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Spontaneous Rupture of Incisional Hernia with Bowel Evisceration in A Patient with Decompensated Chronic Liver Disease: A Case Report and Review of Literature

Ruchi Yadav[1], Ravi Kale[2], Ashish Moudgil[3]
[1]*Junior Resident, Department of General Surgery, Sharda Hospital, Greater Noida, India*
[2]*Associate Professor, Department of General Surgery, Sharda Hospital, Greater Noida, India*
[3]*Professor, Department of General Surgery, Sharda Hospital, Greater Noida, India*
Corresponding Author: Dr. Ruchi Yadav
Department of General Surgery
Sharda Hospital, Greater Noida, India

Abstract
Background
Abdominal wall hernias are common in patients with chronic liver disease (CLD), particularly in the presence of refractory ascites. Persistent elevation of intra-abdominal pressure, impaired wound healing, malnutrition, and muscle wasting contribute to hernia formation and recurrence. Spontaneous rupture of an abdominal wall hernia with bowel evisceration is an extremely rare but life-threatening complication associated with significant morbidity and mortality.
Case Presentation
A 47-year-old male with alcohol-related chronic liver disease, recurrent ascites, and previous umbilical hernia repair presented with spontaneous rupture of a large incisional hernia following a bout of coughing. Clinical examination revealed a 7 × 5 cm abdominal wall defect with eviscerated small bowel loops, ascitic fluid leakage, and overlying skin necrosis. The patient was admitted to the intensive care unit and underwent emergency resuscitation, local debridement, reduction of bowel contents, and temporary closure. ***Despite aggressive*** *supportive treatment, the patient developed progressive hepatic decompensation and ultimately succumbed to his illness.*
Conclusion
Spontaneous rupture of incisional hernias in cirrhotic patients is a surgical emergency associated with poor outcomes. Early recognition of warning signs such as skin ulceration and prompt elective repair before advanced hepatic decompensation may prevent catastrophic complications.

Keywords
Incisional hernia; Chronic liver disease; Ascites; Bowel evisceration; Flood syndrome; Ventral hernia rupture.

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I. INTRODUCTION

Abdominal wall hernias represent one of the most common surgical complications encountered in patients with chronic liver disease and ascites. The incidence of abdominal wall hernias exceeds 20% in patients with compensated cirrhosis and may approach 40% in those with significant ascites.[1]

The pathogenesis is multifactorial. Increased intra-abdominal pressure resulting from persistent ascites, malnutrition, muscle wasting, hypoalbuminemia, impaired collagen synthesis, and delayed wound healing collectively weaken the abdominal wall and predispose to hernia formation.[2]

Incisional hernias are a common consequence of abdominal surgery and develop in approximately 10–20% of patients following laparotomy.[3] In patients with cirrhosis, recurrence rates are considerably higher because of ongoing elevation in intra-abdominal pressure and poor tissue quality.

Although hernia incarceration and strangulation are well-recognized complications, spontaneous rupture with bowel evisceration is exceptionally uncommon. The condition is more frequently reported in umbilical hernias associated with cirrhosis and ascites and is often referred to as "Flood Syndrome."[4] Reports of spontaneous rupture involving incisional hernias are extremely rare.

We report a case of spontaneous rupture of a large incisional hernia with bowel evisceration in a patient with alcohol-related chronic liver disease and refractory ascites and review the existing literature.

II. CASE REPORT

A 47-year-old male presented to the emergency department with sudden protrusion of bowel loops and leakage of ascitic fluid through a defect in the anterior abdominal wall following a severe bout of coughing.

The patient was a known case of alcohol-related chronic liver disease with recurrent ascites for the preceding three years. He had undergone multiple therapeutic paracenteses for symptomatic relief. One year before presentation, he had undergone surgery for an obstructed umbilical hernia.

Six months after surgery, he developed a gradually enlarging swelling over the previous operative site. Initially reducible and painless, the swelling progressively increased in size. Subsequently, the overlying skin became thinned and ulcerated with intermittent foul-smelling discharge.

The patient was a chronic alcoholic and smoker. He also reported chronic constipation and recurrent episodes of cough, both of which likely contributed to repeated increases in intra-abdominal pressure.

Upon arrival, the patient was drowsy, hypotensive, and in shock. Blood pressure was below 90/60 mmHg, and oxygen supplementation was required to maintain adequate oxygen saturation.

Abdominal examination revealed a large anterior abdominal wall swelling with a 7 × 5 cm fascial defect. The overlying skin was necrotic and ulcerated. Small bowel loops were eviscerated through the defect and appeared mildly edematous but viable. Continuous leakage of ascitic fluid was noted.

