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Spontaneous intra-abdominal bleeding as the first sign of liver cirrhosis: a case report

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Abstract

Spontaneous intra-abdominal bleeding in cirrhotic patients is commonly the result of variceal rupture, usually in the gastrointestinal tract. Ectopic varices account for only 2-5% of variceal bleeding in the gastrointestinal tract, yet they carry a fourfold higher risk of hemorrhage compared with esophageal varices and can be associated with mortality rates of up to 40%. We present the case of a 51-year-old male with no past medical history of liver cirrhosis, who was admitted with hypotension and syncope. Imaging and laboratory findings were consistent with liver cirrhosis with ascites and splenomegaly. Exploratory laparotomy revealed massive hemoperitoneum originating from ectopic variceal bleeding. Surgical intervention and supportive management led to an uneventful recovery. This rare case underlines the importance of high clinical suspicion and early surgical intervention in spontaneous hemoperitoneum in cirrhotic patients.

Key words: intra-abdominal bleeding, liver cirrhosis, ectopic varices, spontaneous hemoperitoneum, case report.

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Introduction

Variceal bleeding is a well-established and often primary manifestation of decompensated hepatic cirrhosis. Typically, variceal rupture occurs in the esophageal or gastric regions, leading to gastrointestinal hemorrhage. However, large portosystemic venous collateral pathways can occur anywhere in the gastrointestinal tract. When these veins are located outside the gastroesophageal region, they are referred to as ectopic varices. If they rupture, they can cause spontaneous intra-abdominal bleeding.

Although rare, this form of bleeding may represent the first clinical indication of cirrhotic decompensation. Due to its small frequency and atypical presentation, spontaneous intra-abdominal hemorrhage is often under- or misdiagnosed, resulting in high morbidity and mortality. According to the literature, only a few cases have been reported. In this case, we present a patient with spontaneous hemoperitoneum from ectopic varices in the setting of previously undiagnosed liver cirrhosis. Our aim is to raise awareness of this potentially fatal condition and to highlight the key role of initial surgical intervention.

Case Report

A 51-year-old male with no significant past medical history presented to the emergency department following a syncopal

episode. On primary examination, he was found hypotensive (blood pressure [BP]: 80/40 mmHg, heart rate [HR]: 93 bpm), alert, but reporting increasing diffuse abdominal pain. Laboratory studies revealed mild anemia (red blood cells [RBC] $4 \times 10^6/\mu\text{L}$, hemoglobin [Hb] 11.9 g/dL, hematocrit [Ht] 35.6%), white blood cells (WBC) 10.77×10^3 cell/ μL , and mild thrombocytopenia (platelets $97 \times 10^9/\text{L}$). Despite initial aggressive fluid resuscitation with crystalloids, the patient's BP remained low, and abdominal pain intensified. A new laboratory study was obtained, showing a drop of Ht levels by 8%, suggesting active bleeding.

The patient was considered an emergency, and the attending surgeon decided not to proceed with an abdominal ultrasound but directly with a computed tomography (CT) scan of the chest and abdomen with intravenous contrast (Figure 1). The exam revealed moderate to large-volume ascites, increased size of the spleen (splenomegaly), liver of heterogeneous density with nodularity and lesions consistent with cirrhosis, and no evidence of blunt abdominal trauma or gastrointestinal bleeding. Further investigation of the ascitic fluid was performed via diagnostic paracentesis. The aspirated fluid was grossly bloody, a finding consistent with hemoperitoneum.

Considering the clinical and laboratory results, it was decided to proceed with an emergency exploratory laparotomy. Intraoperative findings included a large hemoperitoneum (of approximately 2.5 L), recent hemorrhagic changes with exten-

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sive hematomas in the gastrocolic and gastrosplenic ligaments, and no clear evidence of perforated viscus or other traumatic lesion. Following that, the gastrocolic and gastrosplenic ligaments were ligated and divided to control bleeding. No active variceal rupture was observed, but the location and the pattern of bleeding suggested rupture of ectopic varices or fragile collateral vessels. After hemostasis, hemodynamic stability was achieved. No tissue was obtained intraoperatively for histological examination.

