



## Ascites in acute pancreatitis: A window into disease severity

Enver Zerem, Dina Zerem, Šeila Vila, Sanja Bajgorić

**Specialty type:** Gastroenterology and hepatology

**Provenance and peer review:** Invited article; Externally peer reviewed.

**Peer-review model:** Single blind

**Peer-review report's classification**

**Scientific Quality:** Grade B, Grade C

**Novelty:** Grade B, Grade C

**Creativity or Innovation:** Grade B, Grade C

**Scientific Significance:** Grade A, Grade C

**P-Reviewer:** Li F, MD, Assistant Professor, China; Yao Y, MD, China

**Received:** August 6, 2025

**Revised:** September 14, 2025

**Accepted:** October 17, 2025

**Published online:** November 21, 2025

**Processing time:** 106 Days and 16.2 Hours



**Enver Zerem,** Department of Medical Sciences, The Academy of Sciences and Arts of Bosnia and Herzegovina, Sarajevo 71000, Bosnia and Herzegovina

**Dina Zerem, Šeila Vila, Sanja Bajgorić,** Department of Health Studies, University Dzemal Bijedić Mostar, Mostar 88000, Bosnia and Herzegovina

**Co-first authors:** Enver Zerem and Dina Zerem.

**Corresponding author:** Enver Zerem, MD, Full Professor, Department of Medical Sciences, The Academy of Sciences and Arts of Bosnia and Herzegovina, Bistrik 7, Sarajevo 71000, Bosnia and Herzegovina. [zerem@anubih.ba](mailto:zerem@anubih.ba)

### Abstract

Acute pancreatitis (AP) is a complex and potentially life-threatening inflammatory condition with a highly variable clinical course, ranging from mild, self-limiting episodes to severe necrotizing forms. Among its common complications ascites has traditionally been viewed as a passive byproduct of peritoneal inflammation and enzymatic leakage with limited diagnostic or prognostic utility. However, growing evidence challenges this perspective, suggesting that ascitic fluid in AP represents a dynamic and clinically meaningful component of disease progression. In this editorial we reflected on the findings presented by Rao *et al*, who highlighted the diagnostic, prognostic, and therapeutic significance of ascitic fluid in AP. Easily accessible markers such as lactate dehydrogenase may provide early prognostic insight while emerging molecular biomarkers and cytokine profiles offer promise for more precise risk stratification and individualized therapy. We argue that the systematic evaluation of ascitic fluid should be integrated into the clinical management of moderate to severe AP. Incorporating ascites analysis into standard diagnostic protocols may enhance early risk assessment, inform therapeutic decisions, and ultimately improve patient outcomes. Ascitic fluid should be recognized as a clinically valuable marker and an important source of information in the evolving landscape of AP care.

**Key Words:** Acute pancreatitis; Ascites; Lactate dehydrogenase; Prognosis; Paracentesis; Pancreatic pseudocyst; Biomarkers; Severity assessment

©The Author(s) 2025. Published by Baishideng Publishing Group Inc. All rights reserved.

**Core Tip:** There is increasing evidence that ascites in acute pancreatitis is more than an epiphenomenon. It reflects disease severity, provides actionable clinical information on inflammatory burden, vascular permeability, and risk of complications, and may serve as a target for personalized therapeutic strategies. Its biochemical and cellular composition can further support early risk stratification and guide treatment decisions.

**Citation:** Zerem E, Zerem D, Vila Š, Bajgorić S. Ascites in acute pancreatitis: A window into disease severity. *World J Gastroenterol* 2025; 31(43): 112797

**URL:** <https://www.wjgnet.com/1007-9327/full/v31/i43/112797.htm>

**DOI:** <https://dx.doi.org/10.3748/wjg.v31.i43.112797>

## INTRODUCTION

In their recent article published in the *World Journal of Gastroenterology*, Rao *et al*[1] emphasized the prognostic value of ascitic fluid analysis in acute pancreatitis (AP), suggesting that early assessment may help identify patients at increased risk for severe complications and mortality[1]. While we agree with these conclusions, our clinical experience indicates that the implications of ascites in AP extend beyond prognostication, influencing disease course, severity stratification, and therapeutic strategy[2-4].

AP is one of the most common gastrointestinal emergencies and is characterized by a broad clinical spectrum ranging from mild, self-limiting inflammation to severe necrotizing disease with systemic complications. Current scoring systems (such as Ranson, APACHE II, Glasgow, Balthazar, BISAP, and others) offer valuable but often incomplete predictions of disease severity, especially in dynamic or emergent settings. Although advances in biomarkers and imaging modalities have enhanced early risk assessment, there remains a need for accessible, reliable, and clinically actionable indicators of disease progression[1-5].

Among emerging prognostic markers ascitic fluid has gained increasing recognition as both a radiological and biochemical marker of disease severity. Despite its omission from classification systems like the revised Atlanta Criteria, early ascites is consistently associated with pancreatic necrosis, infection, multiorgan failure, and adverse clinical outcomes[2,6-9]. Moreover, its biochemical profile including elevated amylase, proteins, and proinflammatory mediators may reflect both local and systemic inflammatory burden[8-12].

In our clinical practice we have observed that the systematic evaluation of ascites, particularly in the first 72 hours, provides actionable information that often precedes changes in conventional scoring systems. This editorial aimed to synthesize the existing evidence while also advocating for a more proactive approach to ascitic fluid analysis, framing it not just as a prognostic tool but as a guide for therapeutic strategy.

