and electrolyte status is necessary to ensure that sodium interventions giving negative impact of the patient's nutritional condition.[7,8]

In addition to a low-sodium diet, pharmacological management for grade 1 ascites begins with low-dose spironolactone (e.g., 100 mg/day). If there is no adequate response after a few days, the spironolactone dose may be titrated up to a maximum of 400 mg/day. Furosemide is added at a dose of 40–160 mg/day if weight loss is suboptimal. Daily monitoring of weight and urine volume is crucial to assess the effectiveness of therapy. A safe weight loss target is < 0.5 kg/day in patients without oedema and up to 1 kg/day in patients with residual oedema. Once the target is achieved, consideration should be given to tapering the diuretic to prevent overdiuresis. In mild to moderate ascites, intravenous fluid infusion is generally not required, and fluid restriction (<1.5 l/day) is only recommended if acute hyponatraemia occurs. The guidelines also emphasise the importance of monitoring diuretic side effects, particularly the risk of hypovolemic hyponatraemia. If severe hyponatraemia occurs during therapy, temporary discontinuation of diuretics and administration of normal saline should be considered.[8,9]

In cases of grade 3 ascites or massive, uncontrolled ascites volume, large volume paracentesis (LVP) is the primary treatment modality. LVP (>5l) is safe and effective for rapidly removing ascites fluid. Following large-volume paracentesis, intravenous albumin administration is crucial (6–8 g per litre of fluid removed) to prevent post-paracentesis circulatory dysfunction (PPCD). Albumin prevents the sharp post-procedural decrease in effective arterial volume that can trigger acute kidney injury or encephalopathy. In a repeated LVP regimen, each litre of ascites fluid contains approximately 21 g of protein (primarily albumin). This indicates that chronic LVP may exacerbate malnutrition and sarcopenia in patients. Albumin infusion after each LVP helps maintain blood volume but also imposes a significant cost and resource burden.[9]

Management of complicated (intractable) ascites

In advanced cirrhosis, intrahepatic fibrosis and vascular constriction increase portal flow resistance, causing hydrostatic pressure in the hepatic sinusoids to rise and plasma fluid to be forced into the peritoneal cavity. The accompanying splanchnic vasodilation reduces mean arterial pressure and effective arterial volume, activating the renin-angiotensin-aldosterone system, antidiuretic hormones, and other vasoconstrictors. This results in progressive sodium and water retention.

These changes in homeostasis ultimately lead to the accumulation of ascites fluid that becomes increasingly difficult to control in the advanced stages of the disease. In patients with intractable ascites, haemodynamic disturbances are difficult to manage, resulting in severely limited fluid mobilisation.[10]

Intractable ascites occurs in approximately 5–10% of patients with decompensated cirrhosis with a poor prognosis (mortality ~50% within 6 months).[1,9] Practically, intractable ascites is defined as ascites that cannot be mobilized or recurs rapidly after large-volume paracentesis (LVP), despite the patient having undergone sodium restriction and maximal diuretic doses.[1,9] Intractable ascites is divided into two subtypes: diuretic-resistant, which is ascites that persists despite maximal diuretic doses, and diuretic-intolerant, which is ascites that continues to occur because diuretic doses cannot be increased due to side effects such as renal dysfunction, hypotension, or hyponatraemia.[9]

The diagnosis of intractable ascites according to the International Club of Ascites (ICA) is ascites that does not improve with intensive diuretic therapy for at least one week, a low-salt diet with sodium intake less than 90 mmol per day, an average weight loss of less than 0.8 kg over four days, and urinary sodium output lower than sodium intake, recurrence of grade two or three ascites within four weeks after initial mobilization, or the development of complications caused by diuretic use.[8]

Currently, known management options for intractable ascites include pharmacotherapy, large-volume paracentesis, transjugular intrahepatic portosystemic shunt, and automated low-flow ascites pump (Figure 1).[11]

