Case Reports

Ascites with a Chance of Flooding: A Rare Complication of Cirrhosis

Escher Howard-Williams, MD[1,2], Margaret Fennell, MD[2], Heather Frank, MD[2], Caroline Repetti, MD[2], Jessica Fuller, MD[1,2]

[1] Department of Hospital Medicine, University of North Carolina at Chapel Hill,
[2] Division of Medicine, University of North Carolina at Chapel Hill

Journal of Brown Hospital Medicine
Vol. 3, Issue 3, 2024

Article Information

Keywords: Cirrhosis, ascites, umbilical hernia, Flood Syndrome

https://doi.org/10.56305/001c.116952

Submitted: March 29, 2024 EST

Accepted: April 23, 2024 EST

Abstract

Flood Syndrome, characterized by ruptured umbilical hernias with ascitic fluid leakage in decompensated liver cirrhosis, presents a rare but potentially fatal complication of cirrhotic liver disease. We present a case study of a patient with decompensated cirrhosis who underwent multiple medical encounters before the severity of his condition, characterized by ascitic fluid leakage, was recognized. Flood Syndrome was ultimately identified on admission to the hospital, prompting multidisciplinary interventions and discussions, including antibiotics, surgical consultation, and optimization for hernia repair. Due to the patient's complexity of his medical condition and surgical risks, a transjugular intrahepatic portosystemic shunt (TIPS) was performed to alleviate ascites before a planned umbilical hernia repair. This case underscores the importance of early recognition of umbilical hernias in cirrhotic patients and highlights the complexity of management decisions, balancing surgical risks with medical optimization to improve outcomes. Further research is warranted to refine treatment strategies and enhance patient care in Flood Syndrome cases.

INTRODUCTION

Ascites represents a prevalent complication in decompensated liver cirrhosis. Studies indicate that as many as 20% of individuals afflicted by ascites ultimately manifest umbilical hernias due to heightened intra-abdominal pressure coupled with compromised abdominal musculature and connective tissue integrity. The occurrence of ruptured umbilical hernias accompanied by leakage of ascitic fluid, called Flood Syndrome, constitutes a rare but grave complication of cirrhosis with ascites characterized by high mortality rates.[1] In this article, we present a case study of a patient with Flood Syndrome, a condition that may not be promptly recognized among healthcare providers. The patient traversed multiple healthcare touchpoints before the urgency and severity of the syndrome were identified. This case highlights the clinical challenges associated with expeditiously recognizing and treating this lesser-known syndrome, emphasizing the importance of awareness and timely intervention in its clinical course.

CASE PRESENTATION

A 46-year-old male with a past medical history of hypertension, alcohol use disorder, and alcoholic cirrhosis decompensated by ascites for approximately one year presented to the emergency department (ED) reporting a single day of ascitic fluid leakage from his umbilicus. The patient states his umbilical hernia, followed by his primary care provider, was first noticed about one year ago and had gradually worsened over time. Upon arrival to the ED, he was afebrile, with a heart rate of 77/min, blood pressure of 108/73 mmHg, normal respirations, and oxygen saturation. Laboratory investigations were not conducted during this encounter. Clinical examination in the ED revealed notable abdominal ascites and a reducible, ulcerated umbilical hernia (**Figure 1**). In the ED, the ulcer was cleaned with sterile saline and the area of leakage was sealed with skin glue. Given that he was hemodynamically stable without associated symptoms of abdominal pain, tenderness of palpation, or otherwise the patient was subsequently discharged home.

One week after the initial encounter, the patient sought medical attention at the outpatient internal medicine clinic for a large volume paracentesis, during which a conspicuous outflow of ascitic fluid from the umbilical hernia was observed. Due to the briskness of ascitic fluid flow, the leaking hernia defect was unable to be sealed with skin glue and was left open. Additionally, erythema was recognized in the right lower quadrant of the abdomen. At that time his active home medications were Lasix 40 mg daily, spironolactone 100 mg daily, acamprosate 666 mg three times daily and atenolol 100 mg daily (presumably on a non-selective beta-blocker due the fact he did not have known esophageal varices at this point in his clinical course). Laboratory workup was ob-

