



Flood Syndrome Complicated by Omental Evisceration in Budd-Chiari Syndrome-Related Ascites: A Case Report

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Abstract:

Introduction and Importance: Umbilical hernia is common in patients with chronic ascites, but spontaneous rupture with evisceration is rare and potentially fatal. Most published reports concern cirrhosis; occurrence in Budd-Chiari syndrome is less frequently documented. This report describes omental evisceration after a short prodrome of umbilical swelling and ascitic leakage in a young woman with Budd-Chiari syndrome. **Presentation of Case:** A 27-year-old woman with Budd-Chiari syndrome and chronic ascites, treated with acenocoumarol, spironolactone and furosemide, was transferred for omental evisceration through an umbilical defect. One week earlier, she had developed an umbilical swelling followed by clear fluid leakage after scratching the overlying skin. She remained afebrile and haemodynamically stable, with preserved bowel transit and no peritoneal irritation. The abdomen was distended and a viable omental segment protruded through a 2-cm cutaneous defect. Laboratory testing showed severe hyponatraemia (118 mmol/L), thrombocytopenia ($90 \times 10^9/L$), a prothrombin time of 40% and an international normalised ratio of 1.89. After critical-care review and correction of hyponatraemia, emergency midline laparotomy confirmed viable eviscerated omentum. Omentectomy and primary umbilical hernia repair with four interrupted X sutures of absorbable suture material size 1 were performed. The immediate postoperative course was reported as uncomplicated. **Clinical Discussion:** Refractory ascites increases intra-abdominal pressure, enlarges the umbilical ring and compromises the overlying skin. Ulceration or ascitic leakage may precede rupture. Evisceration requires urgent operative management after focused resuscitation. Primary suture repair avoids implantation of prosthetic material in an emergency field, although recurrence remains closely linked to postoperative ascites control. **Conclusion:** Umbilical leakage in a patient with ascites should be treated as a warning sign of impending rupture. Exposed omentum or bowel requires protection, physiological optimisation and urgent surgery. Long-term success depends on coordinated control of ascites and the underlying hepatic venous outflow obstruction.

Keywords: Budd-Chiari Syndrome, Ascites, Umbilical Hernia, Omental Evisceration, Flood Syndrome, Case Report.

Case Report

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INTRODUCTION

Umbilical hernias develop in approximately 20% of patients with cirrhosis and ascites and may affect up to 40% of those with large-volume ascites [2]. Persistently increased intra-abdominal pressure, fascial weakening and poor tissue nutrition promote

progressive enlargement, skin thinning and, rarely, spontaneous rupture. The resulting external loss of ascitic fluid is commonly termed Flood syndrome. Reported complications include cellulitis, peritonitis, haemodynamic instability, incarceration and evisceration of omentum or bowel [2, 3].

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Published evidence is dominated by cirrhotic populations. Spontaneous rupture with omental evisceration in a young patient with Budd-Chiari syndrome is unusual and highlights two practical issues: clear umbilical leakage can precede complete disruption, and emergency repair must balance rapid source control against severe electrolyte, coagulation and hepatic abnormalities. This case report has been reported in line with the SCARE 2023 criteria [1].

Case Presentation

Patient Information

A 27-year-old woman, gravida 1 para 1, was transferred from another hospital to a surgical service for management of an umbilical evisceration. She had been followed for three years by gastroenterology and internal medicine for Budd-Chiari syndrome with ascites; the aetiological evaluation was described as ongoing. Her regular treatment comprised acenocoumarol 1mg, spironolactone and furosemide. She had completed treatment for pulmonary tuberculosis in 2018. The patient had no known allergies, did not smoke or consume alcohol, and had no relevant family medical history.

One week before transfer, she noticed a new umbilical swelling. Scratching the overlying skin was followed by continuous clear fluid leakage, clinically compatible with ascitic fluid. At the referring hospital she received a support garment and topical treatment. She subsequently developed protrusion of intra-abdominal tissue through the umbilicus. Bowel movements and flatus continued. She returned to the emergency department and was transferred for specialist surgical care.