## PATHOPHYSIOLOGY AND PROGNOSTIC SIGNIFICANCE OF ASCITES IN AP

Ascitic fluid accumulation in AP reflects both localized pancreatic damage and systemic inflammatory activation. Although not currently included in standardized scoring systems for disease severity, the presence of ascites is increasingly recognized as a marker of more severe disease and a potential contributor to clinical deterioration[6,10-13].

The development of ascitic fluid in AP is multifactorial. A central mechanism involves increased vascular permeability, triggered by intense systemic inflammation and pancreatic tissue autodigestion. Premature activation of digestive enzymes (such as trypsin, phospholipase A2, and elastase) leads to disruption of acinar cells and surrounding peripancreatic tissues, promoting the leakage of enzyme-rich exudate into the peritoneal cavity. Simultaneously, elevated levels of proinflammatory cytokines, including interleukin (IL)-1 $\beta$ , IL-6, and tumor necrosis factor-alpha (TNF- $\alpha$ ) increase endothelial permeability and exacerbate fluid extravasation[11-15]. Structural complications, such as rupture of pancreatic pseudocysts (PPC) or pancreatic duct disruption may also result in persistent or high-volume ascites, particularly in the later stages of disease[4,16-18]. It is also important to note that the etiology of AP may influence the characteristics of ascites. For example, bile-stained ascites is more common in biliary pancreatitis, hemorrhagic ascites in alcoholic pancreatitis, and large-volume sterile effusions in hypertriglyceridemic AP. However, most published studies have not stratified outcomes by AP subtype, highlighting an area for future research[18-20].

In hemorrhagic forms of AP, vascular necrosis or erosion may result in blood-stained ascites, which are often associated with greater disease severity and higher mortality[19]. Biochemically, the ascitic fluid is typically exudative and is characterized by high protein content, elevated concentrations of pancreatic enzymes (particularly amylase) and variable concentrations of inflammatory mediators.

Recent clinical studies have demonstrated a strong correlation between the presence and volume of ascitic fluid detected on contrast-enhanced CT (CECT) and adverse outcomes, including pancreatic necrosis, persistent organ failure, and mortality[20,21]. According to several studies, ascites has emerged as an independent predictor of mortality and is linked with longer hospital stays, a greater need for intensive care, and a more complicated clinical course[20-23]. These findings suggest that the identification of ascites early in the course of AP could enhance prognostic assessment and help guide clinical management.

Given the accumulating evidence, ascites in AP should not be considered a benign or incidental finding. Instead, it reflects active disease progression and may provide valuable information for early therapeutic decisions, including the need for intensified monitoring, more targeted fluid resuscitation, or the application of additional measures in line with the “step-up approach” for managing severe AP[2,3,21-23].

## DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS OF ASCITES IN AP

The presence of ascites in AP although not pathognomonic carries substantial diagnostic and therapeutic implications. It serves as both a radiological and biochemical marker, aiding in the assessment of disease severity and informing clinical management strategies.

### Diagnostic implications

From a diagnostic standpoint ascites is most commonly detected using CECT, which remains the imaging modality of choice in moderate to severe AP. The early identification of peritoneal fluid has been consistently associated with higher CT severity index scores, an increased risk of pancreatic necrosis, and a greater likelihood of persistent organ failure as reported in several studies[24-26]. Importantly, ascites may be visualized during the early interstitial phase of AP, offering prognostic insights before the full clinical picture develops.

Although routine analysis of ascitic fluid is not indicated in all patients, diagnostic paracentesis may be valuable in selected cases especially when infection, hemorrhage, or alternative intra-abdominal pathology is suspected. Biochemical analysis of pancreatic ascites typically reveals elevated amylase and lipase levels, consistent with pancreatic origin. Increased protein concentration and elevated inflammatory mediators such as IL-6 and TNF- $\alpha$  may further reflect the intensity of systemic inflammation[1-3,27,28]. In rare cases ascitic fluid analysis can help differentiate pancreatic ascites from other etiologies, such as cirrhosis or malignancy, especially in patients with overlapping conditions. Moreover, the emergence of ascites in the context of clinical deterioration or signs of abdominal compartment syndrome (ACS) should prompt urgent re-evaluation, including advanced imaging, surgical consultation, and intensified hemodynamic monitoring[18-20].

### Therapeutic implications

While the management of ascites in AP is primarily supportive, its identification can significantly influence the therapeutic approach. Standard care includes aggressive intravenous fluid resuscitation, nutritional support, and close monitoring of organ function. However, in patients with massive ascites, refractory intra-abdominal hypertension, or evidence of secondary infection, therapeutic paracentesis or image-guided percutaneous catheter drainage may be warranted[2,3].

Such interventions can relieve elevated intra-abdominal pressure, improve respiratory mechanics, and reduce the risk of developing ACS. Additionally, serial drainage of enzyme-rich ascitic fluid may help mitigate the peritoneal inflammatory response although robust data from randomized studies remain limited[2,3,19]. In specific cases involving pancreatic duct disruption such as pancreatic ascites secondary to necrosis-related leakage endoscopic retrograde cholangiopancreatography with pancreatic duct stenting may be beneficial in controlling the source of leakage[16,18].