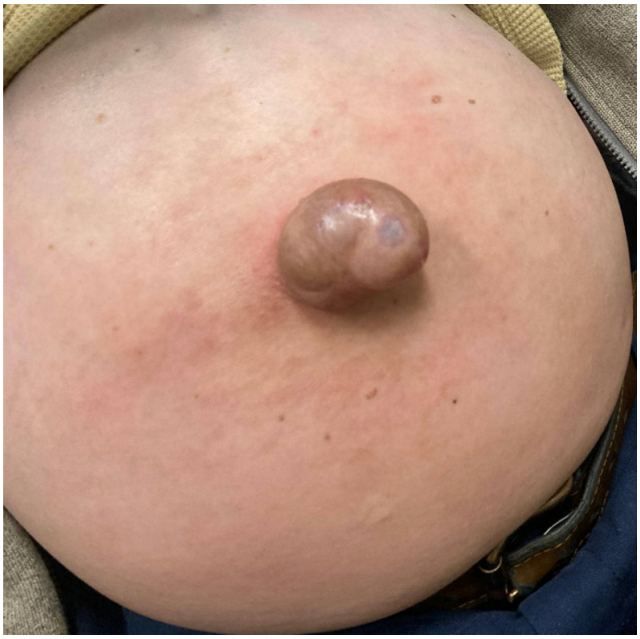

Figure 1. Ascitic abdomen with ulcerated umbilical hernia on initial presentation to emergency department.

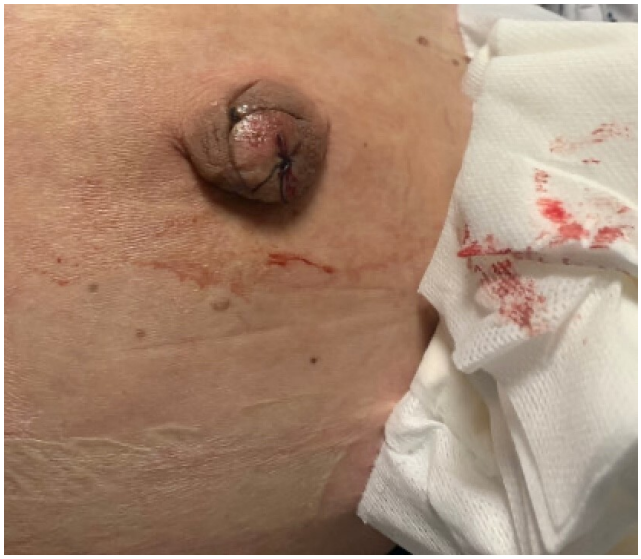

Figure 2. Umbilical hernia post suture repair on admission.

tained showing a mild leukocytosis to 13.5x10*9/L (3.6 - 11.210*9/L) and a new mild acute kidney injury (AKI) with creatinine 1.24 mg/dL (0.60 - 1.10 mg/dL), baseline 0.7 mg/dL. The patient underwent paracentesis with the extraction of 3 liters of ascitic fluid and 5 g of albumin was infused post procedure. The ascitic fluid was sent for analysis and returned with 118 nucleated cells with 1% neutrophils, not consistent with spontaneous bacterial peritonitis (SBP) or secondary peritonitis. No antibiotics were initiated, and he was scheduled for a repeat paracentesis one week later.

He returned to the ED later the same day due to large volume leakage from the umbilicus, and examination revealed persistent umbilical ulceration with more extensive surrounding erythema and a small amount of pus. Initial vital signs were normal including a temperature of 37.1C, heart rate of 76/min, blood pressure of 108/70mmHg, respiratory rate of 18/min, and O2 saturation of 100% on room air. He received 3.375 g of intravenous piperacillin/tazobactam, general surgery was consulted, and the ulcerated hernia leak was temporized with bedside sutures (**Figure 2**). At this juncture, the patient was admitted to the medicine service for further monitoring. Upon admission, the presence of Flood Syndrome was recognized.

