EXTENSIVE HEMORRHAGIC ASCITES AND PERITONEAL NECROSIS IN ACUTE PANCREATITIS: CASE PRESENTATION AND REVIEW OF LITERATURE

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CASE REPORT

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Abstract

Severe Acute Pancreatitis may rarely progress to catastrophic intra-abdominal complications including hemorrhagic ascites and diffuse peritoneal necrosis. These findings reflect advanced enzymatic tissue destruction, severe systemic inflammatory response, and high mortality risk. Emergency surgical intervention remains challenging, particularly in patients presenting with acute abdomen and rapid hemodynamic deterioration. We present the case of a patient admitted with severe acute pancreatitis complicated by extensive hemorrhagic ascites and diffuse peritoneal necrosis requiring emergency surgical management. Clinical presentation, laboratory findings, computed tomography imaging, intraoperative findings, surgical treatment, and postoperative evolution were analyzed. A focused literature review was additionally performed using PubMed/MEDLINE and Google Scholar databases regarding necrotizing pancreatitis associated with hemorrhagic ascites, pancreatic necrosis, vascular complications, abdominal compartment syndrome, and emergency surgical intervention. Computed tomography demonstrated severe acute pancreatitis with extensive intra-abdominal fluid collections and suspected pancreatic necrosis. Due to progressive clinical deterioration and signs of surgical abdomen, emergency exploratory laparotomy was performed. Intraoperatively, large-volume hemorrhagic ascites associated with extensive peri-pancreatic and diffuse peritoneal necrosis was identified. Surgical management included evacuation of hemorrhagic collections, necrosectomy, extensive abdominal lavage, and drainage. Postoperatively, the patient required intensive multidisciplinary support because of persistent systemic inflammatory response and severe metabolic imbalance. Hemorrhagic ascites associated with diffuse peritoneal necrosis represents a rare but severe manifestation of necrotizing acute pancreatitis. Early recognition, prompt multidisciplinary assessment and individualized surgical management are essential in critically ill patients presenting with advanced intr-abdominal complications.

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Introduction

Acute pancreatitis represents one of the most severe inflammatory conditions encountered in emergency digestive surgery, with clinical manifestations ranging from mild edematous disease to extensive pancreatic and extra-pancreatic necrosis associated with multiorgan dysfunction and high mortality rates [1–4]. Despite significant advances in intensive care management, imaging techniques, minimally invasive procedures, and multidisciplinary treatment strategies, severe acute pancreatitis continues to be associated with substantial morbidity and mortality, particularly in patients who develop infected necrosis, persistent organ failure, abdominal compartment syndrome, or hemorrhagic complications [5–7].

The pathophysiological substrate of severe necrotizing pancreatitis involves premature activation of pancreatic enzymes leading to pancreatic autodigestion, microvascular injury, extensive cytokine-mediated inflammatory response, and progressive tissue ischemia [8–10]. In advanced forms, the inflammatory process may extend beyond the pancreas, involving the retroperitoneum, mesentery, peritoneal surfaces, and vascular structures, resulting in extensive intra-abdominal necrosis and severe systemic deterioration [11,12]. Hemorrhagic ascites in acute pancreatitis is considered an uncommon but particularly severe manifestation, usually reflecting advanced enzymatic destruction, vascular erosion, increased capillary permeability, and extensive pancreatic necrosis [13,14]. Several studies have demonstrated that the presence of ascites in acute pancreatitis correlates with increased disease severity, persistent organ failure, infected necrosis, prolonged hospitalization, and increased mortality [13,15].

Diffuse peritoneal necrosis associated with hemorrhagic ascites remains exceptionally rare in contemporary surgical literature. Such cases may mimic mesenteric ischemia, hollow viscus perforation, diffuse peritonitis, or intra-abdominal malignancy, frequently leading to emergency exploratory surgery [16,17]. Furthermore, severe intra-abdominal inflammation and massive fluid collections may contribute to the development of intra-abdominal hypertension and abdominal compartment syndrome, further aggravating organ dysfunction and worsening prognosis [18,19].