Moreover, based on our experience, we believe that early diagnostic paracentesis should be considered more frequently in patients with moderate to severe AP and significant ascites rather than being reserved for cases where infection is already suspected. This proactive approach can yield early biomarker data [*e.g.*, lactate dehydrogenase (LDH), IL-6] that may signal impending deterioration and allow for pre-emptive escalation of care within a step-up approach.

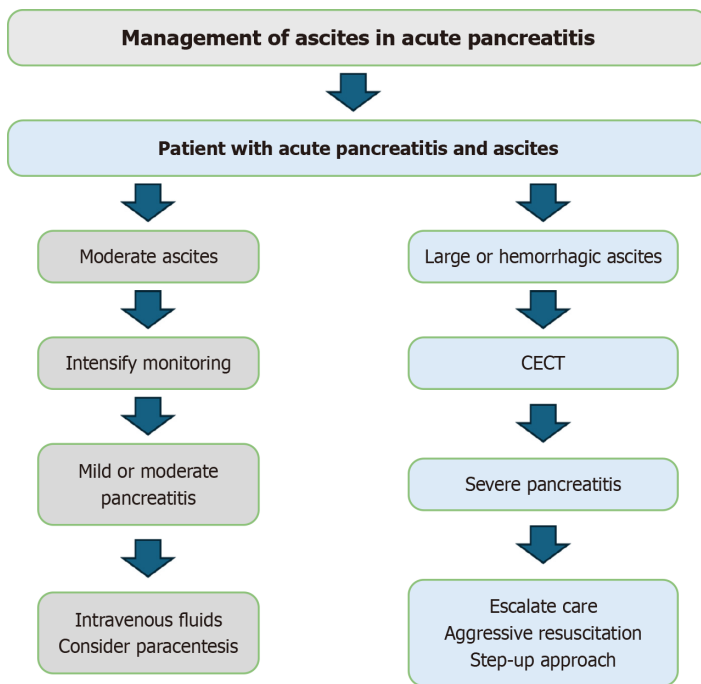
Ultimately, the detection of ascites in the context of AP should prompt increased clinical vigilance, more frequent reassessment of intravascular volume status, and where necessary early multidisciplinary intervention. Timely recognition and appropriate management of ascites may not only aid in early risk stratification but also improve outcomes in patients with severe forms of AP (Figure 1).

## ABDOMINAL PARACENTESIS: A BRIDGE BETWEEN DIAGNOSIS AND INTERVENTION IN ASCITIC AP

Abdominal paracentesis occupies an important role in the multidisciplinary management of AP complicated by ascites, offering both diagnostic clarity and therapeutic benefit. While not routinely performed in all cases, its selective use (particularly in patients with tense ascites, clinical signs of intra-abdominal hypertension, or ACS) can significantly influence decision-making and improve clinical outcomes[2,29-31].

### Diagnostic utility

In patients with new-onset or worsening ascites during the course of AP, paracentesis enables differentiation between sterile inflammatory effusions, infected ascitic collections, hemorrhagic fluid, and pancreatic ductal leaks each associated with specific prognostic and therapeutic implications. Biochemical analysis of the ascitic fluid often reveals elevated amylase levels, high protein content, and neutrophilic predominance in the context of pancreatic leaks or enzymatic peritonitis, which typically respond to conservative management. In contrast, the detection of bacteria on Gram stain or positive ascitic fluid cultures indicates secondary infection, necessitating the initiation of targeted antibiotic therapy and possibly additional interventional or surgical procedures[1-3,29].



**Figure 1 Management of ascites in acute pancreatitis.** The algorithm starts with early identification of ascites (usually by contrast-enhanced CT). If ascites is small and the patient is stable, supportive treatment and monitoring are sufficient. In cases of tense or hemorrhagic ascites or when abdominal compartment syndrome is suspected, diagnostic and therapeutic paracentesis is indicated. If persistent high-volume output, infected fluid, or ductal disruption is confirmed, escalation to percutaneous catheter drainage or endoscopic therapy (endoscopic retrograde cholangiopancreatography with stenting) is recommended. This step-up approach allows timely intervention while avoiding unnecessary invasive procedures. CECT: Contrast-enhanced CT.

### Therapeutic role

Beyond its diagnostic value paracentesis may serve as an initial therapeutic maneuver. Drainage of even a moderate volume of ascitic fluid can relieve intra-abdominal pressure, restore perfusion to vital organs, and improve respiratory parameters often producing immediate though temporary clinical improvement. This is particularly important in patients who are hemodynamically unstable and at risk of developing ACS[2,3,17,18,29].

Serial or repeated paracenteses can assist in monitoring disease progression and the effectiveness of conservative therapy. Persistent high-volume, enzyme-rich ascitic output or the emergence of encapsulated collections with enhancing walls may indicate the evolution toward walled-off pancreatic necrosis (WOPN), (formerly referred to as pancreatic abscess) or infected PPC. Such findings often prompt escalation to image-guided percutaneous catheter drainage or other definitive interventions. Thus, paracentesis functions as a diagnostic-therapeutic bridge within a step-up strategy, allowing for escalation of care only when clinically indicated, thereby avoiding unnecessary or premature procedures[32-36].