The patient's postoperative course was uneventful, with no further bleeding, and he tolerated the recovery well. Liver function tests showed the following results: aspartate aminotransferase 82 U/L; alanine aminotransferase 56 U/L; albumin 2.5 g/dL; total bilirubin 2.8 mg/dL; alkaline phosphatase 185 U/L; creatinine 1.3 mg/dL; international normalized ratio (INR) 1.8; and lactic acid 2.1 mmol/L. The pattern of "elevated total bilirubin levels, low albumin levels, and INR prolongation" indicated decompensated cirrhosis with significant functional compromise (Child-Pugh B). He was discharged on post-operative day 10, with a close follow-up arranged with a hepatologist for long-term management of his liver disease.

Discussion

Spontaneous hemoperitoneum (or non-traumatic hemoperitoneum [NTH]), the presence of blood in the peritoneal cavity without a history of trauma or secondary to a procedure, is a rare but critical presentation of decompensated liver cirrhosis.¹ Table 1 shows the most frequent etiologies of NTH.² While esophageal and gastric varices are the most common source of intra-abdominal variceal bleeding (diagnosed and managed via established protocols), hemorrhage from ectopic varices remains diagnostically and therapeutically challenging.

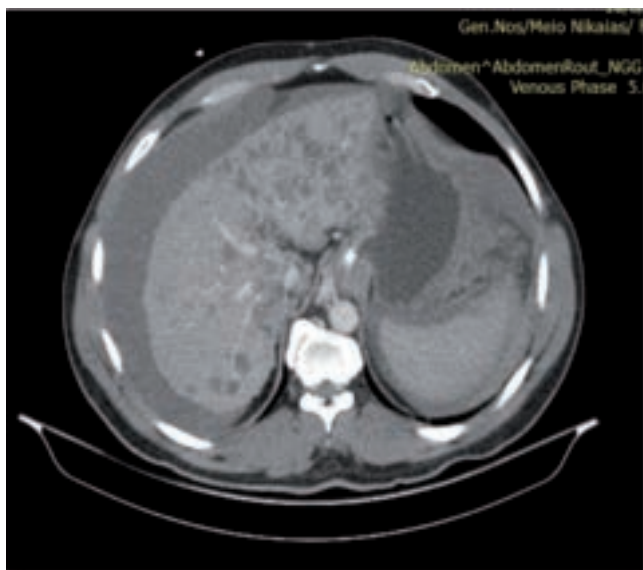


Figure 1. CT of the abdomen (axial) showing the presence of ascitic fluid, splenomegaly, and liver heterogeneity.

The formation of ectopic varices can be distinguished, according to their pathophysiology, into two categories: i) the non-occlusive type, caused by generalized portal hypertension, and ii) the occlusive type, due to splanchnic venous occlusion. More precisely, the occlusive type can be a result of postoperative adhesions, postoperative-altered anatomy (after gastrectomy, ileostomy, and post-balloon-occluded retrograde transvenous obliteration), splenoportal vein thrombosis, chronic pancreatitis, and mesenteric carcinoid.³

Diagnosis of intra-abdominal hemorrhage in cirrhotic patients is difficult due to its rarity and nonspecific presentation.⁴ Ascites may obscure physical findings, whilst hemoperitoneum may be mistaken for spontaneous bacterial peritonitis or other causes of cirrhotic decompensation. Clinical symptoms that must raise awareness include abdominal pain with sudden distension, signs of hypovolemic shock, and signs indicating retroperitoneal bleeding (e.g., Cullen's or Grey Turner's sign).⁵ The imaging diagnostic approach consists of a point-of-care ultrasound (US) as a first step, which can establish the presence of free intra-abdominal fluid or any parenchymal pathologies. A positive US in a cirrhotic patient must be followed by an ascitic-fluid aspiration to diagnose or exclude hemoperitoneum. A CT scan of the abdomen remains the best imaging choice, especially when treating unstable cirrhotic patients; intra-abdominal bleeding can be recognized easily, or other major pathologies (e.g., hepatocellular carcinoma, splenic artery aneurysm) can be excluded, but it is often difficult to pinpoint the exact source of bleeding.^{2,6}

