A Case Report of Untreatable Complication of Umbilical Hernia in Cirrhosis: A Flood Syndrome

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ABSTRACT

Flood syndrome is a rare, life-threatening complication of end-stage liver disease, characterized by the spontaneous rupture of an umbilical hernia. It frequently presents with sepsis, peritonitis, and bowel incarceration, carrying a high mortality rate. The lack of standardized treatment guidelines and the complexities of advanced liver disease make management particularly challenging. We report a case of a 53-year-old woman with decompensated hepatitis B-related cirrhosis (Child-Pugh Class C, MELD score of 26) and refractory ascites, who developed Flood syndrome. She presented with a ruptured umbilical hernia leaking ascitic fluid, accompanied by sepsis, diabetic ketoacidosis, acute kidney injury, and hypoalbuminemia. Surgical intervention was deemed high-risk, and conservative management was undertaken, focusing on infection control, wound care, and medical stabilization of her comorbidities. Despite a multidisciplinary approach, the patient succumbed to the cumulative effects of her advanced disease and associated complications. This case highlights the complexities of managing Flood syndrome, particularly in patients with advanced liver disease. The absence of standardized treatment protocols necessitates a multidisciplinary, individualized approach. Key considerations include managing ascites, preventing infection, optimizing glycaemic control, and carefully evaluating the risks of surgical intervention. This case report illustrates the clinical course, management challenges, and outcomes of Flood syndrome in a patient with advanced cirrhosis, in order to highlight key considerations for clinicians and underscore the urgent need for further research to establish evidence-based guidelines for managing this rare but life-threatening condition.

Keywords: *Cirrhosis, flood syndrome, umbilical hernia*

ABSTRAK

Sindrom Flood merupakan komplikasi langka yang mengancam jiwa dari penyakit hati stadium akhir, yang ditandai dengan rupturnya hernia umbilikalis secara spontan. Kondisi ini seringkali disertai dengan sepsis, peritonitis, dan inkarserasi usus, yang menyebabkan mortalitas yang tinggi. Kurangnya pedoman pengobatan yang terstandar dan kompleksitas penyakit hati stadium lanjut mengakibatkan penanganannya menjadi sangat menantang. Kami melaporkan kasus seorang wanita berusia 53 tahun dengan sirosis terkait hepatitis B yang tidak terkompensasi (Child-Pugh Kelas C, skor MELD 26) dan asites refrakter, yang mengalami sindrom Flood. Pasien datang dengan hernia umbilikalis yang rupture dan mengeluarkan cairan asites, disertai dengan sepsis dan ketoasidosis diabetikum. Kondisinya semakin rumit karena syok, cedera ginjal akut, dan hipoalbuminemia. Intervensi bedah dianggap berisiko tinggi, dan penanganan konservatif dilakukan, dengan fokus pada pengendalian infeksi, perawatan luka, dan stabilisasi dari penyakit penyerta. Meskipun telah dilakukan pendekatan multidisiplin, pasien tersebut meninggal karena efek kumulatif dari penyakitnya yang sudah stadium lanjut dan komplikasi terkait. Kasus ini menunjukkan kompleksitas penanganan sindrom Flood, khususnya pada

pasien dengan penyakit hati stadium lanjut. Tidak adanya protokol pengobatan yang terstandarisasi memerlukan pendekatan multidisiplin dan individual. Pertimbangan utama meliputi penanganan asites, pencegahan infeksi, pengoptimalan kontrol glikemik, dan evaluasi risiko intervensi bedah secara cermat. Laporan kasus ini menggambarkan perjalanan klinis, tantangan penatalaksanaan, serta prognosis dari sindrom Flood pada sirosis lanjut, untuk memberikan wawasan peting bagi klinisi dan menekankan pentingnya penelitian lebih lanjut untuk pengembangan pedoman berbasis bukti untuk kondisi yang langka namun berpotensi mengancam jiwa ini.