Current international guidelines recommend conservative and minimally invasive approaches whenever feasible in necrotizing pancreatitis; however, emergency surgical intervention remains necessary in selected patients presenting with abdominal catastrophe, uncontrolled sepsis, abdominal compartment syndrome, bowel ischemia, or diagnostic uncertainty [2,3,5,20]. The management of extensive hemorrhagic ascites and diffuse peritoneal necrosis in severe acute pancreatitis therefore remains particularly challenging and insufficiently described in the literature.

The present paper describes a rare and severe case of acute necrotizing pancreatitis complicated by massive hemorrhagic ascites and diffuse peritoneal necrosis requiring emergency surgical management, alongside a focused review of the current literature regarding the pathophysiology, prognostic implications, and therapeutic strategies associated with these catastrophic intra-abdominal manifestations.

Case presentation

We present the case of a patient diagnosed with severe Acute Pancreatitis complicated by extensive hemorrhagic ascites and diffuse peritoneal necrosis, admitted to the Department of General Surgery, Emergency County Clinical Hospital Târgu Mureș, Romania. Clinical evaluation included detailed assessment of presenting symptoms, hemodynamic status, laboratory parameters,

inflammatory markers, and contrast-enhanced abdominal computed tomography findings.

The diagnosis of acute pancreatitis and disease severity assessment were established according to the Revised Atlanta Classification criteria [1]. Imaging evaluation focused on the presence and extent of pancreatic necrosis, intra-abdominal fluid collections, vascular involvement, and extra-pancreatic inflammatory extension. Due to progressive clinical deterioration, persistent abdominal pain, signs of generalized peritoneal irritation, and worsening systemic inflammatory response, emergency exploratory laparotomy was performed.

Intraoperative findings, surgical management strategy, postoperative intensive care evolution, and multidisciplinary therapeutic interventions were retrospectively analyzed from the patient's medical records, operative reports, laboratory investigations, and imaging studies. Surgical treatment included evacuation of hemorrhagic ascites, extensive abdominal lavage, necrosectomy, debridement of necrotic tissues, and placement of abdominal drainage systems according to intraoperative findings.

A focused narrative literature review was additionally conducted using PubMed/MEDLINE and Google Scholar electronic databases. The literature search included combinations of the following keywords: "acute pancreatitis," "necrotizing pancreatitis," "hemorrhagic ascites," "pancreatic ascites," "peritoneal necrosis," "abdominal compartment syndrome," "pancreatic necrosis," and "emergency surgery." Relevant English-language articles including clinical studies, guidelines, reviews, meta-analyses, and case reports addressing severe necrotizing pancreatitis and associated hemorrhagic intra-abdominal complications were selected and analyzed.

The present study was conducted in accordance with the principles of the Declaration of Helsinki and institutional ethical standards. Written informed consent for publication of clinical data and intraoperative images was obtained from the patient.

A 31-year-old female patient was transferred emergently from the Department of Gastroenterology with the diagnosis of severe Acute Pancreatitis occurring on a background of acute-on-chronic pancreatitis associated with compensated hypovolemic shock. Associated pathological findings included an immature pancreatic pseudocyst compressing the celiac trunk and spleno-portal axis, extensive omental necrosis, portal hypertension syndrome, extensive adhesion syndrome, massive hemorrhagic ascites, and diffuse peritoneal necrosis.

The patient presented with severe abdominal pain, marked abdominal distension, systemic inflammatory response syndrome, and progressive hemodynamic instability requiring repeated surgical reevaluation because of the dynamic clinical evolution. Imaging and multidisciplinary assessment demonstrated advanced necrotizing pancreatitis associated with severe hemorrhagic intra-abdominal complications and extensive inflammatory involvement.

