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**Case Report**

# Spontaneous umbilical hernia rupture with omental evisceration and Flood syndrome

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## ABSTRACT

Cirrhotic patients are at increased risk of developing umbilical hernias. Many cirrhotic patients' umbilical hernias are not repaired electively due to concerns for high perioperative morbidity and mortality. This case report aims to inform clinicians about the unique challenges that arise during emergency management of umbilical hernias in the cirrhotic patient. A 59-year-old male with Child-Turcotte-Pugh grade B cirrhosis presented to our hospital with an incarcerated umbilical hernia that spontaneously ruptured with large volume ascitic leak (known as Flood syndrome) and omental evisceration. The patient underwent emergency sutured umbilical hernia repair, and required a prolonged post-operative stay in the hospital intensive care unit after suffering from complications including spontaneous bacterial peritonitis, anaphylaxis to antibiotic treatment, aspiration pneumonia, encephalopathy and worsening ascites. He eventually made a good recovery and underwent rehabilitation prior to discharge home. This case highlights the rare complication of spontaneous omental evisceration of an umbilical hernia in the cirrhotic patient and details its subsequent management. It is important to note that elective hernia repair after medical optimisation is high risk in the cirrhotic patient, but is recommended to avoid the high perioperative mortality and morbidity associated with emergency repair.

**Keywords:** Hernia, Evisceration, Cirrhosis, Flood syndrome

## INTRODUCTION

Patients with cirrhosis are inherently at risk of developing abdominal wall hernias due to increased intrabdominal pressure (associated with ascites), weakening of the abdominal fascia, muscle wasting from malnutrition, and recanalisation of the umbilical vein.[1,2] Of all patients with cirrhosis, 20-40% will have umbilical hernias.[1,3] Conservative management of umbilical hernias has been found to be successful in only 23% of cases, with almost half of patients requiring emergency surgery for complications such as incarceration, strangulation, rupture with ascites leak, evisceration or death.[4]

Operating on the patient with cirrhosis is fraught with danger. Decompensation of the cirrhotic patients' liver disease following surgery is common, and can have lethal consequences.[5] The operative mortality rate relative to the severity of liver disease is quoted as up to 10%, 30% and 82% for patients classified as Child-Turcotte-Pugh (CTP) A, B and C respectively.[6,7] In the past, surgeons have opted for non-operative management of hernias due to concerns regarding morbidity, mortality and hernia recurrence.[8] Newer studies have demonstrated that mortality is increased up to sevenfold in the emergency setting compared to elective repairs.[9] Elective umbilical hernia repair is successful in most cirrhotic patients when ascites is well controlled.[8]

This case report details the rare complication of spontaneous rupture, omental evisceration and ascites leak of an umbilical hernia in a patient with CTP B cirrhosis. This case highlights to clinicians the unique challenges that are faced during emergency hernia repair of the cirrhotic patient, and during the post-operative recovery period.

## CASE REPORT

A 59-year-old male with cirrhosis secondary to alcohol abuse, Hepatitis B and Hepatitis C infection (CTP Grade B and model for end-stage liver disease score =9) complicated by massive ascites, portal hypertension and splenomegaly presented to our hospital after spontaneous rupture of his umbilical hernia. He had noticed ascitic leak from a small skin defect overlying his longstanding umbilical hernia approximately 4 days prior to presentation to hospital. He had been collecting the ascitic fluid in ice-cream containers at home and had collected approximately 7 l of fluid. He presented to hospital after a coughing fit precipitated omental evisceration through the skin defect. He reported feeling otherwise well in himself. His other past medical history was significant only for untreated chronic obstructive pulmonary disease, cigarette smoking of 5 cigarettes daily, and peripheral neuropathy.