Kata kunci: *Sirosis, sindrom flood, heria umbilikalis*

INTRODUCTION

Ascites is common in the progression of liver cirrhosis, which occurs in more than 50% of cases, and has been associated to higher rates of morbidity and mortality in patients with liver cirrhosis. Umbilical hernia develops as a result of increased intrabdominal pressure from ascites, pushing the peritoneum through the umbilical ring and into the subcutaneous space[1.] About 20% of cirrhotic patients with ascites develop umbilical hernias. These patients are more likely to develop an umbilical hernia that quickly grows and manifests symptoms.[2] Contrary to the general population, where obesity and female are risk factors for umbilical hernias, cirrhotic patients are more likely men with ascites.[3] Skin attenuation caused by the ascites-filled subcutaneous peritoneal sac that is in close proximity to the skin can result in ulceration and even rupture. Rarely, spontaneous rupture of the umbilical hernia may occur in these patients.[4]

Flood syndrome is the complication of refractory ascites and end-stage liver cirrhosis where ascitic fluid erupts from the opening caused by a spontaneous rupture of an umbilical hernia. Despite the fact that 20% of patients with end-stage liver cirrhosis exacerbated by ascites experience umbilical hernias, it is still a very uncommon complication of the disease.[2] Although the exact cause of spontaneous umbilical hernia rupture in cirrhotic patients is unknown, it is thought that a combination of fascial and abdominal muscle weakness brought on by malnutrition, increased abdominal pressure from ascites, and expansion of pre-existing fascial weaknesses are significant factors to the development of the hernias.[5]

It is debatable on how to manage cirrhotic patients who have umbilical hernias. Expectant care was used traditionally due to complication risks, however it can result in serious issues, such as peritonitis, ascites drainage, hernia incarceration and necrosis, and skin perforation.[2] Studies have demonstrated that surgical outcomes depend on presence of ascites and grade of liver function; elective herniorrhaphy is safe and effective in adequately controlled ascites and should be avoided in uncontrolled ascites.[6] Conservative management is associated with higher morbidity and mortality, therefore, elective hernia repair is preferred. However, patients with Flood syndrome are poor surgical candidates due to high mortality in decompensated cirrhosis. Hence, there is no gold standard for treatment of Flood syndrome, and most approaches are based on case studies.[5]As a result, we present a rare case of Flood syndrome with high mortality and morbidity due to challenges in medical and surgical treatment. This case report illustrates the clinical course, management challenges, and outcomes of Flood syndrome in a patient with advanced cirrhosis, in order to highlight key considerations for clinicians and underscore the urgent need for further research to establish evidence-based guidelines for managing this rare but life-threatening condition.

CASE ILLUSTRATION

A 53-year-old female with decompensated hepatitis B-related cirrhosis (Child-Pugh Class C, Model for End-Stage Liver Disease (MELD) score 26), presented to the emergency department with a one-day history of loss of consciousness, persistent vomiting, and severe abdominal pain localized to a previously known umbilical hernia. The hernia had recently ruptured without any trauma or precipitating factors, with accompanying leakage of serous fluid.

Over the preceding months, the patient's health had progressively worsened, with frequent hospitalizations for the management of refractory ascites and poorly controlled diabetes. She had undergone paracentesis every two to four weeks, with the most recent procedure occurring two weeks prior to her admission, during which 2 liters of ascitic fluid were drained, showing characteristics of a transudate. Additionally, the patient had a history of an 8 cm-diameter umbilical hernia, for which she had undergone herniorrhaphy along with the aspiration of 3500 mL of ascitic fluid a year prior. Routine medications include omeprazole 20 mg once a day, propranolol 20 mg thrice a day, spironolactone 100 mg thrice a day, furosemide 40 mg thrice a day,

glargine 30 unit once a day, aspart 15 unit thrice a day, and folic acid 1 mg once a day. Despite ongoing medical care, her quality of life had been severely impacted, with physical limitations, dependence on her spouse for daily activities, and emotional distress.