Due to progressive deterioration and the presence of massive hemorrhagic ascites associated with diffuse peritoneal involvement, emergency exploratory laparotomy was performed. Intraoperative exploration revealed extensive adhesions involving the upper abdominal cavity together with large-volume hemorrhagic ascites and hemoperitoneum. Approximately 4000 mL of hemorrhagic fluid and blood were evacuated from the abdominal cavity (Figure 1).

Extensive areas of peritoneal necrosis and omental necrosis were identified intraoperatively, confirming severe extra-pancreatic extension of the necrotizing inflammatory process (Figure 2). The operative procedure included extensive adhesiolysis, evacuation of hemorrhagic ascites, peritoneal necrosectomy, resection of

necrotic parietal omentum, Kocher maneuver with duodeno-pancreatic mobilization, and left colo-parietal dissection. Intraoperative exploration additionally identified a retro-gastric immature pancreatic pseudocyst, which was evacuated and internally drained through the lesser curvature of the stomach to permit postoperative lavage. Extensive peritoneal lavage was subsequently performed, followed by placement of multiple drainage systems including retro-gastric pseudocyst drainage, left latero-colic drainage, Douglas pouch drainage, and double subhepatic drainage.

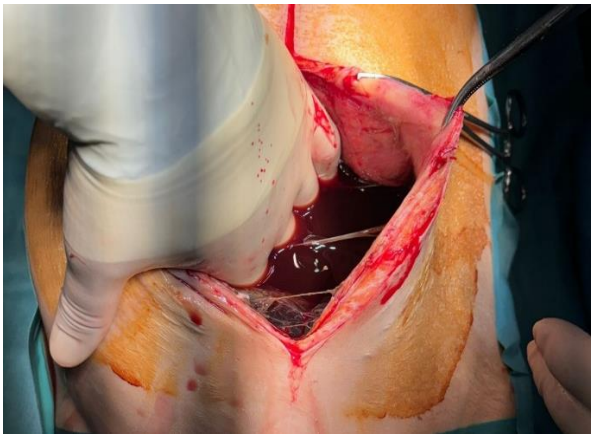

Figure 1. Massive hemorrhagic ascites identified immediately after emergency exploratory laparotomy in severe acute necrotic-hemorrhagic pancreatitis.

Postoperatively, the patient required prolonged intensive care treatment for approximately 30 days, including continuous multidisciplinary management and repeated peritoneal lavage over a two-week period. Despite the severity of the initial presentation and the extensive intra-abdominal necrotic involvement, postoperative evolution was favorable, without major postoperative surgical complications. Progressive clinical improvement and adequate control of abdominal drainage output were achieved during hospitalization. At discharge, one peripancreatic drain remained in place with an output of approximately 150 mL/24 h, subsequently removed two weeks later following readmission to the Municipal Hospital of Odorheiu Secuiesc.

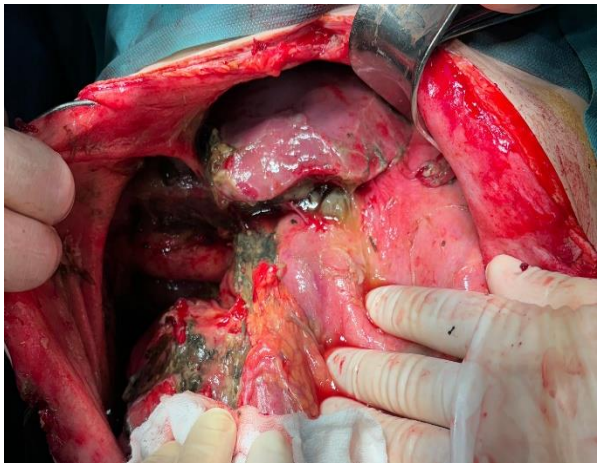

Figure 2. Intraoperative aspect demonstrating advanced peri-pancreatic and peritoneal necrotic involvement with extensive fibrino-necrotic inflammatory tissue destruction in severe necrotizing acute pancreatitis.

