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ASCITIES AFTER LIVER CIRRHOSIS DIAGNOSIS AND TREATMENT

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ABSTRACT

Ascites, the accumulation of fluid in the abdominal cavity, is a common complication in patients with cirrhosis. Approximately 50% of patients with compensated cirrhosis will develop ascites over a 10-year period, marking a significant milestone in the progression of end-stage liver disease. Survival rates decrease after the onset of ascites, with only 50% of patients surviving 2 to 5 years, depending on the underlying cause of cirrhosis, The management of ascites involves salt restriction and diuretic use, which are effective in about 90% of patients in reducing fluid accumulation and symptoms. In cases where ascites does not respond to these measures, additional interventions such as large-volume paracentesis may be necessary as a temporary solution or for symptomatic relief while awaiting liver transplantation, For patients with refractory ascites, the transjugular intrahepatic portosystemic shunt (TIPS) procedure can be considered. This involves creating a shunt between the portal vein and hepatic vein to reduce portal hypertension. TIPS can serve as a bridge to liver transplantation or provide long-term palliation, but careful monitoring for bacterial peritonitis is essential. Patients at high risk for bacterial peritonitis should receive antibiotic prophylaxis to prevent this serious infection. Prompt diagnostic paracentesis is necessary when spontaneous bacterial peritonitis is suspected, involving the removal and analysis of fluid from the abdominal cavity.

INTRODUCTION

Ascites, a common complication of liver cirrhosis, has a poor prognosis. It is important to first exclude non-cirrhotic causes of ascites, such as malignant tumors, tuberculosis, and pancreatitis. For mild to moderate ascites, appropriate measures include limiting sodium intake, administering diuretics like spironolactone, and gradually increasing their dosage. In cases of large amounts of ascites, drainage should be performed along with supplementation of colloidal solution and diuretics. Difficult-to-treat ascites may require repeated massive pumping or the use of intrahepatic portosystemic shunt (TIPS) through the jugular vein. TIPS can improve renal function, sodium metabolism, and the overall condition of patients, but its impact on survival is uncertain. It is important to pay special attention to potential complications associated with different treatment methods, as well as the complications and contraindications of discontinuing diuretics and TIPS. Liver transplantation should be considered for all ascites patients before renal function is impaired, as it can improve prognosis. This article provides a comprehensive review of the advancements in the diagnosis and treatment of ascites. Cirrhosis accounts for more than 75% of ascites cases, while the remaining 25% are caused by malignant tumors (10%), heart failure (3%), pancreatitis (1%), tuberculosis (2%), and other rare causes. Approximately half of patients with liver cirrhosis develop ascites within 10 years. The International Ascites Association and the American Society of Hepatology have both updated their guidelines for the diagnosis and treatment of ascites caused by cirrhosis. In recent years, there have been significant advancements in the management of ascites caused by cirrhosis. Refractory ascites, which does not respond to therapeutic paracentesis and diuretic treatment, currently lacks approved medical therapy. Management of these patients relies on procedures such as large-volume paracentesis and transjugular intrahepatic portosystemic shunt (TIPS).[1,2]

Etiology

The etiology of ascites formation remains poorly understood, although the prevailing theory suggests that portal hypertension, particularly sinusoidal hypertension, plays a central role in the pathophysiology. The elevation of portal pressure induces splanchnic vasodilation, primarily attributed to the augmented local production of

nitric oxide, resulting in a hyperdynamic circulation. Consequently, this leads to an increase in capillary pressure and permeability, accompanied by a decrease in effective arterial blood volume. To counterbalance this reduction, the body employs compensatory mechanisms such as an expansion in plasma volume and cardiac output. Additionally, the activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS) triggers sodium and water retention, thereby facilitating the development of ascites.[3]

Table 1: Aetiology according to the serum ascites albumin gradient.