On physical examination, the patient was lethargic, with a Glasgow Coma Scale (GCS) score of 9/15 (E2M5V2). Her blood pressure was 90/50 mmHg, heart rate 120 bpm, respiratory rate 30 breaths per minute, and oxygen saturation 95% via room air. She was febrile with a temperature of 38.9°C and exhibited cold extremities. Abdominal examination revealed a massive, erythematous umbilical hernia measuring 10x12 cm with ulceration and leakage of straw-colored fluid **(Figure 1).** The abdomen was distended and tender on palpation.

Figure 1. Abdomen showing distention, erythematous umbilical hernia (8 x 10 cm) with non-bleeding ulceration of the overlying skin and straw-colored ascitic fluid draining.

Laboratory results revealed haemoglobin of 10.5 g/dL, platelet count of 85 $\times 10^3$/L, and white blood cell count of 15.5 $\times 10^3$/L. With a serum urea level of 110 mg/dL and creatinine level of 1.79 mg/dL, she may have had an acute kidney injury, most likely due to hepatorenal syndrome. In addition, she developed hyponatremia (Na 127 mmol/L), hyperkalaemia (7.0 mmol/L), and hypoalbuminemia (2.78 g/dL). Her random blood glucose was extremely elevated at 419 mg/dL with blood ketone level of 0.7 mmol/L and HbA1C of 9.2%, indicating inadequate management of her diabetes resulting in diabetic ketoacidosis (DKA). Blood gas analysis was done showing pH 7.360, pO2 124 mmHg, pCO2 27.9 mmHg, and HCO3 15.4 mmol/L, indicating compensated metabolic acidosis. The electrolyte blood gas revealed a sodium level of 141 mmol/L, potassium level of 4.30 mmol/L, and chloride level of 106 mmol/L. The calculated anion gap is 19.6 mEq/L, which further supporting the diagnosis of metabolic acidosis, likely due to DKA.

Diagnostic paracentesis was performed upon admission. Ascitic fluid analysis revealed a serum-ascites albumin gradient (SAAG) of 2.17 g/dL, consistent with portal hypertension. The total protein content of ascitic fluid was low at 1.15 g/dL, supporting a transudative process typically seen in cirrhosis-related ascites. The total white blood cell count was 97 cells/µL, with 12% polymorphonuclear neutrophils (PMNs), yielding an absolute PMN count of approximately 12 cells/µL—below the diagnostic threshold of 250 cells/µL for spontaneous bacterial peritonitis (SBP). Additionally, the ascitic fluid glucose was elevated at 172 mg/dL and lactate dehydrogenase (LDH) was low at 55 U/L, making secondary peritonitis unlikely. Overall, the ascitic fluid findings were consistent with uncomplicated portal hypertensive ascites, with no evidence of SBP or secondary peritonitis.

Initial abdominal ultrasonography **(Figure 2)** performed a year ago showed umbilical hernia filled with fluid sized +/- 8.6 cm x 2.49 cm. Colour Doppler flow imaging (CDFI) did not show any increased intra-lesion vascularization, suggesting no active blood flow or inflammation within the hernia.

