

Introduction

Flood syndrome is a rare life-threatening complication of spontaneous paracentesis from a ruptured umbilical hernia typically in the setting of refractory ascites and end-stage liver cirrhosis. While conservative management is associated with higher mortality compared to surgical management, herniorrhaphy in patients with end-stage liver cirrhosis remains difficult to plan due to a high-risk of morbidity and mortality. We present a case of flood syndrome and our multidisciplinary approach for management.

Case Description

A 59-year-old male, with decompensated alcoholic cirrhosis including esophageal varices, recurrent ascites, and hepatocellular carcinoma, presented with hepatic encephalopathy and a ruptured umbilical hernia leaking ascites. On presentation he was hemodynamically stable and afebrile. Most initial labs were unremarkable, except for elevated ammonia of 178 and lactic acidosis of 4.9. His MELD-Na score on admission was 30. On physical examination the patient was cachectic, toxic-appearing, positive for scleral icterus, and jaundiced. He had significant abdominal distention with a slightly compressible ulceration at the site of the umbilical hernia. A CT head was obtained which was unremarkable. A CT abdomen was negative for incarcerated hernia or obstruction. After obtaining cultures, he was started on broad spectrum antibiotics for possible peritonitis in the setting of flood syndrome. Additionally, he received albumin, lactulose, and rifaximin.

Medical and surgical planning included evaluation by gastroenterology, infectious disease, and surgery for medical optimization with the aim of pursuing elective repair when the patient was at his lowest MELD score. Unfortunately, he deteriorated clinically with worsening encephalopathy and hemodynamic instability. Ultimately, he was considered too unstable for herniorrhaphy.

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Flood syndrome: A challenge for the clinician

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Case Description



On day 2/3 the patient developed new onset hypoxic respiratory failure requiring intubation. He was hypotensive likely secondary to hypovolemia and systemic vasodilation secondary to liver disease. He was also found to be in acute renal failure with concerns for hepatorenal syndrome in the setting of decompensated liver disease. The patient was started on norepinephrine, midodrine, octreotide and albumin

On day 15 the patient was noted to have profuse rectal bleeding due to rectal varices, requiring a hemostat and vicryl stitch. At that time no further surgical intervention was recommended as the patient was not hemodynamically stable. On the following day, he continued to deteriorate, therefore he was transitioned to comfort measures and subsequently he expired.



His hospital course was complicated by Staphylococcus lugdunensis and vancomycin-resistant Enterococcus faecium peritonitis and sepsis despite multiple antimicrobials.

Figure 1. A 6.5 cm Umbilical hernia



Discussion

Flood syndrome has a poor prognosis with a high mortality rate. Patients with end-stage liver disease and recurrent ascites are at higher risk due to increased intra-abdominal pressure, weakened abdominal muscle and fascia, and malnutrition. There is no standard of care for the management, but literature favors early surgical intervention. Our case shows the difficulty and complexity of managing patients with flood syndrome. Despite utilization of a multidisciplinary approach to care for our patient, the severity of our patient's liver disease and his frailty made him ineligible for surgical intervention. Early surgical intervention to prevent rupture of these umbilical hernias may ensure the best chance at positive patient outcomes.

Learning points:

- Physicians should be suspicious for flood syndrome if they note any umbilical changes in patients with refractory ascites and liver cirrhosis including color changes, ulcerations or any leaking.
- Postoperative control is essential for the prevention of further complications and recurrence of umbilical hernias.

References

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