<11g/l	Infection, Nephrotic syndrome, Malignancy, Pancreatitis
>11g/l	Cirrhosis, Budd-Chiari syndrome, Veno-occlusive disease Alcoholic hepatitis, Congestive heart failure

Classification of Ascites

Ascites can be classified into different grades based on severity, with Grade 1 being considered mild ascites that is usually detected sub-clinically by ultrasound and typically does not require pharmacological treatment. In this case, sodium intake restriction and regular follow-up to monitor the progression of ascites are usually sufficient. For Grade 2 ascites, which is classified as moderate, treatment typically involves the initiation of diuretics along with dietary modifications to reduce sodium intake. This step is crucial in managing the condition effectively and preventing further complications. In cases of Grade 3 ascites, known as symptomatic tense ascites, medical intervention is necessary. Regardless of the response to medical treatment, management usually involves large volume paracentesis (LVP) along with albumin infusion. If the volume of ascitic fluid removed does not exceed 5 liters, a synthetic plasma expander may be used instead of albumin. Total volume paracentesis with the administration of intravenous albumin is the preferred approach in such cases. Refractory ascites, which is a more severe form of the condition, requires a standard of care that includes LVP with simultaneous administration of intravenous albumin. This treatment regimen also involves diuretic therapy, salt restriction, and careful monitoring of the patient's condition. In some cases, TIPS placement may be considered for patients with rapid recurrence of ascites and preserved liver function, provided they meet specific criteria.[4]

Table 2: Classification of ascites Severity.

Grade 1 (mild)	Not clinically evident, diagnosed on ultrasound Proportionate sensible abdominal distension Noticeable tense distension of abdomen
Grade 2 (moderate)	Not infected or associated with HRS
Grade 3 (severe) Uncomplicated Refractory	Cannot be mobilized, early recurrence after LVP, not prevented satisfactorily with medical treatment (after 1 week)
Diuretic-resistant Diuretic-intractable	No response to intensive diuretic treatment Drug-induced adverse effects preclude diuretic treatment

Diagnoses

It is crucial to identify the underlying cause of ascites. Abdominal puncture is a procedure that is generally safe and can be performed in most cases, except for individuals with coagulation disorders and disseminated intravascular coagulation. The lower left quadrant of the abdominal wall, located approximately 2 fingers above the anterior iliac spine and 2 fingers wide from the midline, is thinner and serves as a larger reservoir for fluid accumulation.[5] Therefore, puncturing this area has a higher success rate. In cases where obesity makes it difficult to locate the puncture site, ultrasound examination can be utilized. The protein content of ascites can be a useful diagnostic tool, as it can help differentiate between exudate and transudate. Approximately 55% of ascites cases can be distinguished by testing the protein content, with levels above 25g/L indicating exudate and levels below 25g/L indicating transudate. Interestingly, the protein content of ascites in patients with heart failure may be lower than that of leaked fluid. In liver cirrhosis, around 15% of ascites cases have a protein content greater than 25g/L, while in malignant tumor patients, approximately 20% have a protein content less than 25g/L. The plasma to ascites protein ratio (SAAG) is a valuable tool in distinguishing between portal hypertension and inflammation/abdominal tumors. A SAAG ratio of less than 1.1 suggests portal hypertension, while a ratio greater than or equal to 1.1 indicates inflammation or abdominal tumors. This ratio has an impressive accuracy rate of up to 97%. Patients with cirrhosis and ascites who are being treated as outpatients should also be monitored for spontaneous bacterial peritonitis (SBP). It is important to examine the ascitic fluid under a microscope and perform blood cultures, as Gram-positive bacteria are often found in these cases. A diagnosis of SBP can be made if the neutrophil count in the ascitic fluid is greater than 0.25/109 /L, although Gram staining of the ascitic fluid is usually not meaningful. In cases where malignant tumors or

pancreatitis are suspected, it is recommended to test the ascitic fluid for cytology or amylase. This can provide valuable information for diagnosis. In addition to blood cultures, routine analysis of the ascitic fluid should include cell count with differential, albumin, bacterial culture.[6,7]

Table 3: Findings suggesting secondary peritonitis.

No decrease in ascitic fluid PMN counts 48 h after treatment initiation. Ascitic fluid culture result is not monomicrobial.
At least two of the following criteria present: Ascitic fluid protein >1 g/dl
Ascitic fluid glucose <50 mg/dl
Ascitic fluid lactate dehydrogenase >225 mU/ml

Simple ascites, also known as non-infected ascites or ascites without hepatorenal syndrome (HRS), can be categorized into three levels based on its severity. The first level is characterized by mild ascites, which can be detected through ultrasound examination. The second level involves moderate symmetrical swelling of the abdomen. Lastly, the third level is characterized by a significant accumulation of abdominal fluid, resulting in noticeable swelling in the tertiary system.[8]