### Safety and practical considerations

When performed under ultrasound guidance, abdominal paracentesis is generally safe, minimally invasive, and cost-effective. Nonetheless, potential complications such as hemorrhage, secondary infection, or bowel injury underline the importance of proper technique, anatomical knowledge, and judicious patient selection[2].

In the context of AP with ascites, abdominal paracentesis emerges as a valuable tool that bridges diagnosis and intervention. It facilitates early biochemical and microbiological assessment, provides therapeutic decompression, and serves as a gateway to timely escalation within a step-up management framework. When applied selectively and appropriately, paracentesis may reduce morbidity and contribute to improved clinical outcomes.

## RELATIONSHIP BETWEEN PANCREATIC ASCITES AND THE FORMATION OF PPC OR WOPN

Pancreatic ascites arises from the leakage of pancreatic enzymes into the peritoneal cavity, most commonly due to disruption of the main pancreatic duct or its side branches or rupture of a peripancreatic fluid collection. Although relatively uncommon, it is a clinically significant manifestation of severe AP and is closely associated with the formation of PPC or WOPN, especially in the later stages of the disease.

The transition from enzyme-rich ascitic fluid to PPC formation follows a pathophysiological sequence involving persistent leakage of active pancreatic enzymes, primarily amylase and lipase, into the retroperitoneal or peritoneal space. This leakage results from ductal injury or necrosis and if not spontaneously resolved or externally drained may trigger the body's attempt to contain the fluid through the development of a fibrous capsule, leading to PPC or WOPN formation after approximately 4 weeks[32-35,37].

Clinically, ongoing or recurrent pancreatic ascites is a strong predictor of PPC formation, especially in patients with disconnected pancreatic duct syndrome or confirmed main pancreatic duct disruption. Imaging modalities such as magnetic resonance cholangiopancreatography and endoscopic retrograde cholangiopancreatography are instrumental in identifying the site and extent of ductal disruption and visualizing communication between the ductal system and the peritoneal cavity[18,32-34].

Several studies have demonstrated that patients with pancreatic ascites have a heightened risk of developing large and symptomatic PPC, many of which require interventional management. The likelihood of PPC or WOPN formation correlates with both the volume and persistence of ascitic fluid, especially in cases with unresolved inflammation or inadequately managed ductal injury during the early stages of the disease[2,18,33-35].

From a clinical management perspective, the early identification of pancreatic ascites should prompt detailed evaluation for possible ductal disruption and close radiological monitoring, especially beyond the third or fourth week of illness. Timely intervention ranging from conservative management to endoscopic stenting may prevent the progression from free enzyme leakage to encapsulated PPC formation[4,18].

## ASCITES IN AP: FROM PASSIVE EFFUSION TO PROGNOSTIC BIOMARKER

Ascitic fluid is frequently encountered in patients with moderate to severe AP. Once considered a passive byproduct of pancreatic enzyme leakage, vascular hyperpermeability, and peritoneal irritation, ascites is now increasingly recognized as both a marker and a potential driver of disease progression and systemic deterioration[2,3,38-40]. Its formation is primarily due to enzyme-rich leakage from inflamed or necrotic pancreatic tissue, accompanied by widespread inflammatory activation.

The volume and biochemical composition of ascitic fluid may reflect the extent of inflammation, particularly in necrotizing AP or cases involving pancreatic ductal disruption[11,13,18,40,41]. While elevated pancreatic enzymes are a hallmark of pancreatic ascites, their diagnostic value extends beyond enzymatic activity. Paracentesis provides access to ascitic fluid, allowing assessment of diverse cellular and biochemical markers that inform disease severity, infectious status, and clinical course.

Several ascitic parameters have been linked with poor outcomes in AP. Elevated neutrophil counts and high LDH levels indicate intense systemic inflammation, correlate with organ dysfunction, and are often associated with prolonged hospitalization. Similarly, early radiological detection of ascites (within 72 h of symptom onset) correlates with persistent organ failure, intensive care unit admission, and increased mortality, independent of traditional severity scores[1,23-25].