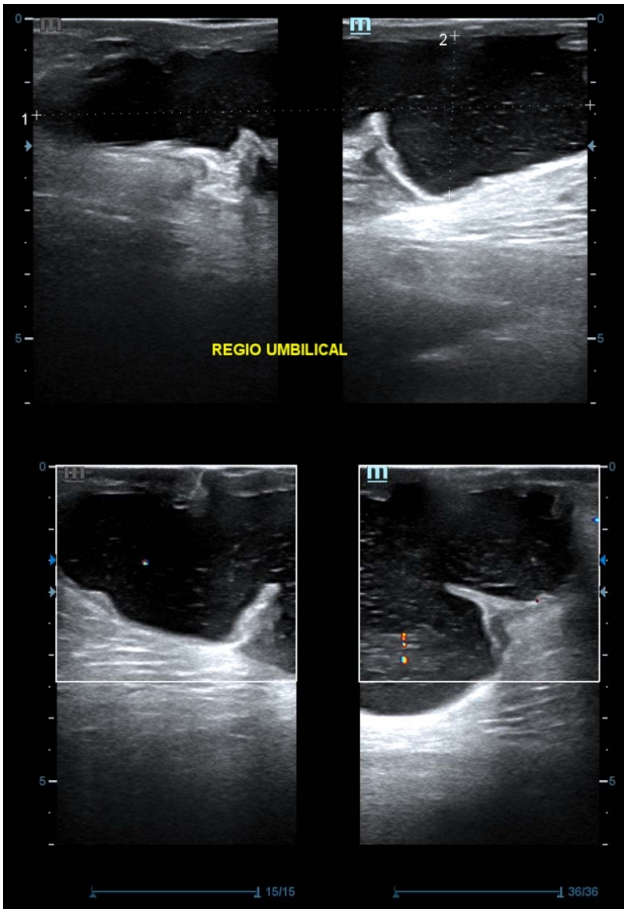

Figure 2. Abdominal ultrasonography showed initial umbilical hernia filled with fluid sized +/- 8.6 cm x 2.49 cm with color Doppler flow imaging (CDFI) does not show increased intra-lesion vascularization

Abdominal ultrasonography performed upon admission showed recurrent umbilical hernia containing intestines at the abdominal superior to the umbilical region **(Figure 3).** This recurrence was associated with the previously noted hernia and indicated a more complicated presentation. Further ultrasound findings (**Figure 4**) showed heterogenous rough parenchymal echogenicity with blunt liver tip, suggesting cirrhotic changes. Additionally, free fluid was observed in the peri-hepatic, peri-splenic, and peri-vesical regions, consistent with massive ascites.

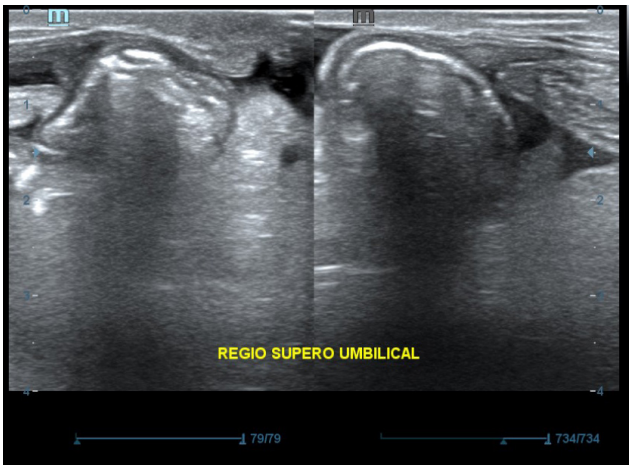

Figure 3. Abdominal ultrasonography showed recurrent umbilical hernia containing intestines at the abdominal superior to the umbilical region.

Given the patient's severe and complex presentation, a comprehensive treatment plan was initiated. On Day 1 of admission, empiric antibiotic therapy with intravenous cefotaxime 2 g every eight hours was initiated to treat spontaneous bacterial peritonitis (SBP), a common and potentially life-threatening complication in cirrhosis. The wound site was cleaned and covered with sterile dressing, and topical metronidazole powder was applied to prevent local infection. Intravenous albumin infusions were given to improve intravascular volume and reduce third-spacing of fluids. Diuretics were withheld temporarily due to hypotension. Her insulin regimen was adjusted with rapid-acting insulin (aspart) 15 unit thrice daily and long-acting insulin (glargine) 45 unit once daily. Blood glucose monitoring was intensified. Sodium bicarbonate 500 mg thrice daily was prescribed to correct metabolic acidosis. Folic acid 1 mg once daily is given as patients with chronic liver disease are at risk of folate deficiency, preventing anaemia. Lactulose 15 mL once daily was given to reduce lower blood ammonia levels, which prevent hepatic encephalopathy. Although an upper gastrointestinal endoscopy was not performed and the presence of oesophageal varices could not be confirmed, propranolol 10 mg thrice daily was continued as empiric primary prophylaxis for and