Refractory ascites, as defined by the International Ascites Association, pertains to the condition where ascites are challenging to reverse or swiftly reappear post-treatment. This category can be further classified into two types: diuretic resistant ascites and refractory ascites, encompassing type 0 hepatorenal syndrome, diluted hyponatremia, and weight loss. Approximately 5% to 10% of ascites fall under the classification of refractory ascites. The diagnostic criteria for refractory ascites are characterized by specific parameters. These include a prolonged treatment duration, where intensified diuresis necessitates more than a week with insufficient therapeutic outcomes. Additionally, the average weight loss is less than 0.8kg every 4 days, coupled with urinary sodium excretion lower than sodium intake. Another criterion is the rapid reappearance of ascites within 4 weeks post-release, leading to secondary or tertiary ascites. Furthermore, major diuretic side effects such as hepatic encephalopathy, plasma creatinine levels exceeding 167mol/L, blood sodium levels below 125mmol/L, and blood potassium levels either below 3mmol/L or above 6mmol/L are indicative of refractory ascites. These stringent diagnostic criteria aid in the accurate identification and management of this challenging condition.[9,10]

Prognosis & Managment of Ascites

Liver cirrhosis can lead to ascites, which is associated with a poor prognosis and a 2-year mortality rate of 40%. The prognosis becomes even more unfavorable in cases of refractory ascites and spontaneous peritonitis, highlighting the severity of the condition and the challenges it poses in terms of patient outcomes. The primary goal in managing ascites in liver cirrhosis is to improve sodium balance and enhance blood circulation function. This involves a comprehensive approach that aims to address the underlying causes and complications of ascites, with a focus on optimizing patient health and well-being. It is crucial to avoid alcoholic cirrhosis, as liver function can significantly improve following the resolution of ascites. This underscores the importance of addressing lifestyle factors and comorbidities that may exacerbate liver cirrhosis and its associated complications.[11]

The first-line treatment for ascites in liver cirrhosis typically involves sodium restriction (88mmol/d [2000mg/d]) and the use of diuretics such as oral spironolactone and furosemide. These interventions are aimed at reducing fluid retention and managing the symptoms of ascites effectively. In cases where plasma sodium levels fall below 120-125mmol/L, fluid restriction may be necessary to prevent further complications. Patients with tense ascites may require therapeutic abdominal puncture, in addition to sodium restriction and oral diuretics, to alleviate symptoms and improve overall health. Patients who do not respond adequately to diuretic therapy may require multiple abdominal punctures, further sodium restriction, and ongoing consideration for liver transplantation. This highlights the importance of individualized treatment plans and close monitoring to optimize patient outcomes in cases of severe ascites in liver cirrhosis.[12,13]

Treatment of simple ascites

Ascites, a condition characterized by the accumulation of fluid in the abdominal cavity, does not typically require specialized treatment. However, it is important to closely monitor the condition and take steps to reduce sodium intake. One approach to managing ascites is through bed rest, as standing upright can activate the sodium retention system, which hinders renal perfusion and sodium excretion. Resting can enhance the patient's response to diuretics, although there is a lack of clinical trials demonstrating that bed rest significantly improves the effectiveness of medication treatment.[14] Another important aspect of managing ascites is sodium restriction. Patients without ascites do not need to restrict sodium intake preventively. However, patients with urinary sodium/potassium ratios greater than 1 or 24-hour urinary sodium levels exceeding 78mmol, and who have not experienced weight loss, may be resistant to diuretics and should limit their sodium intake. It is recommended to limit sodium salt in food to 5.2g per day (90mmol) and maintain this restriction over the long term. Some patients may require even stricter limitations

on sodium intake. By restricting sodium intake or increasing urinary sodium excretion, a negative sodium balance can be achieved, leading to a reduction in ascites in approximately 10% to 15% of patients. However, it is important to note that strict sodium restriction (22mmol/L) can have adverse effects, such as kidney damage caused by diuretics and dilutive hyponatremia. In a controlled study, a mild reduction in salt intake (120mmol/L) had the same effect as a low salt diet (50mmol/L) in patients with ascites. While a low salt diet does not significantly impact the overall survival rate of patients, it can improve the survival rate of patients with a history of gastrointestinal bleeding. In cases of diluted hyponatremia, treatment typically involves limiting water intake, although there is a lack of clinical trials supporting its effectiveness, and it may also lead to low blood volume. Salt-resistant corticosteroids, such as spironolactone, are commonly used to treat secondary aldosteronism, which is a major factor contributing to sodium retention in the distal convoluted tubules and collecting tubules of the nephron. Spironolactone and canrenoate are more effective than the loop diuretic furosemide in terms of promoting urinary sodium excretion. The recommended daily dosage of spironolactone is typically between 100-200mg.[15,16]