Paracentesis revealing hemoperitoneum in cirrhotic patients, with the presence of hemodynamic instability, should prompt emergent laparotomy. In stable patients with an identified bleeding source preoperatively, interventional radiology can play a major role in diagnosis and treatment (e.g., angiography, embolization, transjugular or transhepatic decompression).⁷ However, rapid deterioration necessitates immediate surgical management. Several different surgical procedures have been described, although crucial points include peri-hepatic packing, venous or ligament ligation, and meticulous hemostasis.^{2,3} Tissue taken during the operation and sent for histological examination can provide significant evidence for the etiology of the hemoperitoneum source.

Despite the surgical outcomes, such patients remain at high risk due to their underlying liver dysfunction. It is critical for these patients to be referred to special hepatologists for close follow-up and prevention of future cirrhosis decompensation and further variceal bleeding.^{2,4,7}

This case highlights key clinical insights in the management of spontaneous hemoperitoneum in cirrhotic patients. Firstly, it demonstrates the timely recognition of the condition in the context of cirrhosis, which facilitated prompt diagnostic and therapeutic measures. Early diagnostic paracentesis also played a critical role in triggering immediate surgical intervention, contributing to a better clinical outcome through ligation of an otherwise unexplored bleeding source. Nonetheless, the case presents certain limitations. There was no access to pre-operative angiographic identification of the bleeding vessel, which could possibly have provided precise localization and planning. Furthermore, *in situ* confirmation of variceal rupture was not possible intraoperatively, leaving the exact etiology of the bleeding presumptive rather than definitive.

Table 1. Common etiologies of non-traumatic hemoperitoneum.

Etiology	Clinical presentation	Risk factors	Diagnostic considerations	Management strategies
Ruptured hepatocellular carcinoma	<ul style="list-style-type: none"> - Sudden onset of RUQ or diffuse abdominal pain - Abdominal distension - Hypotension - Signs of hemorrhagic shock - Acute anemia 	<ul style="list-style-type: none"> - Underlying cirrhosis - Viral hepatitis B or C - Portal hypertension - Coagulopathy 	<ul style="list-style-type: none"> - Contrast-enhanced CT demonstrating hemoperitoneum with ruptured hepatic mass - Falling hemoglobin levels - Elevated alpha-fetoprotein (supportive for the diagnosis) 	<ul style="list-style-type: none"> - Initial hemodynamic stabilization (fluid resuscitation-blood transfusion) - Transarterial embolization - Surgical management
Ruptured gastrointestinal varices	<ul style="list-style-type: none"> - Acute abdominal pain - Coexist with or follow gastrointestinal bleeding - Hypotension/tachycardia 	<ul style="list-style-type: none"> - Portal hypertension - Advanced liver cirrhosis - Portal vein thrombosis 	<ul style="list-style-type: none"> - Contrast-enhanced CT revealing hemoperitoneum with dilated variceal structures - Endoscopy for identification of possible luminal variceal source of bleeding (may not localize the intraperitoneal source) 	<ul style="list-style-type: none"> - Initial resuscitation (fluids - transfusion) - Vasoactive agents (e.g., octreotide) - Radiological interventions (e.g., TIPS or embolization) - Surgery only for refractory cases
Rupture of ectopic varices	<ul style="list-style-type: none"> - Sudden abdominal pain - Hypovolemic shock - Usually without overt gastrointestinal bleeding - Often unexpected and rapidly progressive 	<ul style="list-style-type: none"> - Portal hypertension - Previous abdominal surgery - Portal or splenic vein thrombosis 	<ul style="list-style-type: none"> - Contrast-enhanced CT revealing intraabdominal bleeding - Angiography identifying bleeding from ectopic varices (e.g., mesenteric, omental, retroperitoneal) - Diagnosis often delayed due to rarity 	<ul style="list-style-type: none"> - Hemodynamic stabilization - Endovascular embolization or TIPS (by interventional radiologist) - Surgical ligation (in cases unresponsive to endovascular therapy)
Post-procedural hemoperitoneum	<ul style="list-style-type: none"> - Abdominal pain and distension developing hours to days after invasive procedures - Anemia - Possible hypotension 	<ul style="list-style-type: none"> - Recent liver biopsy - Paracentesis - Endoscopic or laparoscopic interventions - Coagulopathy - Anticoagulant drugs 	<ul style="list-style-type: none"> - Serial hemoglobin measurements - Contrast-enhanced CT scan to confirm hemoperitoneum and identify source of active bleeding - Review of recent procedures 	<ul style="list-style-type: none"> - Observation (for stable patients) - Correction of coagulopathy - Blood transfusion - Interventional radiology or surgical exploration (for unstable patients-ongoing bleeding)
Rupture of splenic artery aneurysm	<ul style="list-style-type: none"> - Sudden severe abdominal or LUQ pain - Hypotension - Rapid progression to shock 	<ul style="list-style-type: none"> - Pregnancy - Portal hypertension - Liver transplantation - Atherosclerosis - Connective tissue disorders 	<ul style="list-style-type: none"> - Contrast-enhanced CT (and angiography protocol) showing hemoperitoneum and aneurysmal rupture - High suspicion index in pregnant and cirrhotic patients 	<ul style="list-style-type: none"> - Initial resuscitation (fluids-transfusion) - Endovascular embolization - Urgent surgical procedure or splenectomy (if unstable)
Miscellaneous	<ul style="list-style-type: none"> - Variable, ranging from mild abdominal pain to fulminant shock 	<ul style="list-style-type: none"> - Anticoagulation therapy - Hematologic disorders - Vascular malformations - Gynecologic conditions (e.g., ruptured ovarian cyst) - Inflammatory or neoplastic diseases 	<ul style="list-style-type: none"> - CT imaging - Laboratory evaluation for coagulopathy - Targeted investigations based on clinical suspicion 	<ul style="list-style-type: none"> - Cause-specific treatment - Supportive care - Reversal of anticoagulation (when indicated) - Surgical or interventional management for uncontrolled bleeding