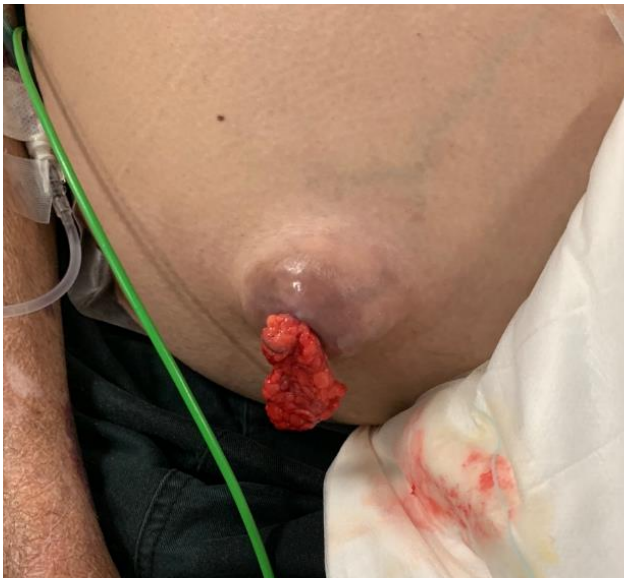

**Figure 1: A tongue of omentum can be seen protruding through the skin defect overlying the incarcerated umbilical hernia. The patient's abdomen is distended by retained ascitic fluid, with visible caput medusae.**

He was hemodynamically stable and afebrile at presentation, with infarcted omentum seen to be protruding through the skin defect overlying his umbilical hernia (Figure 1). The umbilical hernia was tense, tender and irreducible. His abdomen was mildly generally tender on examination. His initial bloods revealed a lactate level of 2.6 mmol/l (0.5-2.2 mmol/l), pH 7.36 (7.32-7.43), sodium 136 mmol/l (135-145 mmol/l), potassium 4.0 mmol/l (3.5-5.2 mmol/l), estimated glomerular filtration rate 73 ml/min/1.73 $m^2$ (>60 ml/min/1.73 $m^2$), albumin 20 g/l (35-50 g/l), total bilirubin 15 umol/l (<20 umol/l), conjugated bilirubin 5 (<4 umol/l), alkaline phosphatase 90 u/l (30-110 u/l), gamma glutamyl transferase 24 u/l (<55 u/l), alanine aminotransferase 15 u/l (<45 u/l), aspartate aminotransferase 35 u/l (<35 u/l), lipase 64 u/l (< 60 u/l), haemoglobin 121 g/l (135-180 g/l), white cell count 10.7 × $10^9$/l (4.0-11.0×$10^9$/l), platelets 234×$10^9$/l ( 140-400×$10^9$/l), international normalised ratio 1.2 (0.9-1.2) and a positive hepatitis C Ab IgG screen.

While awaiting surgical review, he was administered intravenous antibiotics (ceftriaxone and metronidazole) to treat for presumed associated bacterial peritonitis. He had no known allergies. Unfortunately, he rapidly developed an anaphylactic reaction to ceftriaxone, with ensuing bronchospasm and severe respiratory acidosis (venous blood gas pH 7.12 [7.32-7.43], $pCO_2$ 81 mmHg [32-48 mmHg], $pO_2$ 30 mmHg [83-108 mmHg], $HCO_3$ 26 mmol/l [22-32 mmol/l], lactate 5.5 mmol/l [0.5-2.2 mmol/l]). He was successfully treated with intramuscular, then intravenous adrenaline, and stabilised. The intensive care team, anaesthetics team and surgical team consulted on the patient with respect to optimal timing for surgical intervention given his recent anaphylaxis. When the patient had stabilised, he was taken to theatre on an adrenaline infusion to prevent intraoperative recurrence of anaphylaxis. No pre-operative imaging of the abdomen was undertaken as the requirement for surgical intervention was clear and there was no concern at the time for other surgical complications (for example, bowel strangulation).