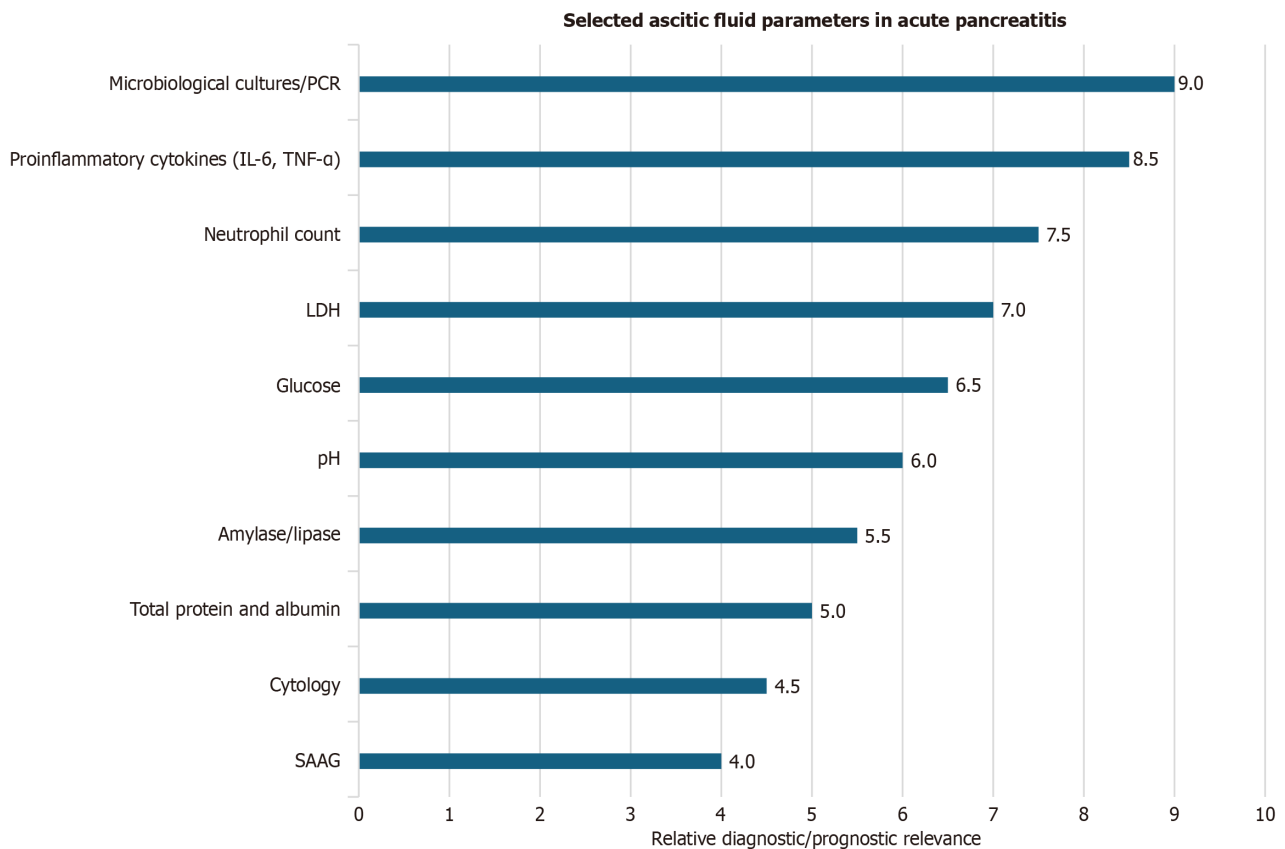
Among biochemical markers LDH remains the most widely utilized. As a cytosolic enzyme released during cell injury, it reflects ongoing tissue damage, inflammation, and microvascular compromise. In AP elevated ascitic LDH has been associated with pancreatic necrosis, systemic inflammatory response syndrome, multiorgan dysfunction, and extended hospitalization[2,42-45]. Its broad availability, low cost, and rapid turnaround make it particularly valuable for early risk stratification and escalation of care. Moreover, LDH may help distinguish between sterile and infected ascites an important clinical distinction guiding antimicrobial therapy and invasive interventions. When interpreted alongside findings such as neutrophilic predominance or low glucose levels, elevated LDH may suggest infection or peritonitis-like evolution, especially in patients exhibiting sepsis[18,45-47].

Despite its practicality, LDH is not among the most specific biomarkers for complications in AP-associated ascites. Proinflammatory cytokines such as IL-6 and TNF- $\alpha$  provide greater specificity for predicting infected necrosis and organ failure, but their clinical adoption is limited by cost, longer turnaround times, and restricted availability. Recent studies suggest that combining markers (for example, LDH with neutrophil count or IL-6) may further enhance prognostic accuracy compared with any single parameter. Thus, while LDH remains the most feasible biomarker in routine practice, cytokine-based assays may provide incremental value in selected patients or specialized centers[42-45,48-50]. Selected ascitic fluid parameters with potential diagnostic and prognostic value in AP are summarized in **Figure 2**.

Key ascitic fluid parameters with recognized diagnostic and prognostic utility include: (1) Bacterial cultures/PCR. Critical for detecting superinfection or infected necrosis; (2) Proinflammatory cytokines (*e.g.*, IL-6, TNF- $\alpha$ ). Emerging biomarkers for disease severity and systemic involvement; (3) Neutrophil count. Reflects the degree of local and systemic inflammation; (4) LDH. Distinguishes between sterile and infected ascites; (5) Glucose. Decreased levels may suggest infection or immune activation; (6) Potential of hydrogen. Acidosis may indicate infection or severe inflammation; (7) Amylase/Lipase. Indicative of pancreatic duct leakage or fistula; (8) Total protein and albumin. Characterize fluid as exudative and indicate vascular permeability; (9) Serum. Ascites albumin gradient-differentiates portal hypertension from pancreatic ascites; and (10) Cytology. Assists in excluding malignancy or detecting reactive changes. When interpreted in context and combined with early imaging (particularly CECT within 72 h) these parameters can significantly improve risk stratification, support prompt therapeutic decisions, and reduce delays in care[1,18-20,50].

A major unresolved gap in the current literature is the lack of large, prospective studies that establish and validate threshold values for ascitic fluid biomarkers (*e.g.*, LDH, IL-6) in predicting clinically relevant outcomes such as infected necrosis or organ failure. Future investigations should prioritize the determination of these cutoff points and their integration into multimodal prognostic models to improve risk stratification and guide evidence-based clinical decision-making.