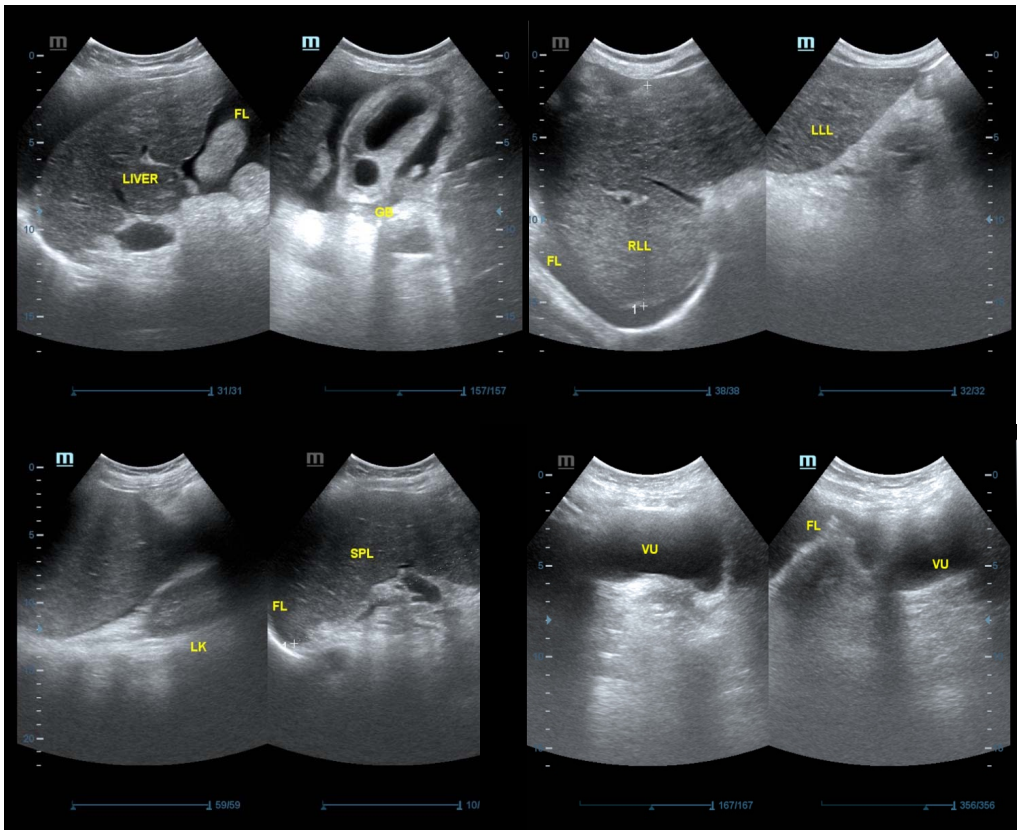

Figure 4. Abdominal ultrasonography showed heterogenous rough parenchymal echogenicity with blunt liver tip. Free fluid was observed in the peri-hepatic, peri-splenic, and peri-vesica region, indicating massive ascites

variceal haemorrhage, supported by clinical evidence of portal hypertension including a SAAG of 2.17 g/dL, decompensated cirrhosis, and massive ascites.

Over the next two days, the patient's condition was closely monitored. Insulin doses were adjusted based on frequent blood glucose testing. Spironolactone and furosemide were reintroduced cautiously after hemodynamic stabilization to manage refractory ascites. Despite aggressive medical management, the patient's clinical status began to deteriorate. Her abdominal distention worsened, and she developed further signs of systemic infection, including worsening sepsis. Given her advanced liver disease, poor functional status, and high MELD score, surgical intervention was deemed high-risk. Conservative measures, including wound care and close monitoring, were advised. Counselling sessions were offered to address anxiety and depression, and a palliative care consultation was arranged to discuss goals of care and end-of-life preferences. Despite the intensive efforts to stabilize her, the patient's health continue to decline, and she passed away on Day 4, surrounded by her family members. Her death marked the culmination of a long and challenging journey with end-stage liver disease, complicated by DKA, sepsis, and multiple organ dysfunction.