The infarcted eviscerated omentum was resected, and the hernia defect closed primarily using interrupted sutures of 1/0 nylon. A sample of ascitic fluid was drained through the incision and sent for culture (no growth found after 5 days of culture). However, the full volume of ascites was not drained due to concerns regarding haemodynamic instability, as the patient began to require further ionotropic support to maintain his blood pressure during the procedure. The small bowel was not delivered and inspected in full as it was visibly not involved in the hernia and appeared grossly normal. The overlying ruptured umbilical skin was also excised and the incision was closed primarily with a combination of dermal 2/0 vicryl and staples to the skin.

Post-operatively the patient was transferred intubated to the intensive care unit for further management, on dual ionotrope therapy. His admission was complicated by aspiration pneumonia, decompensation of his liver disease with encephalopathy and worsening ascites, and feed intolerance requiring total parenteral nutrition. An ascitic drain was inserted post-operatively, with 500 ml of 4% albumin administered for every 2 l drained. He was treated with meropenem for 7 days total for sepsis

presumed secondary to bacterial peritonitis, although his ascitic cultures remained negative. His renal function remained stable throughout his admission with no evidence of hepatorenal syndrome. He was eventually stepped down to the ward for continuing cares after 14 days in the intensive care unit. After undergoing rehabilitation, he was discharged to his home after 32 days in hospital. His hernia had not recurred when reviewed in clinic several months following his procedure, and he remained independent at home.

## DISCUSSION

Traditionally, surgeons have advocated for non-operative management of hernias in the cirrhotic patient due to the high risks of anaesthesia and surgery in these comorbid patients.[1] However, recent research has demonstrated that conservative management of these hernias is associated with considerably worse mortality and morbidity than elective surgical repair after medical optimisation.[1]

In our case, the patient's umbilical hernia ruptured in the setting of gross ascites and required emergency repair. Flood syndrome is the eponym for spontaneous umbilical hernia rupture with drainage of ascitic fluid, a rare complication that usually occurs in the setting of ascites and cirrhosis. It was first described in 1961 by Frank Flood. It carries a mortality rate of approximately 30%, usually from bacterial peritonitis and associated sepsis.[3] Nonsurgical management of Flood syndrome carries a mortality rate of 60-80%, and therefore elective repair following medical optimisation is advocated for.[10] Only a few cases of Flood syndrome associated with evisceration have been reported.[11-17] Due to our patient's decompensated liver disease at the time of presentation, his post-operative course was complicated by encephalopathy, worsening ascites (managed with ascitic drain), aspiration pneumonia and bacterial peritonitis, requiring a prolonged stay in hospital. His initial antibiotic treatment for presumed associated intrabdominal infection resulted in an anaphylactic reaction that substantially increased his risk of mortality. His hernia repair remained intact throughout his stay and on follow up, although the literature quotes a recurrence rate as high as 60% after repair in this patient subset.[18] Primary closure with non-absorbable sutures, and not mesh, is the recommended surgical technique to avoid infection in this patient cohort.[10]

Perioperative optimisation of the patient's cirrhosis remains the mainstay of their care. Some centres advocate for concomitant portal venous decompression (via transjugular intrahepatic portosystemic shunting) to decrease the risk of postoperative recurrence due to ascites.[17] Maximal medical management with paracentesis, intravenous volume replacement with albumin, nutritional optimisation, broad spectrum antibiotics, diuretics, lactulose, reversal of coagulopathy with vitamin K, and cessation of hepatotoxic medications, is critical to manage decompensation and prevent hernia recurrence.[10,5,19]

In summary, umbilical hernia repair in the elective setting after medical optimisation in patients with cirrhosis is daunting, but recommended to avoid emergency repair and its associated high mortality and morbidity.[20] Pre-operative medical optimisation of the patient's cirrhosis is critical to prevent hernia recurrence and decrease post-operative complications. Emergency repair of the ruptured umbilical hernia with evisceration in the decompensated cirrhotic is possible, and can achieve a good recovery as in our patient. Post-operative management of the patient's cirrhosis is the most challenging aspect of their care and requires a multidisciplinary team approach.

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