In addition, evidence from clinical practice suggests that the ascitic fluid LDH-to-serum amylase ratio, a simple and readily obtainable parameter, warrants systematic evaluation due to its potential to differentiate sterile from infected collections at an earlier stage than conventional clinical signs. The timely recognition of such prognostic indicators could refine diagnostic accuracy and facilitate earlier therapeutic interventions, thereby improving patient outcomes.



**Figure 2 Selected ascitic fluid parameters with potential diagnostic and prognostic value in acute pancreatitis.** Microbiological cultures/PCR are critical for detecting superinfection or infected necrosis. Proinflammatory cytokines (interleukin-6, tumor necrosis factor- $\alpha$ ) are specific indicators of systemic inflammation and risk of organ failure though less available and more costly. Neutrophil count reflects inflammatory burden and is useful in resource-limited settings. Lactate dehydrogenase is inexpensive and widely available but relatively nonspecific. Glucose and pH at reduced levels suggest infection. Amylase/Lipase support the diagnosis of pancreatic ascites due to ductal disruption. Total protein and albumin characterize the exudative nature and indicate vascular permeability. Serum-ascites albumin gradient differentiates pancreatic ascites from portal hypertension. Cytology excludes malignancy. When interpreted together, combinations such as LDH plus neutrophil count may increase predictive accuracy. IL-6: Interleukin-6; TNF: Tumor necrosis factor; LDH: Lactate dehydrogenase; SAAG: Serum-ascites albumin gradient.

Beyond their diagnostic role, large or persistent ascitic collections may actively worsen outcomes by raising intra-abdominal pressure, contributing to ACS, and impairing both perfusion and ventilation[18-20,48]. Serial imaging studies have shown that unresolved ascites often precedes complications such as infected necrosis, PPC, or persistent ductal leakage[2,36,37,51,52].

Altogether, these observations reinforce that ascitic fluid in AP is not merely a passive effusion but a dynamic and pathophysiologically active component of disease progression. Easily accessible markers such as LDH provide practical value in early prognostication while advanced biomarkers including cytokines and molecular diagnostics may enhance specificity and therapeutic precision. Incorporating ascitic fluid analysis into routine clinical evaluation may substantially improve the accuracy and timeliness of AP management.

## CONCLUSION

Ascitic fluid in AP should be regarded not only as a byproduct of pancreatic injury but also as a clinically meaningful indicator of disease severity. Its presence warrants heightened clinical vigilance, timely reassessment, and when indicated escalation of care. Future prospective studies are essential to validate the prognostic accuracy of ascitic fluid biomarkers, to establish clinically applicable cut-off values, and to integrate fluid analysis into multimodal prognostic frameworks. Importantly, such studies should also evaluate the prognostic relevance of ascites across different AP subtypes as pathophysiological mechanisms and fluid characteristics may vary by etiology. A clear research agenda should include large multicenter trials, development of a standardized ascitic fluid risk score, and systematic evaluation of early, protocol-driven paracentesis to guide fluid management and antibiotic use. Although ascitic fluid analysis is not yet routine, its potential to enhance early prognostication and support personalized management in AP is substantial, warranting inclusion in future research protocols and guidelines.

## FOOTNOTES

**Author contributions:** Zerem E contributed to the conception and design of the paper, writing of the paper, and final revision; Vila Š, Bajgorić S and Zerem D contributed to the literature search, writing of the paper, and final revision of the paper.

**Conflict-of-interest statement:** The authors declare that they have no conflicts of interest.

**Open Access:** This article is an open-access article that was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution NonCommercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <https://creativecommons.org/licenses/by-nc/4.0/>

**Country of origin:** Bosnia and Herzegovina

**ORCID number:** Enver Zerem 0000-0001-6906-3630; Dina Zerem 0000-0003-0347-9881; Šeila Vila 0009-0004-8511-1929; Sanja Bajgorić 0009-0006-6077-0287.