Salt Restrictions and Diuretics Treatment

The role of secondary aldosteronism in promoting sodium retention in the distal convoluted tubules and collecting tubules of the nephron is crucial. Spironolactone is the preferred medication for treating this condition due to its effectiveness in combating sodium retention.

Spironolactone, along with canrenoate, is more effective than the loop diuretic furosemide in increasing urinary sodium excretion. The recommended daily dosage of spironolactone typically ranges from 100 to 200 mg, but in cases of severe aldosteronism with intense sodium retention, the dosage may be increased up to 400 mg. It is important to be aware that one of the main side effects of spironolactone is the development of gynecomastia, or enlargement of male breasts, as well as breast tenderness in females. Patients may also experience hyperkalemia or non-hyperkalemic metabolic acidosis, especially those with compromised renal function, Other potassium-preserving diuretics, such as amiloride and aminopterin, also act on the distal tubules. The efficacy of amiloride, typically given at a dosage of 20 -60 mg per day, is considered lower than canylic acid, which is prescribed at doses ranging from 150 to 500 mg per day. If spironolactone is not effective, loop diuretics like furosemide are often used. The standard dosage of furosemide ranges from 20 to 40 mg per day, but may be increased to 160 mg per day for short-term use. However, caution is advised as furosemide can lead to adverse effects such as hypokalemia, metabolic hypochloremic alkalosis, hyponatremia, and hypovolemia, which could potentially result in renal failure. Assessment of diuretic response is crucial in the management of patients with fluid overload. Daily weight measurement serves as a reliable indicator, with weight loss targets varying based on the presence or absence of peripheral edema. For individuals without edema, a weight reduction exceeding 0.5kg per day is considered optimal, while those with edema should aim for a decrease of less than 1kg per day. The majority of patients without renal impairment exhibit positive responses to treatment with anti-mineralocorticoids and loop diuretics, complemented by sodium restriction. These therapeutic interventions have proven to be effective in promoting diuresis and alleviating fluid retention in such individuals. A promising development in the management of ascites is the introduction of aquaretics, which are selective V2 receptor antagonists. By inhibiting the action of antidiuretic hormone in the collecting tubules, these agents enhance urinary output and facilitate the clearance of free water. Aquaretics show potential benefits in addressing hyponatremia, although their use has been limited to clinical trials thus far. Prior to the widespread adoption of aquaretics in clinical practice, further investigation is warranted to determine optimal dosages and assess potential side effects. Research efforts focused on refining the utilization of these agents will be essential in enhancing their safety and efficacy profiles for the management of fluid overload conditions.[8,17,18]

Complications of diuretic treatment

Common complications that may arise during diuretic therapy include hyponatremia, renal injury, and hepatotoxicity. Encephalopathy and muscle cramps are also potential issues that can be reversed by discontinuing diuresis and correcting low blood volume. In cases of mild hepatic encephalopathy (type 1), diuretics can still be used while managing hepatic encephalopathy as usual. However, for severe hepatic encephalopathy, diuretics should be temporarily halted and their effects reevaluated. Muscle cramps in patients with ascites are often due to a decrease in effective blood volume, and for severe paralytic muscle cramps, diuretics should be reduced or discontinued. Effective treatments for these complications may include albumin, quinidine, quinine, and zinc sulfate. Special complications such as hypokalemia, hyperkalemia, metabolic acidosis, or gynecomastia caused by spironolactone should be monitored closely, For patients experiencing hypokalemia due to loop diuretics, if blood potassium levels drop below 3.5mmol/L, furosemide should be adjusted or discontinued. Hyperkalemia is more common in patients with refractory ascites, renal dysfunction, and those requiring high-dose diuretic therapy. In cases where blood potassium levels exceed 5.5mmol/L or 6mmol/L, spironolactone dosage should be reduced or stopped. Other symptoms to watch for include renal failure, elevated sodium clearance rates, and bacterial infections. Patients with liver or renal issues stemming from low blood volume should use diuretics cautiously. When blood sodium levels fall below 120mmol/L, diuretics should be temporarily

halted. However, there is no conclusive evidence supporting the complete avoidance of diuretics in patients with initial renal dysfunction like diabetic nephropathy. Type 2 hepatorenal syndrome typically shows a poor response to diuretic therapy.