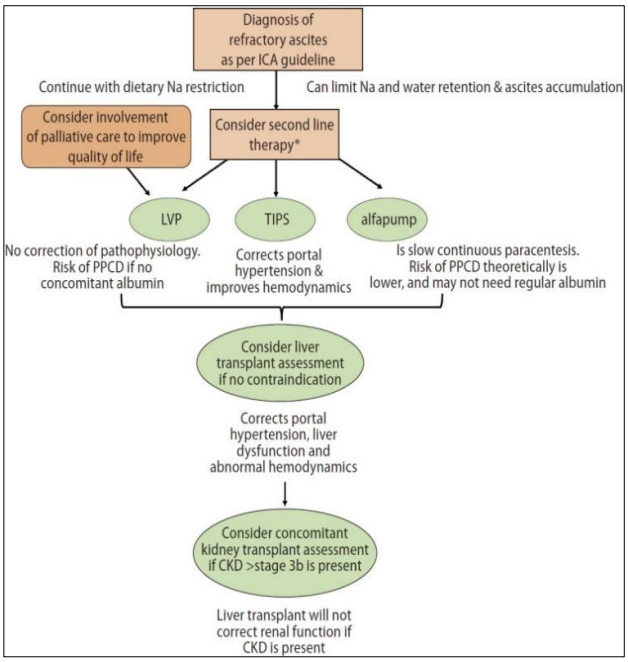

Figure 1: Diagram of Intractable ascites management.

Large volume paracentesis (LVP)

According to the recommendations of the American Association for the Study of Liver Diseases (AASLD), the primary management of intractable ascites is performed with periodic LVP. This therapy is generally considered effective and safe for patients, taking into account the volume of fluid removed. Plasma volume expansion with albumin (8 g/l of the ascitic fluid removed) must always be performed after LVP to prevent post-paracentesis circulatory dysfunction (PPCD). A lower albumin dose (4 g/l of ascitic fluid removed) has been proposed for patients undergoing paracentesis with an ascitic fluid volume of less than 5l. According to the main guidelines, in such cases, albumin administration may be considered on a case-by-case basis (e.g., patients at risk of renal dysfunction or failure).[8,12]

Pharmacological therapy

Pharmacological management using diuretics needs to be modulated and the risk of side effects such as worsening glomerular filtration rate and electrolyte disturbances must be considered. According to the guidelines of the European Association for the Study of Liver Diseases (EASL), diuretics should be discontinued in patients with intractable ascites who do not excrete >30 mmol/day of sodium during diuretic treatment.[12]

Another therapeutic option mentioned is the use of non-selective beta blockers (NSBBs), which can be administered unless there is severe hypotension, hyponatraemia, or renal failure. The use of non-selective beta-blockers in patients with refractory ascites remains controversial. On one hand, NSBBs are effective in reducing portal pressure and preventing variceal bleeding, and are reported to have additional protective effects by reducing intestinal permeability and inflammation in decompensated cirrhosis. However, reanalysis of several clinical trials shows that one-year mortality is not significantly different between NSBB users and non-users, and discontinuation of this medication during follow-up is associated with an increased risk of death, hospitalization, bleeding, infection, and hepatorenal syndrome.[8] Conversely, the use of NSBBs in conditions such as spontaneous bacterial peritonitis, severe alcoholic hepatitis, or refractory ascites can cause systemic hypotension and reduced cardiac reserve, thereby decreasing survival and increasing post-parase circulatory complications. Research also shows that propranolol doses below a certain threshold reduce mortality, while high doses increase the risk of death.[8,9]

The concept of the ‘therapeutic window’ suggests that NSBBs are beneficial only in selected cirrhosis patients without refractory ascites, hypotension, infection, or renal failure. Therefore, international guidelines recommend cautious use of NSBBs in refractory ascites, with immediate discontinuation if haemodynamic or renal dysfunction occurs, and avoidance of carvedilol and high-dose propranolol.[8,9]

Trans-jugular intra-hepatic portosystemic shunt

Another treatment for intractable ascites is trans-jugular intra-hepatic portosystemic shunt (TIPS). The mechanism of action of TIPS is to create an artificial pathway between the portal vein and the hepatic vein, thereby drastically reducing portal pressure. This reduction in portal hypertension increases cardiac output and renal perfusion, as well as stimulating natriuresis, thereby helping to remove excess ascites fluid. The primary goal of TIPS is to reduce portal pressure and decrease complications associated with ascites in cirrhosis.[8]