DISCUSSION

The case of this patient highlights the intricate challenges posed by Flood syndrome, a rare and life-threatening complication of end-stage liver disease with ascites. Umbilical hernias account for 6-14% of adult abdominal wall hernias.[6] Common risk factors include obesity, nulliparity, and female gender. However, in patients with liver cirrhosis, male gender and ascites are significant contributors to umbilical hernia development, affecting approximately 20% of this population.[3] Flood syndrome, characterized by the spontaneous rupture of an umbilical hernia, is associated with a high mortality rate of 30%.[7] It arises due to increased intra-abdominal pressure causing rapid hernia enlargement and rupture, which can lead to sepsis, peritonitis, and bowel incarceration. Skin excoriation or ulceration often precedes the rupture, serving as a critical warning sign.[8] The patient's prognosis was further worsened by cirrhosis and hypotension, complicating effective management.

Management of Flood syndrome generally involves dietary salt and fluid restriction, diuretics, regular paracentesis, and avoidance of alcohol and non-steroidal anti-inflammatory drugs.[9] However, therapeutic paracentesis, particularly large-volume drainage, can exacerbate hernia formation due to sudden shifts in intra-abdominal pressure.[3] Refractory ascites, unresponsive to standard diuretic therapy, necessitates frequent paracentesis and increases the likelihood of hernia recurrence and rupture. The patient's shock condition added another layer of complexity, requiring a delicate balance between diuresis and hemodynamic stability. The rupture of her hernia and the subsequent sepsis, exacerbated by her immunocompromised state, necessitated cautious fluid management to prevent worsening of ascites.

Due to the rarity of Flood syndrome, there are no standardized treatment protocols, and management decisions often need to be individualized. In emergencies, the primary objectives are hemodynamic stabilization and infection control. Initial measures include wound cleansing, sterile dressing, and broad-spectrum antibiotics targeting skin flora and bacteria associated with peritonitis. While friable umbilical tissue may limit the effectiveness of sutures, they can help prevent fluid loss and wound expansion in some cases. If fluid leakage persists, a temporary use of an ostomy bag may be considered.[10] SBP is a common and life-threatening complication in patients with cirrhosis, with an incidence ranging from 18% to 23%, particularly in those with decompensated liver disease and ascites.[3] The decision to use cephalosporin monotherapy with cefotaxime and refrain from combination therapy in this case was based on several clinical considerations. The first-line treatment for SBP typically includes third-generation cephalosporins, such as cefotaxime, provide broad-spectrum coverage against the most common pathogens causing SBP, particularly Gram-negative bacteria like *E. coli*, without the need for more potent, broader regimens. Combination therapy is typically reserved for cases where the patient is at high risk for multi-drug resistant (MDR) organisms. While the patient has risk factors for MDR organisms, particularly due to frequent hospitalizations and cirrhosis-related complications, there was no immediate indication in this case that the infection was caused by a resistant organisms. Additionally, using combination therapy could increase the risk of toxicity, drug interactions, and adverse effects, particularly in a cirrhotic patient with liver and renal dysfunction and hemodynamic instability. The decision also considered the potential complications of nephrotoxicity or hepatotoxicity from more aggressive antibiotics. Cefotaxime monotherapy

was chosen in this case to balance effective treatment of the infection while minimizing the potential harms of overusing antibiotics, especially in a patient with advanced cirrhosis.

Given the advanced liver disease, high MELD score, and the overall poor prognosis, the goal of care included more emphasis on supportive care and palliative management rather than aggressively pursuing broader-spectrum antibiotics. This approach focuses on alleviating symptoms and improving quality of life rather than pursuing aggressive treatments that may not change the patient's outcome. Spontaneous drainage of large-volume ascites requires intravenous fluid replacement to prevent hemodynamic instability.[4] Although pigtail catheter placement was considered, it was deemed unsuitable for long-term management due to infection risks.[9]