Puncture aspiration is the most recommended approach for managing grade III ascites, and it is crucial to take into account diuresis and sodium limitation. It is considered safe to eliminate all the accumulated fluid at once, even if there is a substantial quantity of ascites. To avoid kidney complications, it is necessary for all patients, including those with peripheral edema, to increase their plasma volume. In situations where the extracted fluid is below 5L, artificial plasma substitutes can be employed. However, if the quantity surpasses 5L, it is advised to administer 8g of albumin for every liter of ascites.[19-22] Paracentesis Sodium restriction and diuretic therapy can effectively reduce ascites in approximately 90% of patients. However, if ascites persists despite maximum diuretic treatment or if severe side effects like renal impairment occur, it is classified as refractory. For patients with refractory ascites, there are various therapeutic options available, with the least invasive option being large volume paracentesis (LVP), which involves removing more than 5 liters of ascitic fluid. LVP is a safe and effective outpatient procedure for relieving symptoms associated with ascites, but it does not address the underlying mechanisms causing ascites formation. One potential complication of LVP is paracentesis-induced circulatory dysfunction (PICD), which is characterized by a significant increase in plasma renin concentration compared to baseline levels by day 6 after the procedure.

The development of PICD is attributed to a decrease in systemic vascular resistance (SVR), primarily caused by an exacerbation of existing arterial vasodilation. The exact mechanism by which paracentesis leads to additional arterial vasodilation is still unclear, but PICD triggers compensatory activation of the renin-angiotensin-aldosterone system (RAAS).[23,25]

Treatment of refractory ascites

The initial approach to managing puncture and drainage involves removing fluid repeatedly. To reduce the need for multiple drainage procedures, patients are advised to take diuretics for an extended period. If complications arise or urinary sodium levels drop below 20mmol/d, diuretic therapy should be stopped. Patients who struggle with frequent punctures and fluid extractions may find relief by considering the use of Transjugular Intrahepatic Portosystemic Shunt (TIPS). Puncture procedures provide a quick way to remove a significant amount of ascites that may not respond well to medication. The repeated extraction of large amounts of ascites (5L/d) along with albumin infusion (6-8g of protein per 1L of ascites removed) has been proven to effectively reduce ascites, shorten hospital stays, and decrease the risk of complications. Both a single withdrawal of 5 L and multiple smaller withdrawals of 5 L are equally safe and effective. However, fluid aspiration can lead to a sudden increase in cardiac output and a decrease in systemic venous volume, potentially causing a drop in blood pressure. After a puncture procedure, there is a rapid decrease in right atrial pressure and pulmonary capillary wedge pressure (PCWC) within 6 hours, resulting in reduced intrathoracic pressure. This can lead to a decrease in effective blood volume (PPH) and circulatory dysfunction hours or days after the procedure. It is important to promptly increase blood volume post-puncture, with albumin supplementation proving more beneficial than salt in this regard. The medical community is currently discussing the best approach for expanding capacity after fluid aspiration, as complications are common in around 30% of patients. Surprisingly, only 16% of patients receive albumin infusion as a treatment. These complications can range from kidney damage to hyponatremia, elevated plasma renin levels, and aldosteronism. It is essential to expand volume after fluid extraction to prevent hypovolemia, hyponatremia, and kidney injury. Crystals are typically the first-line medication used, with albumin considered a secondary option. Intravenous albumin infusion is not required for patients with less than 4L of fluid, but the cost and potential risks of human serum albumin should be considered. The American Society of Hepatology has denied any association with non-specific viral infections or virus-related diseases. Alternative plasma extenders, such as dextran, hydroxyethyl starch, collagen-based colloids, 706th generation plasma, and mixed plasma extenders, have been found to be equally effective in preventing complications from fluid extraction. However, there are differing opinions on the effectiveness of albumin compared to mixed plasma vasodilators in preventing conditions such as PPH, increased plasma renin activity, or aldosteronism. When considering the contraindications and complications of fluid extraction, it is important to keep a few factors in mind. Relative contraindications include systolic blood pressure (SBP), renal failure, and severe hepatic coma, as these conditions can pose risks or complications during the fluid extraction process. It is crucial for medical professionals to thoroughly evaluate and assess the patient's overall health and medical history before proceeding with fluid aspiration.[26,27]