Data from various clinical trials and meta-analyses indicate that TIPS effectively controls ascites fluid accumulation, with approximately half of patients achieving remission without an increased risk of hepatic encephalopathy. In addition to ascites control, recent evidence also indicates that TIPS can improve survival, particularly in patients with preserved liver and renal function and when the procedure is performed earlier in the recurrent phase of ascites rather than waiting until the refractory phase.[9,13]

Timing and patient selection for TIPS are critical. Traditionally, TIPS has been recommended for refractory ascites unresponsive to maximal medical therapy; however, recent studies suggest earlier consideration in patients with recurrent ascites requiring multiple large-volume paracenteses. Key indicators include liver function status, renal condition, and history of hepatic encephalopathy. To minimize post-TIPS risks such as encephalopathy and cardiac dysfunction, several

predictive models are used to evaluate procedure survival and tolerability.[13]

In addition to portal pressure reduction, cardiac and renal function are also critical determinants of TIPS success. Sudden increases in cardiac preload can trigger cardiac decompensation in patients with latent heart failure; therefore, echocardiographic screening and assessment of biomarkers such as BNP/NT-proBNP are recommended prior to the procedure. The use of initial stent-grafts with a diameter <8 mm allows for gradual expansion based on clinical response, thereby reducing post-TIPS haemodynamic burden.[13]

On the renal side, while TIPS generally improves renal perfusion and has shown improvement in some patients with hepatorenal syndrome, the risk of contraindications remains high in severe renal dysfunction. Demographic and nutritional factors—including advanced age and sarcopenia—also influence post-TIPS tolerance and prognosis. Therefore, the decision to perform TIPS should be made on an individual basis, considering all these predictive factors, while continuing to evaluate liver transplantation as a definitive therapeutic option.[13]

Alfapump

The automated low-flow ascites pump system (alfapump®) is a therapeutic alternative to TIPS and liver transplantation in patients with refractory ascites. The device was developed by Sequana Medical (Ghent, Belgium) and received CE certification in July 2011. Alfapump® consists of a battery-powered pump implanted subcutaneously in the abdominal wall, connected to two catheters: one to the peritoneal cavity to drain ascites fluid, and another to the bladder so that the fluid can be excreted through urine. An automatic pressure sensor adjusts the pump cycle to stop when peritoneal pressure is too low or bladder pressure is too high.[14]

In an observational cohort study involving 106 patients with cirrhosis and refractory ascites who did not meet TIPS criteria, the implantation of the alfapump® significantly reduced the frequency of large-volume therapeutic paracentesis without improving survival compared to the pre-implantation period.[15] Furthermore, in the main POSEIDON trial with 81 subjects, the alfapump® nearly eliminated the need for therapeutic paracentesis within 6 months post-implantation and reduced the ascites symptom score (Ascites Q) from an average of 51.0 to 32.[2] ($p<0.001$). Physical components of the SF-36 improved significantly, while mental components did not change meaningfully. There were 24 cases of stage 1 acute kidney injury (AKI), all of which were reversible, and 5 deaths not directly related to the device.[16]

Although effective in controlling fluid accumulation and improving quality of life, the alfapump® is not without important side effects. Infection complications—including spontaneous bacterial peritonitis and urinary tract infections—were fairly common, though routine antibiotic prophylaxis could reduce them. Some patients also experienced impaired kidney function or kidney failure, suspected to be a form of paracentesis-induced circulatory dysfunction due to continuous fluid removal without albumin. Periodic albumin administration has been proposed, but the optimal dose and timing require further clinical validation. To date, there is no evidence that the alfapump® improves survival, so routine use of this device cannot yet be considered a standard therapy for refractory ascites.[7]

CONCLUSION

Intractable ascites or RA represents worsening disease in cirrhosis when ascites no longer responds to diuretic treatment. Even in this stage, maintaining sodium restriction is still an essential part of management. LVP remains the primary therapy, although precautions are needed to prevent PPCD. The use of regular albumin infusions may offer clinical benefits, but evidence is still limited. In carefully selected patients, a TIPS can provide long-term control of ascites. For those who are not suitable candidates for TIPS, an alfapump system may be used to continuously and slowly drain ascites, which can reduce abdominal distension, improve appetite, and enhance mobility. However, this approach requires prolonged antibiotic prophylaxis to reduce infection risk. Because the presence of intractable ascites significantly worsens prognosis in cirrhotic patients, timely evaluation for liver transplantation is crucial. In individuals who also have CKD, a combined liver–kidney transplant should be considered.