Clinical findings

On arrival, she was conscious, afebrile and jaundiced. Blood pressure was 110/80 mmHg, heart rate 113 beats/min and peripheral oxygen saturation 98% on an unspecified oxygen setting. She had bilateral pitting lower-limb oedema. The abdomen was distended but soft and non-tender, without guarding or rigidity. Shifting dullness was present. Neither the liver nor spleen was palpable. Viable omentum protruded through an approximately 2-cm umbilical skin defect (Figure 2). The remaining hernial orifices were clinically free. Right-sided chest percussion was dull. No

dilated abdominal wall veins or spider naevi were documented.



Figure 1: Admission chest radiograph showing near-complete opacification of the right hemithorax

Diagnostic assessment

Haemoglobin was 13 g/dL, white-cell count $6.0 \times 10^9/L$, platelet count 90 000 /mm³ and C-reactive protein 41 mg/L. Serum sodium was 118 mmol/L and potassium 4.8 mmol/L. Prothrombin time was 40% and the international normalised ratio was 1.89. Reported liver tests were total bilirubin 71.17 mg/L, direct bilirubin 31.50 mg/L, indirect bilirubin 40 mg/L, aspartate aminotransferase 87 UI/L, alanine aminotransferase 50 UI/L, gamma-glutamyl transferase 79 UI/L and alkaline phosphatase 203 UI/L. A chest radiograph showed near-complete opacification of the right hemithorax (Figure 1), clinically interpreted in the context of right-sided dullness.

The combination of an umbilical defect, exposed viable omentum and preceding clear ascitic leakage established the diagnosis of spontaneous umbilical hernia rupture with omental evisceration. Bowel evisceration, strangulation and peritonitis were specifically assessed because they would influence resection and contamination; ongoing bowel transit, a soft abdomen, the viable appearance of the protruding tissue and the absence of peritoneal signs argued against these complications. Cross-sectional imaging was not required to establish the diagnosis and would have delayed definitive treatment.



Figure 2: Omental evisceration through the ruptured umbilical hernia. The protruding omentum appeared viable at presentation. The approximately 2-cm cutaneous defect is visible at the umbilicus

Timeline

Table 1: Clinical timeline

Time	Setting	Event
Day -7	Home	New umbilical swelling; scratching of the skin followed by clear fluid leakage.
After onset	Referring hospital	Support garment and topical treatment prescribed.
Day 0	Referring emergency department	Omental evisceration developed with preserved passage of stool and flatus; patient transferred.
Day 0	Receiving hospital	Tachycardia, tense ascites and viable omental evisceration documented; sodium 118 mmol/L, platelets 90 000 /mm ³ and INR 1.89.
Preoperative period	Critical care and surgical services	Hyponatraemia corrected after critical-care review; 5 g sodium chloride by syringe pump followed by 4 g by infusion was documented.
Operation	Operating theatre	Midline laparotomy, omentectomy and primary four-point X-suture repair with absorbable suture material size 1.
Postoperative period	Surgical ward	Immediate postoperative course described as uncomplicated.

Therapeutic Intervention

The exposed omentum was judged viable and the patient had no clinical evidence of bowel obstruction or diffuse peritonitis. Emergency surgery was therefore indicated to remove exposed tissue, close the abdominal wall and prevent contamination, strangulation and further fluid loss. Because serum sodium was 118 mmol/L, correction was initiated after critical-care consultation. Following critical care consultation, correction of hyponatraemia was initiated with a documented 5 g of sodium

chloride administered via syringe pump, followed by 4 g by intravenous infusion.

The procedure was performed under general anaesthesia with the patient in the supine position. A midline laparotomy was performed. Exploration confirmed that the eviscerated content consisted of viable omentum, with no evidence of bowel compromise or intraperitoneal contamination. The exposed omentum was resected, and the umbilical defect was closed primarily with four interrupted X-shaped sutures using size 1

absorbable suture material. No mesh was implanted.

Follow-up and outcomes

The postoperative course was uneventful, with favourable clinical and laboratory evolution. The patient's regular treatment was resumed before discharge on postoperative day 5. Wound healing was satisfactory, with no hernia recurrence or further ascitic leakage during the two-month follow-up period.

DISCUSSION

This case documents spontaneous rupture of an umbilical hernia with omental evisceration in a young woman with chronic ascites caused by Budd-Chiari syndrome. Its most instructive feature is the warning sequence: recent umbilical swelling, skin trauma, persistent clear leakage and then evisceration. Although Flood syndrome is usually defined by spontaneous ascitic leakage through a ruptured umbilical hernia, the present case represents its complicated end of the spectrum because intra-abdominal tissue protruded through the defect.