Figure 1

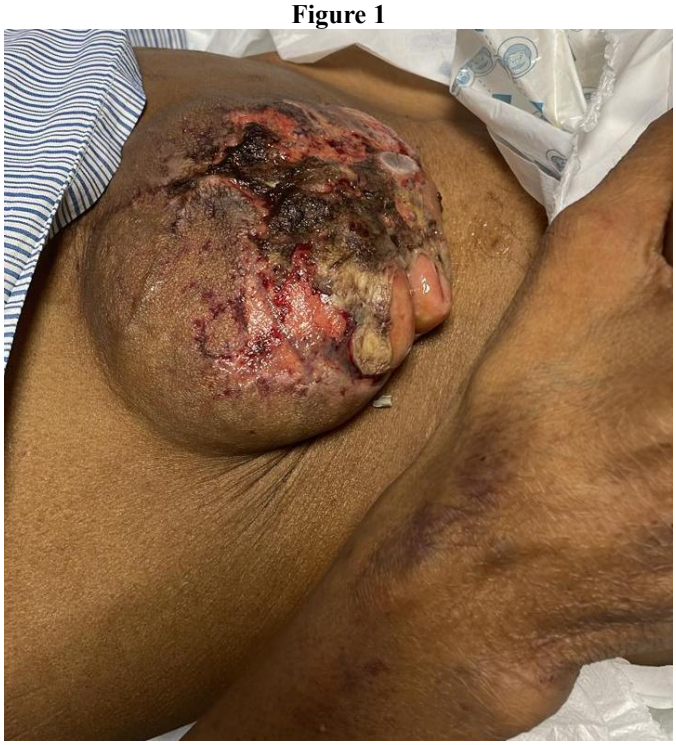

Figure 1: Clinical photograph demonstrating a large incisional hernia with overlying skin ulceration and necrosis prior to rupture. Areas of skin breakdown and impending evisceration are visible, representing warning signs of imminent catastrophic rupture.

Ultrasonography demonstrated cholelithiasis, mild splenomegaly, attenuated hepatic echotexture, and moderate ascites.

Laboratory investigations were consistent with chronic liver disease. The patient was categorized as Child-Pugh Class C with a MELD score of 10.

The patient was admitted to the intensive care unit and managed with aggressive fluid resuscitation, broad-spectrum antibiotics, intravenous albumin, correction of electrolyte abnormalities, and supportive care.

After thorough cleansing of the exposed bowel, local debridement was performed. The eviscerated bowel was reduced, and temporary closure was achieved using skin approximation and protective coverage. Gastroenterology consultation was obtained.

Medical management included:

- Intravenous albumin
- Broad-spectrum antibiotics
- Diuretics (spironolactone and furosemide)
- Lactulose

- Rifaximin
- Nutritional support
- Hepatic supportive therapy

Definitive abdominal wall reconstruction was deferred owing to severe hepatic dysfunction, refractory ascites, and high perioperative risk.

Despite maximal supportive treatment, the patient's clinical condition progressively deteriorated because of advanced liver disease and multiorgan dysfunction, ultimately resulting in death.

III. DISCUSSION

Abdominal wall hernias are frequently encountered in cirrhotic patients and constitute an important source of morbidity.

Several factors contribute to hernia formation in chronic liver disease:

- Persistent ascites
- Elevated intra-abdominal pressure
- Sarcopenia
- Malnutrition
- Hypoalbuminemia
- Impaired collagen metabolism
- Delayed wound healing

Patients with previous abdominal surgery are particularly vulnerable because surgical scars represent areas of reduced tensile strength.

Spontaneous rupture generally occurs after progressive thinning and ulceration of the overlying skin. Skin discoloration, ulceration, and necrosis should be regarded as signs of impending rupture and warrant urgent surgical evaluation.

The present patient possessed several recognized risk factors:

- Alcohol-related cirrhosis
- Refractory ascites
- Previous abdominal surgery
- Chronic cough
- Chronic constipation
- Malnutrition

Episodes of coughing or straining can abruptly increase intra-abdominal pressure and precipitate rupture.

The condition is associated with several potentially fatal complications:

- Bowel incarceration
- Strangulation
- Peritonitis
- Septic shock
- Massive fluid loss
- Electrolyte imbalance

Mortality rates of conservatively managed ruptured hernias have been reported to range from 30–60%.[5]

Historically, surgery in cirrhotic patients was avoided because of perceived operative risk. However, recent evidence suggests that elective repair in optimized patients is associated with significantly better outcomes than emergency surgery.

Marsman et al. demonstrated that emergency procedures in cirrhotic patients carry substantially greater morbidity and mortality compared with elective repair.[6]

Similarly, Carbonell et al. reported improved outcomes when surgical intervention was performed before complications such as incarceration or rupture developed.[7]

Emergency management requires:

1. Immediate resuscitation.
2. Protection of exposed bowel.
3. Prevention of infection.
4. Ascites control.
5. Nutritional optimization.
6. Definitive repair whenever physiologically feasible.

The present case highlights the devastating consequences of delayed presentation and advanced hepatic decompensation.

IV. REVIEW OF LITERATURE

Author	Year	Clinical Characteristics	Management	Outcome
Chatzizacharias et al.	2015	Cirrhosis with umbilical hernia rupture	TIPS followed by repair	Successful
D'Orazio et al.	2021	HCV cirrhosis with ruptured hernia	Fibrin glue and mesh repair	No recurrence
Srivastava et al.	2022	NASH cirrhosis with rupture	Mesh repair	Recovery
Leva et al.	2022	HCV cirrhosis with spontaneous rupture	Fascial repair	Recovery
Jiang et al.	2022	Alcoholic liver disease	Conservative treatment	Poor outcome
Present Case	2025	Alcoholic CLD, ascites, incisional hernia	Temporary closure	Mortality

Our case is unique because:

- Rupture occurred through an incisional rather than primary umbilical hernia.
- Extensive skin necrosis preceded rupture.
- Bowel evisceration occurred following coughing.
- Outcome was fatal despite aggressive management.

V. CONCLUSION

Spontaneous rupture of an incisional hernia in patients with chronic liver disease and ascites is a rare but potentially fatal surgical emergency. Progressive skin ulceration and necrosis should be recognized as warning signs of impending rupture. Early elective repair following optimization of liver function and ascites control may prevent catastrophic complications. Multidisciplinary management involving surgeons, intensivists, and hepatologists is essential to improve outcomes.

DECLARATIONS

Ethical Approval

Not applicable for a single case report according to institutional policy.

Consent

Written informed consent for publication of clinical details and photographs was obtained from the patient's legal representative.

Conflict of Interest

The authors declare no conflict of interest.

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