RUQ, right upper quadrant; LUQ, left upper quadrant; CT, computed tomography; TIPS, transjugular intrahepatic portosystemic shunt.

Conclusions

Spontaneous hemoperitoneum due to rupture of ectopic varices is a rare but potentially lethal complication of portal hypertension. A high index of clinical suspicion is essential when evaluating cirrhotic patients presenting with hypotension, abdominal pain, ascites, and declining Ht levels. In such cases, early diagnostic paracentesis plays a critical role in guiding prompt and appropriate management. For unstable patients, surgical intervention with exploratory laparotomy

and possible variceal ligation remains the definitive measure for treatment. Long-term management of cirrhosis must not be underestimated, as it is vital to reducing the risk of future variceal hemorrhagic events.

References

1. Kasotakis G. Spontaneous hemoperitoneum. *Surg Clin North Am* 2014;94:65-9.

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2. Aseni P, Di Domenico SL, Barbosa F, et al. Hemoperitoneum in cirrhotic patients in the absence of abdominal trauma. *Expert Rev Gastroenterol Hepatol* 2019;13:867-76.
3. Saad WEA, Lippert A, Saad NE, Caldwell S. Ectopic Varices: Anatomical Classification, Hemodynamic Classification, and Hemodynamic-Based Management. *Tech Vasc Interv Radiol* 2013;16:108-25.
4. Ma YJ, Chen EQ, Lu JJ, et al. Hemoperitoneum in cirrhotic patients without abdominal trauma or tumor. *Hepatobiliary Pancreat Dis Int* 2011;10:644-8.
5. Aslam N, Waters B, Riely CA. Intraperitoneal rupture of ectopic varices: two case reports and a review of literature. *Am J Med Sci* 2008;335:160-2.
6. Lucey BC, Varghese JC, Anderson SW, Soto JA. Spontaneous hemoperitoneum: a bloody mess. *Emerg Radiol* 2007;14:65-75.
7. Lucey BC, Varghese JC, Soto JA. Spontaneous hemoperitoneum: causes and significance. *Curr Probl Diagn Radiol* 2005;34:182-95.

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