**S-Editor:** Fan M

**L-Editor:** Filipodia

**P-Editor:** Lei YY

## REFERENCES

- Rao JW, Li JR, Wu Y, Lai TM, Zhou ZG, Gong Y, Xia Y, Luo LY, Xia L, Cai WH, Huang W, Zhu Y, He WH. Ascites characteristics in acute pancreatitis: A prognostic indicator of organ failure and mortality. *World J Gastroenterol* 2025; **31**: 108926 [RCA] [PMID: 40741476 DOI: 10.3748/wjg.v31.i28.108926] [FullText] [Full Text(PDF)]
- Zerem E, Kunosić S, Zerem D, Boloban A, Zerem O, Zlomužica E. Benefits of abdominal paracentesis drainage performed ahead of percutaneous catheter drainage as a modification of the step-up approach in acute pancreatitis with fluid collections. *Acta Gastroenterol Belg* 2020; **83**: 285-293 [RCA] [PMID: 32603048] [FullText]
- Zerem E, Imamović G, Latić F, Mavija Z. Prognostic value of acute fluid collections diagnosed by ultrasound in the early assessment of severity of acute pancreatitis. *J Clin Ultrasound* 2013; **41**: 203-209 [RCA] [PMID: 22987623 DOI: 10.1002/jcu.21995] [FullText]
- Zerem E, Imamović G, Omerović S, Ljuca F, Haracić B. Percutaneous treatment for symptomatic pancreatic pseudocysts: Long-term results in a single center. *Eur J Intern Med* 2010; **21**: 393-397 [RCA] [PMID: 20816592 DOI: 10.1016/j.ejim.2010.06.015] [FullText]
- He WH, Zhu Y, Zhu Y, Jin Q, Xu HR, Xion ZJ, Yu M, Xia L, Liu P, Lu NH. Comparison of multifactor scoring systems and single serum markers for the early prediction of the severity of acute pancreatitis. *J Gastroenterol Hepatol* 2017; **32**: 1895-1901 [RCA] [PMID: 28419583 DOI: 10.1111/jgh.13803] [FullText]
- Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS; Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; **62**: 102-111 [RCA] [PMID: 23100216 DOI: 10.1136/gutjnl-2012-302779] [FullText]
- Lee PJ, Papachristou GI. New insights into acute pancreatitis. *Nat Rev Gastroenterol Hepatol* 2019; **16**: 479-496 [RCA] [PMID: 31138897 DOI: 10.1038/s41575-019-0158-2] [FullText]
- Song Z, Zhu Q, Zhang Y, Yan X, Pan X. Ascites Volume Quantification via Abdominal CT: A Novel Approach to Predict Severity in Acute Pancreatitis. *Med Sci Monit* 2023; **29**: e940783 [RCA] [PMID: 37545114 DOI: 10.12659/MSM.940783] [FullText]
- Bush N, Rana SS. Ascites in Acute Pancreatitis: Clinical Implications and Management. *Dig Dis Sci* 2022; **67**: 1987-1993 [RCA] [PMID: 34036465 DOI: 10.1007/s10620-021-07063-6] [FullText]
- Metri A, Bush N, Singh VK. Predicting the severity of acute pancreatitis: Current approaches and future directions. *Surg Open Sci* 2024; **19**: 109-117 [RCA] [PMID: 38650599 DOI: 10.1016/j.sopen.2024.03.012] [FullText] [Full Text(PDF)]
- Zeng QX, Wu ZH, Huang DL, Huang YS, Zhong HJ. Association Between Ascites and Clinical Findings in Patients with Acute Pancreatitis: A Retrospective Study. *Med Sci Monit* 2021; **27**: e933196 [RCA] [PMID: 34737257 DOI: 10.12659/MSM.933196] [FullText] [Full Text(PDF)]
- Yadav D, O'Connell M, Papachristou GI. Natural history following the first attack of acute pancreatitis. *Am J Gastroenterol* 2012; **107**: 1096-1103 [RCA] [PMID: 22613906 DOI: 10.1038/ajg.2012.126] [FullText]
- Wang GJ, Gao CF, Wei D, Wang C, Ding SQ. Acute pancreatitis: etiology and common pathogenesis. *World J Gastroenterol* 2009; **15**: 1427-1430 [RCA] [PMID: 19322914 DOI: 10.3748/wjg.15.1427] [FullText] [Full Text(PDF)]
- Hu JX, Zhao CF, Wang SL, Tu XY, Huang WB, Chen JN, Xie Y, Chen CR. Acute pancreatitis: A review of diagnosis, severity prediction and prognosis assessment from imaging technology, scoring system and artificial intelligence. *World J Gastroenterol* 2023; **29**: 5268-5291 [RCA] [PMID: 37899784 DOI: 10.3748/wjg.v29.i37.5268] [FullText] [Full Text(PDF)]
- Yao J, Zhang S, Zhou F, Zhuang M, Fei S. The relationship between inflammatory cytokines and in-hospital complications of acute pancreatitis. *Immun Inflamm Dis* 2024; **12**: e1203 [RCA] [PMID: 38411379 DOI: 10.1002/iid3.1203] [FullText]
- Kanneganti K, Srikanthapudi S, Acharya B, Sindhaghata V, Chilimuri S. Successful Management of Pancreatic Ascites with both Conservative Management and Pancreatic Duct Stenting. *Gastroenterology Res* 2009; **2**: 245-247 [RCA] [PMID: 27942284 DOI: 10.4021/gr2009.08.1306] [FullText] [Full Text(PDF)]
- Venkatesh V, Lal SB, Rana SS, Anushree N, Aneja A, Seetharaman K, Saxena A. Pancreatic ascites and Pleural Effusion in Children: Clinical Profile, Management and Outcomes. *Pancreatolgy* 2021; **21**: 98-102 [RCA] [PMID: 33349510 DOI: 10.1016/j.pan.2020.12.010] [FullText]
- Bathobakae L, Farhan H, Mücahit D, Rohira D, Chauhan K, Cavanagh Y, Baddoura W, Christian DJ. Pancreatic ascites: update on diagnosis and management. *Ann Gastroenterol* 2025; **38**: 247-254 [RCA] [PMID: 40371206 DOI: 10.20524/aog.2025.0961] [FullText] [Full Text(PDF)]
- Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain ML, De Keulenaer B, Duchesne J, Bjorck M, Leppaniemi A, Ejike JC,