Laboratory findings on admission revealed ongoing AKI with a creatinine of 1.17 mg/dL (0.60 - 1.10 mg/dL), leukocytosis of 14x10*9/L (3.6 - 11.210*9/L), albumin of 1.8 g/dL (3.4-5 g/dL), and INR 1.05. A calculated MELD 3.0 score was 11, and abdominal ultrasound showed patent hepatic vasculature with normal flow direction, sluggish flow in the main and anterior branch of the right portal vein with recanalized umbilical vein and large volume ascites. Also noted was an echogenic liver with nodular contour, consistent with history of chronic liver disease. Two sets of blood cultures were drawn and were negative for growth.

In response to concerns regarding cellulitis and the heightened risk of secondary bacterial peritonitis resulting from the ruptured hernia, the patient was initiated on intravenous Ceftriaxone. Simultaneously, consultations were sought from gastroenterology and general surgery, facilitating multidisciplinary deliberations regarding treatment strategies, particularly in consideration of the significant morbidity and mortality associated with Flood Syndrome.

Efforts were undertaken to medically optimize the patient for surgical hernia repair through the utilization of albumin-assisted diuresis with Lasix and Spironolactone. Regrettably, these endeavors proved unsuccessful due to worsening of renal injury.

Given the patient's persistent decompensated cirrhosis accompanied by active ascites, the perceived surgical risk was deemed prohibitively high for an immediate hernia repair. Consequently, the patient underwent a transjugular intrahepatic portosystemic shunt (TIPS) procedure alongside repeated large volume paracentesis. He was transitioned to oral ciprofloxacin for spontaneous and secondary bacterial peritonitis prophylaxis while the umbilical draining tract healed. Following optimization of the patient's fluid status, outpatient follow up for hernia repair was arranged.

DISCUSSION

Coined by Frank B. Flood in 1961, Flood Syndrome describes an umbilical hernia rupture in decompensated cirrhosis with spontaneous leakage of ascitic fluid.[2-4] Though initially described in 1901 by Johnson, Flood Syndrome is a rare and underrecognized clinical diagnosis

with high morbidity and mortality that should prompt inpatient admission for stabilization and further workup.[5] The syndrome is thought to arise from a combination of factors, including increased abdominal wall pressure from ascites potentially worsened by recurrent pressure changes from repeated large-volume paracenteses, poor nutritional status leading to decreased synthetic function and muscle integrity, and possible fascial tissue defects arising from recanalization of the umbilical vein from portal hypertension.[1,6,7] Complications of Flood Syndrome include bowel incarceration or evisceration, cellulitis, peritonitis, sepsis, and hemodynamic instability.[6,8] Given the significant associated morbidity and mortality, inpatient and outpatient providers should familiarize themselves with this syndrome to facilitate prompt inpatient evaluation.

Effective management strategies for Flood Syndrome pose a challenge, primarily attributed to the paucity of available data and the inherent complexity arising from the elevated surgical risk associated with end-stage liver disease (ESLD). Treatment modalities encompass a spectrum ranging from supportive measures such as superficial suture repair, fibrin glue injection, ostomy bag placement, and drain implementation to more decisive surgical interventions such as hernia repair.[4]

Common surgical treatments include umbilical hernia repair, TIPS, peritoneovenous shunting (PVS), and concomitant portal venous decompression.[7] Postoperative complications including wound dehiscence, evisceration caused by reaccumulation of ascitic fluid, bleeding due to coagulopathy from liver dysfunction, wound infection, umbilical hernia recurrence, prolonged ileus, and encephalopathy.[7-9]

Current recommendations suggest that patients with Flood Syndrome receive antibiotics for secondary and spontaneous bacterial peritonitis prophylaxis as the patient is also at risk for secondary peritonitis from skin pathogens with the loss of integrity of the abdominal wall.[1,10] Indications and ideal timing for hernia repair are controversial, with current practice recommending elective repair of the hernia once medical optimization of ascites is achieved. In a large study of Veterans with cirrhosis, 30-day mortality was 1.2% after nonemergency umbilical hernia repair and 12.2% after emergency umbilical hernia repair.[11] Deciding which course to pursue is difficult, as mortality rates with supportive management alone are 60-80%, while postoperative mortality rates are 6-20% but with significantly higher morbidity (up to 71%) as described above.[7,8,12] In our case, TIPS was used to decrease ascites over time to optimize for future hernia repair. One review suggests that Flood Syndrome treatment with TIPS is rare, with only 9 cases identified to date, but this is an area for further research.[6]

We have presented a case of Flood Syndrome, where the severity of this patient's clinical condition went unrecognized by multiple healthcare providers over several medical encounters before its eventual identification. Early recognition of umbilical hernias in the setting of decompensated cirrhosis with ascites is crucial in patient care. Once the hernia has progressed to ulceration and ascitic fluid leakage, the condition is considered a medical emergency. Early identification of umbilical hernias improves morbidity and mortality outcomes and enables comprehensive management strategies. Management strategies can include optimizing volume status and promptly engaging appropriate surgical and gastroenterological expertise for further definitive treatment.