Elective herniorrhaphy is typically recommended for cirrhotic patients with well-controlled ascites and minimal comorbidities. However, in cases involving complex hernias or uncontrolled ascites, the risk of postoperative complications—including infections, ascitic fluid leakage, liver failure, and recurrence—significantly increases. Non-emergent repairs, conducted after optimal ascites control, are associated with lower mortality rates compared to emergency surgeries (2% versus 14%).[11] Laparoscopic herniorrhaphy, a minimally invasive approach, may offer advantages in cirrhotic patients with complex umbilical hernias.[12,13] Despite these potential benefits, spontaneous rupture of umbilical hernias in cirrhotic patients remains a rare occurrence and presents unique challenges in management due to the underlying liver disease. The Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores are crucial tools for assessing the degree of metabolic derangement and predicting the perioperative risks. For patients with low CTP and MELD scores, primary anatomic repair with double drain placement is considered a safe and effective strategy for managing Flood syndrome. However, in those with high CTP (Class C) and MELD scores exceeding 30, the perioperative risks must be carefully weighed against the potential benefits of surgery.[14] In this case, the patient's advanced liver disease, reflected in her high MELD score and poor functional status, made her a poor surgical candidate.[15-16] These factors underscore the complexity of surgical decisions in patients with advanced liver disease, highlighting the need for individualized management based on the patient's overall clinical condition.

The patient's poorly controlled diabetes and resultant diabetic ketoacidosis (DKA) further complicated her clinical course. Hyperglycaemia increases susceptibility to infections, impairs wound healing, and exacerbates inflammatory responses, all of which contributed to her frailty and heightened infection risk.[17] Additionally, the elevated anion gap observed in her blood gas analysis (19.6 mEq/L) indicated the presence of metabolic acidosis, likely secondary to DKA. The elevated anion gap is consistent with the accumulation of ketoacids, which is a hallmark of DKA and reflects the severity of her condition. Although lactate levels were not measured in this case, it is important to note that elevated lactate levels would have provided additional insight into whether lactic acidosis was also contributing to the metabolic acidosis. In the absence of lactate measurements, the elevated anion gap remains a strong indicator of DKA, as it is primarily driven by ketoacids. Adjustments to her insulin regimen were critical to achieve better glycaemic control, prevent further complications, and promote recovery. In parallel, acute kidney injury (AKI), likely secondary to hepatorenal syndrome, further worsened her prognosis. AKI, a common complication in advanced cirrhosis, not only disrupts electrolyte balance but also exacerbates liver dysfunction. The presence of AKI complicated her management, particularly because aggressive diuresis could not be done due to her hypotension and volume depletion. Both DKA and AKI played significant roles in the patient's overall clinical deterioration, and their combined effects underscored the complexity of her treatment. Addressing these underlying conditions was critical in attempting to stabilize her, but the severity of each condition ultimately led to her poor prognosis.

A previous case involved an 80-year-old with NASH-related cirrhosis and a lower MELD score.[18] The patient benefited from timely placement of transjugular intrahepatic portosystemic shunt (TIPS), which stabilized portal hypertension, reduced ascites, and improved quality of life. Management also focused on non-invasive interventions, including frequent paracenteses and empiric antibiotics. However, the present case faced a more severe disease burden, including septic shock, DKA, and hepatorenal syndrome, limiting therapeutic options and precluding TIPS placement. Both cases underscore the importance of a multidisciplinary approach tailored to the patient's clinical condition, emphasizing the need for proactive management in those with advanced liver disease.

This case illustrates the complexity of managing Flood syndrome, a condition with no standardized treatment protocols. The patient's care focused on conservative management due to her frailty and high perioperative mortality risk. Her multifaceted condition underscores the importance of a multidisciplinary approach tailored to the unique challenges of each patient.

CONCLUSION

Flood syndrome, a rare and potentially fatal complication of end-stage liver disease with ascites, presents significant challenges in management due to the lack of standardized treatment protocols. This case highlights the complexity of care, emphasizing the limitations of current interventions such as paracentesis, which, while providing temporary relief, can contribute to recurrent hernias and other complications. The patient's frailty and high surgical risk necessitated a conservative approach, reflecting the critical role of individualized, multidisciplinary care in addressing the unique needs of such patients. This underscores the need for further research to establish evidence-based guidelines for managing this challenging condition.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Consent for Publication

Oral consent for the publication of this case report, including all accompanying data and anonymized information, was obtained from the patient.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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