



Flood Syndrome: A Rare Case of Ruptured Umbilical Hernia

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Introduction

- Flood syndrome is an extremely rare condition defined as a **rupture of an umbilical hernia** secondary to long standing ascites in patients with end-stage liver disease.¹
- This was first published by Dr. Frank Flood in 1961¹ and since then, reports in the literature have remained few.
- Interestingly, approximately 20% of patients with liver disease go on to develop an umbilical hernia.²
- The theory behind this high rate of hernia formation is increased intraabdominal pressure due to ascites (and large pressure changes after large volume paracentesis)³⁻⁵ and the inherent weakness of the linea alba.⁶
- Many patients with advanced liver disease and concomitant hernias are not offered surgical intervention due to their increased risk of surgical complication.
- This puts them at risk for going on to develop hernia rupture although the pathophysiology for it is not very well understood.
- It is hypothesized that a combination of the pressure changes, poor nutrition/hypoalbuminemia, and portal hypertension are responsible for this occurrence.⁷
- Hernia skin ulceration and/or excoriation nearly always precedes the rupture.⁸
- Flood syndrome can be potentially fatal. Complications include hernia incarceration, bowel evisceration, cellulitis, peritonitis, and sepsis.^{5,9}

Case Description

History of Present Illness:

- A 45-year-old female with a past medical history significant for Von Willebrand type 1 and severe Child's C alcoholic cirrhosis with a MELD score of 18 presented to the emergency department (ED) with **fluid leaking** from a known umbilical hernia
- The patient previously underwent weekly large volume paracentesis to manage her ascites; however, this had recently been switched to every-other-week
- She endorsed knowing about the umbilical hernia for many years; however, it had gotten larger in the months prior to her presentation with fluid leaking from it
- She was awaiting liver transplantation
- She has been sober from alcohol for 2 years

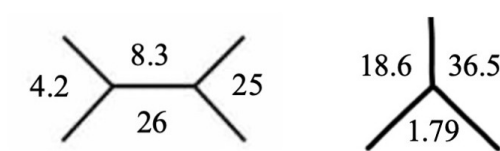
Past Medical History

- Alcohol cirrhosis
- Alcohol use disorder
- Anemia
- Anxiety and depression
- Ascites
- Esophageal varices
- Pancreatitis
- Supraventricular tachycardia
- Von Willebrand disease, type 1

Medications

- Furosemide 40 mg PO daily
- Lactulose 30 g PO TID
- Pantoprazole 40 mg PO daily
- Propranolol 20 mg PO daily
- Rifaximin 550 mg PO BID
- Spironolactone 100 mg PO daily
- Thiamine 100 mg PO daily
- Egocalciferol 50,000 U PO weekly
- Folic acid 1 mg PO daily

Laboratory Analysis



TBili: 3.4 mg/dL
DBili: 1.0 mg/dL
AST: 46 U/L
ALT: 16 U/L
AlkPhos: 100 U/L

Physical Examination

Vital Signs

- T:** 98.6°F
- HR:** 127 bpm
- BP:** 116/76 mmHg
- SPO₂:** 98% RA

Physical Examination

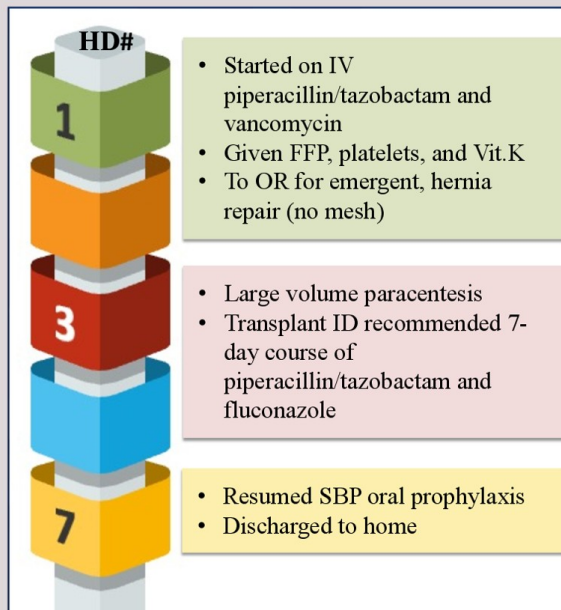
- Gen:** Alert, NAD
- CVS:** tachycardic but regular
- Abd:** softly distended, tender over the umbilicus which was weeping a copious amount of straw-colored fluid and had an area of central necrosis (**Image 1**)

Image 1: Umbilicus at presentation



Umbilicus with central necrosis and expression of ascitic fluid

Hospital Course



Discussion

- Due to its rarity there is no standard of care for the treatment of Flood syndrome; however, mortality rates are extremely high without surgery.¹⁻³
- Without surgical intervention, mortality rates approach 80%.¹⁻³
- With* surgical intervention, mortality rates remain high but drop dramatically to 6-20%.¹⁻³
- In addition to herniorrhaphy, peritoneovenous shunting, or transjugular intrahepatic portosystemic shunting can be considered for management.^{3,9}
- Spontaneous bacterial peritonitis prophylaxis is recommended for all patients with Flood syndrome.⁴

Conclusion

- Flood Syndrome is a rare but life-threatening complication of chronic liver disease, most often associated with end-stage cirrhosis and ascites. Prompt recognition and timely surgical intervention are critical, as conservative management results in high morbidity and mortality.

References

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