Transjugular Intrahepatic Portal

The utilization of Transjugular Intrahepatic Portal Cava Shunt Surgery (TIPS) remains a viable option for treatment in cases where the frequency of fluid draws surpasses three times per month, there are contraindications for fluid removal, presence of abdominal adhesions, encapsulated ascites, and recurrent large volumes of hepatic pleural effusion. TIPS serves as an alternative therapeutic approach for refractory ascites, particularly in situations where patients have a high demand for, or exhibit poor tolerance to, Large Volume Paracentesis (LVP). The primary mechanism of action of TIPS involves the reduction of portal pressure, which in

turn decreases the likelihood of ascites development in individuals with cirrhosis when the pressure falls below 12 mmHg. Following the placement of TIPS, there is an observed increase in urinary sodium excretion within a period of 7 to 30 days post-stent insertion. This phenomenon is associated with a decline in the activity of the Renin-Angiotensin-Aldosterone System (RAAS) and enhancement of effective arterial blood volume. The implementation of TIPS as a treatment strategy offers a promising avenue for managing complex cases of ascites, especially in scenarios where conventional methods may not be as effective or well-tolerated by patients. By addressing the underlying pathophysiology through the modulation of portal pressure and subsequent physiological responses, TIPS presents a valuable option in the therapeutic armamentarium for individuals with challenging ascites-related conditions.[28,29]

Complications and contraindications of TIPS

Hepatic encephalopathy is linked to 30% of cases involving Transjugular Intrahepatic Portosystemic Shunt (TIPS). Following surgery, there is a high incidence of shunt tube stenosis or blockage, reaching about 70%, although the occurrence of such issues in polytetrachloroethylene TIPS shunt tubes may be lower. Noteworthy complications include cardiovascular disease and hemolytic anemia, with TIPS-related mortality rates potentially rising in patients classified as ChildC grade. The primary contraindications for TIPS procedures encompass hepatic encephalopathy, individuals aged over 70, heart failure, and a Child-Pugh score surpassing. Cardiac ejection fraction serves as a valuable indicator for TIPS suitability, with surgery being a viable option if the ejection fraction exceeds 60%. Moreover, it is worth noting that 50% to 60% of individuals undergoing TIPS may be predisposed to heart failure. Managing diluted hyponatremia poses challenges, particularly in cases of liver disease where it is compounded by hematogenous hyponatremia. This form of hyponatremia, which is ineffective in diluting blood and can lead to an increase in blood volume, significantly reduces brain tissue osmotic pressure. Furthermore, there may be a correlation between diluted hyponatremia and the development of encephalopathy. Close monitoring and necessary precautions are essential in patients with progressive liver cirrhosis and ascites to prevent bacterial translocation, which can lead to serious abdominal infections. Abdominal venous bypass surgery is a last resort option for patients who are not suitable candidates for liver transplantation, TIPS, or repeated heavy fluid extraction. This surgical procedure is typically reserved for cases where other treatment options have been exhausted and has limited effectiveness. Liver transplant patients with difficult-to-treat ascites have a grim prognosis if routine internal medicine treatments fail. Without effective management, a significant percentage of these patients may not survive beyond 6 months or 1 year. In such cases, liver transplantation becomes a critical consideration for improving survival rates and overall outcomes, with a relatively high one-year survival rate of 85%. The decision on liver transplantation candidacy is complex and depends on various factors, including the patient's willingness to undergo the procedure and regional considerations such as donor availability and waiting times. Patients with refractory ascites should be evaluated for liver transplantation as it may offer the best chance for long-term survival and improved quality of life. All patients with ascites should be considered as potential candidates for liver transplantation to optimize their outcomes.[30,33]

liver transplantation

Administering cefotaxim intravenously at a dosage of 2 g every 12 hours has been found to be effective in managing ascites in patients with cirrhosis. The development of ascites in these patients is often indicative of a significant decline in survival rates. Therefore, it is crucial to promptly evaluate the need for liver transplantation in individuals with ascites, ideally before complications such as spontaneous bacterial peritonitis (SBP) and hepatorenal syndrome (HRS) arise. Early consideration of liver transplantation can potentially improve patient outcomes and increase the chances of a successful transplant procedure.[34]