Author Contributions

All authors have reviewed the final manuscript prior to submission. All the authors have contributed significantly to the manuscript, per the International Committee of Medical Journal Editors criteria of authorship.

- Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND
- Drafting the work or revising it critically for important intellectual content; AND
- Final approval of the version to be published; AND
- Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Disclosures/Conflicts of Interest

All authors declare that there are no acknowledgements, conflicts of interest nor funding to disclose.

Corresponding author

Escher Howard-Williams, MD
University of North Carolina, School of Medicine
Department of Medicine, Division of Hospital Medicine
Associate Professor of Medicine
101 Manning Dr, Chapel Hill, NC 27599
Phone: (984) 974-1901
Email: Eschermc@med.unc.edu

REFERENCES

1. Coelho JCU, Claus CMP, Campos ACL, Costa MAR, Blum C. Umbilical hernia in patients with liver cirrhosis: A surgical challenge. *World J Gastrointest Surg*. 2016;8(7):476-482. doi:10.4240/wjgs.v8.i7.476

2. Belghiti J, Durand F. Abdominal wall hernias in the setting of cirrhosis. *Semin Liver Dis*. 1997;17(3):219-226. doi:10.1055/s-2007-1007199

3. Dokmak S, Aussilhou B, Belghiti J. Umbilical hernias and cirrhose. *J Visc Surg*. 2012;149(5 Suppl):e32-9. doi:10.1016/j.jviscsurg.2012.04.002

4. Flood FB. Spontaneous perforation of the umbilicus in Laennec's cirrhosis with massive ascites. *N Engl J Med*. 1961;264:72-74. doi:10.1056/NEJM196101122640204

5. Blanco Terés L, Valdés de Anca Á, Correa Bonito A, Gancedo Quintana Á, Martín Pérez E. Flood Syndrome: A severe complication of umbilical hernia. *Cirugía Española (English Edition)*. 2020;98(8):490-491. doi:10.1016/j.cireng.2020.09.006

6. Liu GF, Srinivasan A, Mutnuri S, Yerramadha MR, Agraharkar M. Acute abdomen from umbilical hernia rupture to Flood Syndrome: A case report and review of literature. *J Med Cases*. 2019;10(10):309-311. doi:10.14740/jmc3375

7. Chatzizacharias NA, Bradley JA, Harper S, et al. Successful surgical management of ruptured umbilical hernias in cirrhotic patients. *World J Gastroenterol*. 2015;21(10):3109-3113. doi:10.3748/wjg.v21.i10.3109

8. Strainiene S, Peciulyte M, Strainys T, et al. Management of Flood Syndrome: What can we do better? *World J Gastroenterol*. 2021;27(32):5297-5305. doi:10.3748/wjg.v27.i32.5297

9. Baron HC. Umbilical hernia secondary to cirrhosis of the liver. Complications of surgical correction. *N Engl J Med*. 1960;263:824-828. doi:10.1056/NEJM196010272631702

10. Lemmer JH, Strodel WE, Knol JA, Eckhauser FE. Management of spontaneous umbilical hernia disruption in the cirrhotic patient. *Ann Surg*. 1983;198(1):30-34. doi:10.1097/00000658-198307000-00006

11. Johnson KM, Newman KL, Berry K, et al. Risk factors for adverse outcomes in emergency versus nonemergency open umbilical hernia repair and opportunities for elective repair in a national cohort of patients with cirrhosis. *Surgery*. 2022;172(1):184-192. doi:10.1016/j.surg.2021.12.004

12. Lee JL, Jiang J. Flood Syndrome. *Gastroenterology Res*. 2022;15(4):217-224. doi:10.14740/gr1508