Ascites creates sustained radial stress on the umbilical ring. Progressive hernia enlargement stretches and thins the overlying skin, while oedema and poor perfusion impair healing. Local trauma or a sudden rise in intra-abdominal pressure can precipitate disruption [2]. Omental or bowel evisceration is uncommon but well described [3]. The appearance of clear umbilical leakage should therefore trigger urgent surgical and hepatology assessment rather than simple topical care or external compression.

Emergency management begins with a sterile saline-moistened covering for exposed viscera, analgesia, fluid and electrolyte assessment, infection prevention and prompt surgical evaluation. Severe hyponatraemia, thrombocytopenia and prolonged coagulation tests complicate anaesthesia and haemostasis, but optimisation must be focused and time-limited when viscera are exposed. Published

reviews report mortality of 6%-20% after urgent repair compared with 60%-80% with supportive treatment alone, although these estimates arise from small retrospective series and older reports [2].

The operative strategy depends on tissue viability, contamination, defect size and the ability to control ascites. Primary suture repair is commonly favoured in a ruptured or contaminated field. Mesh reduces recurrence in selected patients, but ascitic leakage and infection can lead to mesh contamination; its role is better established in elective repair after medical optimisation [4]. Omentectomy combined with primary closure was appropriate in this emergency setting. No hernia recurrence or further ascitic leakage was observed during the two-month follow-up.

Outcomes are consistently poorer after emergency than elective umbilical hernia repair in patients with cirrhosis. A national cohort reported 30-day mortality of 12.2% after emergency repair versus 1.2% after non-emergency repair [5], and a systematic review confirmed higher mortality and postoperative morbidity in cirrhotic patients, while grading the evidence as very low quality [6]. The CRUCIAL randomised trial showed that elective repair can be feasible in selected patients with cirrhosis and ascites, although complications remained frequent [7]. These data support surveillance of umbilical hernias and consideration of planned repair after ascites optimisation rather than waiting for rupture.

Postoperative control of ascites is central because recurrent high intra-abdominal pressure promotes leakage, dehiscence and hernia recurrence [2, 8]. Management should include sodium restriction, rational diuretic therapy, evaluation for therapeutic paracentesis with albumin when indicated and treatment of the underlying portal hypertensive disorder. In Budd-Chiari syndrome, current guidance recommends confirmation of hepatic venous outflow

obstruction and a stepwise strategy incorporating anticoagulation, endovascular treatment and transjugular intrahepatic portosystemic shunting when appropriate [11]. Decisions must be individualised with hepatology, interventional radiology and surgery.

Strengths and Limitations

This report provides direct clinical and radiographic documentation of a rare emergency and describes its surgical management. The case is of multidisciplinary relevance to emergency surgery, hepatology, anaesthesia, and critical care. Its main limitation is the lack of detailed data regarding the severity of Budd-Chiari syndrome and ascites, as the etiological and prognostic assessment was still ongoing at the time of surgery. This limitation precluded a comprehensive assessment of operative risk and postoperative hepatic outcomes.

CONCLUSION

Persistent clear discharge from an umbilical hernia in a patient with ascites should be regarded as impending rupture. Omental or bowel evisceration requires protection of the exposed tissue, rapid correction of immediately reversible physiological abnormalities and urgent operative repair. Primary suture closure is a reasonable emergency option when prosthetic implantation is undesirable. Prevention of recurrence depends on sustained control of ascites and definitive management of the underlying liver disease.

Highlights

- Spontaneous rupture of an umbilical hernia in tense ascites is a surgical emergency with substantial morbidity and mortality.
- Omental evisceration may follow a warning phase of umbilical swelling, skin injury and clear ascitic leakage.
- Initial management should protect the exposed viscera and correct reversible physiological abnormalities without avoidable delay to surgery.

- Primary fascial closure without mesh can be appropriate during emergency repair when contamination, ascitic leakage or poor tissue quality increases implant-related risk.
- Durable ascites control and hepatology follow-up are central to preventing wound failure and recurrence.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

Ethical Approval: Ethical approval was not required for this single case report.

Author Contributions: All authors have read and approved the final version of the manuscript.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

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