ACKNOWLEDGEMENTS

Authors would like to thank the internal medicine department, Wangaya General Hospital, Denpasar, Bali Indonesia.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Biggins SW, Angeli P, Garcia-Tsao G, Ginès P, Ling SC, Nadim MK, et al. Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. Hepatology. 2021;74(2):1014-48.
2. Mustapha S. Cirrhotic ascites: A review of pathophysiology and management. Nigerian J Gastroenterol Hepatol. 2020;12(1):3.
3. Tandon P, Walling A, Patton H, Taddei T. AGA Clinical Practice Update on Palliative Care Management in Cirrhosis: Expert Review. Clin Gastroenterol Hepatol. 2021;19(4):646-56.e3.

4. Sakr MA, Abdelaaty Abdelkader N, Fouad Y, El Zahaby A, Okasha H, El Amin H, et al. Algorithmic Approach for Evaluation of a Patient with Ascites: Expert Opinion by Egyptian Club of Ascites (ECA). Afro-Egypt J Infect Endem Dis. 2022;12(2):186-93.
5. Aithal GP, Palaniyappan N, China L, Härmälä S, Macken L, Ryan JM, et al. Guidelines on the management of ascites in cirrhosis. Gut. 2021;70:9-29.
6. Cho J, Jensen TP, Reierson K, Mathews BK, Bhagra A, Franco-Sadud R, et al. Recommendations on the Use of Ultrasound Guidance for Adult Abdominal Paracentesis: A Position Statement of the Society of Hospital Medicine. J Hosp Med. 2019;14:E7.
7. Zaccherini G, Tufoni M, Iannone G, Caraceni P. Management of Ascites in Patients with Cirrhosis: An Update. J Clin Med. 2021;10(22):5226.
8. Gallo A, Dedionigi C, Civitelli C, Panzeri A, Corradi C, Squizzato A. Optimal Management of Cirrhotic Ascites: A Review for Internal Medicine Physicians. J Transl Int Med. 2020;8(4):220-36.
9. Gao Y, Liu X, Gao Y, Duan M, Hou B, Chen Y. Pharmacological Interventions for Cirrhotic Ascites: From Challenges to Emerging Therapeutic Horizons. Gut Liver. 2024;18(6):934-48.
10. Pocha C, Rios-Perez C. Ascites – the Old, the Current, and the Future Ways of Management. Curr Hepatol Rep. 2025;24(19).
11. Wong F. Management of refractory ascites. Clin Mol Hepatol. 2023;29(1):16-32.
12. Carrier P, Loustaud-Ratti V, Debette-Gratien M, Elkrief L. Ascites in cirrhotic patients: a comprehensive review. Exploration of Digestive Diseases. 2024;3(5):362-81.
13. Lan T, Chen M, Tang C, Deltenre P. Recent developments in the management of ascites in cirrhosis. United European Gastroenterol J. 2024;12(2):261-72.
14. Weil-Verhoeven D, Di Martino V, Stirnimann G, Cervoni JP, Nguyen-Khac E, Thévenot T. Alfapump® implantable device in management of refractory ascites: An update. World J Hepatol. 2022;14(7):1344-56.
15. Stirnimann G, Berg T, Spahr L, Zeuzem S, McPherson S, Lammert F, et al. Final safety and efficacy results from a 106 real-world patients registry with an ascites-mobilizing pump. Liver Int. 2022;42(10):2247-59.
16. Wong F, Vagas HE, Reddy KR, Pagadala MR, Pocha C, Sundaram V, et al. The Effects of Alfapump on Ascites Control and Quality of Life in Patients With Cirrhosis and Recurrent or Refractory Ascites. Am J Gastroenterol. 2025;120(10):2291-301.

Cite this article as: Lestari IDAMD, Suryana K. Management of intractable ascites in cirrhosis: review article. Int J Adv Med 2026;13:56-61.