Prevention of Ascites

Proper management of ascites is essential for improving the overall outlook for individuals with liver cirrhosis. Treatment typically involves addressing the root cause, following a low-sodium diet, and using diuretics, with medications like spironolactone and furosemide being commonly prescribed in the US and Europe. Spironolactone is generally preferred over furosemide in ascites treatment protocols. When ascites becomes difficult to treat, patients may consider Transjugular Intrahepatic Portosystemic Shunt (TIPS) therapy, which has specific criteria and a higher risk of hepatic encephalopathy. Researchers have been working on predictive models, such as the factor index model, to predict survival rates for cirrhotic patients with refractory ascites undergoing TIPS therapy. These models have identified serum bilirubin and creatinine levels as important prognostic indicators, but further research is needed to validate these findings due to limited sample sizes and lack of robust validation.

Table 4: Cirrhotic patients eligible for spontaneous bacterial peritonitis (SBP) prophylaxis.

Short Term Prophylaxis	Long Term Prophylaxis
Norfloxacin 400 mg twice daily for 7 days Patients with gastrointestinal bleeding	Norfloxacin 400 mg daily Patients recovered from episode of SBP Patients with ascites and low ascitic fluid protein count (<10g/l) (no consensus)

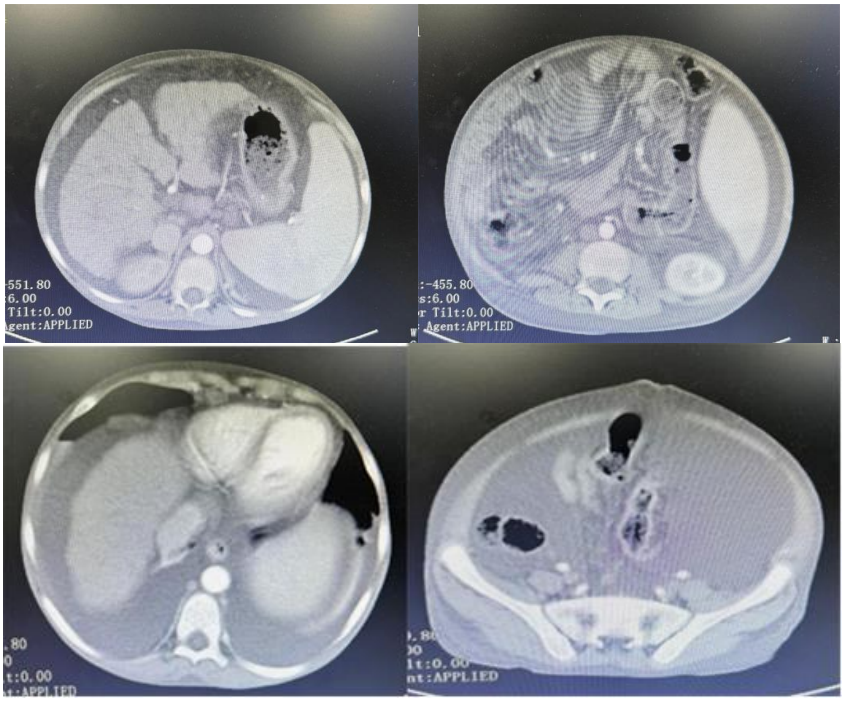

Figure 1: CT of Patient with Ascities.

SUMMARY

Cirrhosis is often accompanied by ascites, which is considered one of the most common complications associated with this condition. Approximately half of patients with compensated cirrhosis will develop ascites, which is a significant indicator of poor long-term survival outcomes. The treatment approach for uncomplicated ascites typically involves a combination of sodium and fluid restriction, along with a carefully managed diuretic regimen. It is important to note that overly strict sodium restriction is not recommended, as it may result in patient noncompliance and other adverse effects. The preferred initial therapy for managing ascites involves the use of aldosterone antagonists in conjunction with a loop diuretic. For patients with more complex or severe cases of ascites, such as those with complications, Transjugular Intrahepatic Portosystemic Shunt (TIPS) may be considered. In some instances, individuals with advanced ascites due to end-stage liver disease may ultimately require a liver transplant for optimal management of their condition.

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