The focused literature review demonstrated that hemorrhagic complications occur in approximately 2% of acute pancreatitis cases, while hemoperitoneum has been reported in nearly 4% of patients with severe disease. The principal mechanisms implicated include vascular erosion secondary to activated pancreatic enzymes, pseudoaneurysm rupture, hemorrhage into necrotic pancreatic tissue or pseudocysts, and coagulation abnormalities. Current literature supports emergency surgical intervention in selected patients presenting with hemodynamic instability, massive intraperitoneal hemorrhage, abdominal compartment syndrome, failed endovascular management, or rapidly progressive necrotizing disease (Table 1).

Author	Year	Study Type	Main Hemorrhagic Complication	Surgical Treatment	Outcome
Barge et al. [19]	2012	Review	Vascular complications/pseudoaneurysm	Endovascular ± surgery	Variable
Mentula et al. [21]	2010	Surgical series	Abdominal compartment syndrome	Surgical decompression	Improved survival
van Santvoort et al. [6]	2010	RCT	Infected necrotizing pancreatitis	Step-up approach	Reduced morbidity
Mier et al. [15]	1997	Surgical study	Severe necrotizing pancreatitis	Early vs delayed necrosectomy	Better delayed outcomes
Bush & Rana [25]	2021	Review	Pancreatic ascites	Conservative/endoscopic	Variable
Rana et al. [27]	2018	Case series	Pancreatic ascites from duct disruption	Endoscopic drainage	Favorable
Present case	2026	Case report	Massive hemorrhagic ascites and diffuse peritoneal necrosis	Emergency laparotomy + necrosectomy	Favorable recovery

Table 1. Literature review of hemorrhagic complications in severe acute pancreatitis. RCT – randomized controlled study

Discussions

Severe Acute Pancreatitis remains one of the most complex and life-threatening conditions encountered in emergency abdominal surgery, particularly in patients who develop extensive pancreatic necrosis associated with hemorrhagic and extra-pancreatic complications [1–5]. The present case illustrates an exceptionally severe form of acute necrotic-hemorrhagic pancreatitis complicated by massive hemorrhagic ascites, diffuse peritoneal necrosis, omental necrosis, portal hypertension syndrome, and immature pancreatic pseudocyst formation requiring emergency surgical management. Such extensive intra-abdominal necrotic involvement is rarely described in contemporary literature and represents a major diagnostic, technical, and therapeutic challenge.

The pathophysiological mechanisms responsible for hemorrhagic complications in acute pancreatitis are multifactorial and include enzymatic vascular erosion, progressive inflammatory-mediated tissue destruction, microvascular ischemia, pseudoaneurysm formation, and hemorrhage into necrotic pancreatic or peri-pancreatic tissues [12,19]. Activated pancreatic enzymes, particularly elastase and phospholipase, contribute to progressive vascular wall injury and destruction of surrounding tissues, explaining the occurrence of hemorrhagic ascites and diffuse necrotic changes involving the peritoneum and omentum [12,13,19]. In the present case, the presence of approximately 4000 mL of hemorrhagic ascites associated with extensive peritoneal necrosis reflects an advanced stage of extra-pancreatic inflammatory extension and severe intra-abdominal enzymatic injury.

Hemorrhagic ascites is considered an uncommon but highly unfavorable finding in acute pancreatitis. Zerem demonstrated that the presence of ascites in severe pancreatitis is

strongly associated with pancreatic necrosis, persistent organ failure, prolonged hospitalization, infected collections, and increased mortality [24]. Similarly, Bush and Rana emphasized that pancreatic ascites and hemorrhagic intra-abdominal collections often indicate advanced pancreatic duct disruption, extensive necrosis, and severe inflammatory activity [25]. The present case supports these observations, as the patient developed massive hemorrhagic intraperitoneal collections associated with severe systemic inflammatory response and extensive abdominal involvement.

Diffuse peritoneal necrosis in acute pancreatitis remains exceptionally rare. Most reports available in the literature focus predominantly on retroperitoneal necrosis or infected pancreatic collections, while extensive necrotic involvement of the peritoneal surfaces is infrequently documented [4,13,31]. The severe inflammatory and enzymatic process observed in the current patient resulted not only in pancreatic necrosis but also in diffuse destruction of the omentum and peritoneal tissues, requiring extensive peritoneal necrosectomy and debridement. Furthermore, the extensive adhesion syndrome encountered intraoperatively likely reflected repeated inflammatory episodes related to the underlying acute-on-chronic pancreatitis.

Current international guidelines favor conservative and minimally invasive approaches whenever feasible in necrotizing pancreatitis [2,3,5,16]. The step-up approach described by van Santvoort et al. significantly reduced morbidity compared with primary open necrosectomy and remains the preferred strategy in many patients with infected pancreatic necrosis [6,16]. Nevertheless, emergency surgical intervention continues to play an essential role in selected critically ill patients presenting with abdominal catastrophe, uncontrolled hemorrhage, abdominal compartment syndrome, bowel ischemia, perforation, or diagnostic uncertainty [3,17,21]. In the present case, emergency exploratory laparotomy was justified by progressive hemodynamic deterioration, massive hemorrhagic ascites, diffuse peritoneal involvement, and extensive necrosis requiring immediate surgical source control.

Another important aspect of the present case was the coexistence of an immature pancreatic pseudocyst compressing the celiac trunk and spleno-portal axis. Vascular compression and vascular complications are well-recognized consequences of severe pancreatitis and may contribute to portal hypertension, venous thrombosis, ischemic complications, and progressive hemorrhage [19]. Surgical drainage of the retro-gastric pseudocyst through the lesser curvature of the stomach allowed both decompression and postoperative lavage, contributing to local control of the inflammatory process.

The prolonged postoperative intensive care management required in this case reflects the extreme severity of the disease. Patients with severe necrotizing pancreatitis frequently require multidisciplinary treatment involving general surgery, gastroenterology, intensive care, interventional radiology, and nutritional support teams [3–5]. Despite the catastrophic intraoperative findings, the patient demonstrated favorable postoperative evolution without major surgical complications, emphasizing the importance of aggressive multidisciplinary management and individualized operative decision-making.

The present report additionally highlights the rarity of diffuse hemorrhagic peritoneal necrosis associated with acute pancreatitis. Most available publications describe isolated hemorrhagic complications,

pseudoaneurysm rupture, pancreatic ascites, or infected necrosis, while reports describing extensive peritoneal necrosis remain extremely limited [19,24,25]. Consequently, the current case contributes valuable clinical and surgical information regarding the operative management of advanced necrotizing pancreatitis with catastrophic intra-abdominal extension.

From a surgical perspective, hemorrhagic pancreatitis remains both a technical and tactical challenge. Operative management must balance adequate source control and necrosectomy with minimization of additional tissue trauma in an already critically inflamed abdominal environment. Extensive lavage, drainage, staged management strategies, and prolonged postoperative monitoring remain fundamental components of treatment in such severe presentations [15,30–34].

To our knowledge, reports describing simultaneous massive hemorrhagic ascites and diffuse peritoneal necrosis requiring emergency necrosectomy in acute pancreatitis remain exceptionally limited in the current literature.

Conclusions

Massive hemorrhagic ascites associated with diffuse peritoneal necrosis represents an exceptionally rare and life-threatening manifestation of Acute Pancreatitis. The present case highlights the aggressive extra-pancreatic extension that may occur in advanced necrotizing disease and emphasizes the importance of early recognition, multidisciplinary evaluation, and individualized surgical management in critically ill patients. Emergency exploratory laparotomy, extensive necrosectomy, abdominal lavage, and prolonged intensive care support may be lifesaving in selected cases presenting with severe hemorrhagic and necrotic intra-abdominal complications.

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