- Sugrue M, Cheatham M, Ivatury R, Ball CG, Reintam Blaser A, Regli A, Balogh ZJ, D'Amours S, Debergh D, Kaplan M, Kimball E, Olvera C; Pediatric Guidelines Sub-Committee for the World Society of the Abdominal Compartment Syndrome. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. *Intensive Care Med* 2013; **39**: 1190-1206 [RCA] [PMID: 23673399 DOI: 10.1007/s00134-013-2906-z] [FullText] [Full Text(PDF)]
- 20 **Yusufi FK**, Zaka-Ur-Rab A, Siddiqi SS, Siddiqui KR, Kolari A, Yusufi HK. Role of Scoring Systems in Prognosticating Outcomes of Patients With Acute Pancreatitis: A Prospective Cohort Study. *Cureus* 2025; **17**: e79738 [RCA] [PMID: 40161108 DOI: 10.7759/cureus.79738] [Full Text]
- 21 **Mikó A**, Vigh É, Mátrai P, Soós A, Garami A, Balaskó M, Czakó L, Mosdósi B, Sarlós P, Eröss B, Tenk J, Rostás I, Hegyi P. Computed Tomography Severity Index vs. Other Indices in the Prediction of Severity and Mortality in Acute Pancreatitis: A Predictive Accuracy Meta-analysis. *Front Physiol* 2019; **10**: 1002 [RCA] [PMID: 31507427 DOI: 10.3389/fphys.2019.01002] [FullText] [Full Text(PDF)]
- 22 **Tyberg A**, Karia K, Gabr M, Desai A, Doshi R, Gaidhane M, Sharaiha RZ, Kahaleh M. Management of pancreatic fluid collections: A comprehensive review of the literature. *World J Gastroenterol* 2016; **22**: 2256-2270 [RCA] [PMID: 26900288 DOI: 10.3748/wjg.v22.i7.2256] [FullText] [Full Text(PDF)]
- 23 **Yang E**, Nguyen NH, Kwong WT. Abdominal free fluid in acute pancreatitis predicts necrotizing pancreatitis and organ failure. *Ann Gastroenterol* 2021; **34**: 872-878 [RCA] [PMID: 34815654 DOI: 10.20524/aog.2021.0666] [FullText] [Full Text(PDF)]
- 24 **Qi M**, Lu C, Dai R, Zhang J, Hu H, Shan X. Prediction of acute pancreatitis severity based on early CT radiomics. *BMC Med Imaging* 2024; **24**: 321 [RCA] [PMID: 39604925 DOI: 10.1186/s12880-024-01509-9] [FullText]
- 25 **Cheng T**, Han TY, Liu BF, Pan P, Lai Q, Yu H, Cao Y. Use of Modified Balthazar Grades for the Early Prediction of Acute Pancreatitis Severity in the Emergency Department. *Int J Gen Med* 2022; **15**: 1111-1119 [RCA] [PMID: 35153503 DOI: 10.2147/IJGM.S350383] [Full Text] [Full Text(PDF)]
- 26 **Balthazar EJ**, Ranson JH, Naidich DP, Megibow AJ, Caccavale R, Cooper MM. Acute pancreatitis: prognostic value of CT. *Radiology* 1985; **156**: 767-772 [RCA] [PMID: 4023241 DOI: 10.1148/radiology.156.3.4023241] [FullText]
- 27 **Mititelu A**, Grama A, Colceriu MC, Pop TL. Overview of the cellular and immune mechanisms involved in acute pancreatitis: In search of new prognosis biomarkers. *Expert Rev Mol Med* 2025; **27**: e9 [RCA] [PMID: 39757373 DOI: 10.1017/erm.2024.40] [FullText] [Full Text (PDF)]
- 28 **Wu H**, Liao B, Ji T, Huang J, Ma K, Luo Y. Diagnostic value of CRP for predicting the severity of acute pancreatitis: a systematic review and meta-analysis. *Biomarkers* 2024; **29**: 494-503 [RCA] [PMID: 39417604 DOI: 10.1080/1354750X.2024.2415463] [FullText]
- 29 **van Santvoort HC**, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH, van Goor H, Schaapherder AF, van Eijck CH, Bollen TL, van Ramshorst B, Nieuwenhuijs VB, Timmer R, Laméris JS, Kruyt PM, Manusama ER, van der Harst E, van der Schelling GP, Karsten T, Hesselink EJ, van Laarhoven CJ, Rosman C, Bosscha K, de Wit RJ, Houdijk AP, van Leeuwen MS, Buskens E, Gooszen HG; Dutch Pancreatitis Study Group. A step-up approach or open necrosectomy for necrotizing pancreatitis. *N Engl J Med* 2010; **362**: 1491-1502 [RCA] [PMID: 20410514 DOI: 10.1056/NEJMoa0908821] [FullText]
- 30 **Basha J**, Maharshi TS, Lakhtakia S. Endoscopic Step-Up Approach in Management of Necrotizing Pancreatitis. *Gastroenterol Clin North Am* 2025; **54**: 37-51 [RCA] [PMID: 39880532 DOI: 10.1016/j.gtc.2024.10.002] [FullText]
- 31 **IAP/APA/EPC/IPC/JPS Working Group**. International Association of Pancreatology Revised Guidelines on Acute Pancreatitis 2025: Supported and Endorsed by the American Pancreatic Association, European Pancreatic Club, Indian Pancreas Club, and Japan Pancreas Society. *Pancreatology* 2025; **25**: 770-814 [RCA] [PMID: 40651900 DOI: 10.1016/j.pan.2025.04.020] [FullText]
- 32 **Binda C**, Perini B, Coluccio C, Giuffrida P, Fabbri S, Gibiino G, Vizzuso A, Giampalma E, Fabbri C. Metal stent and percutaneous endoscopic necrosectomy as dual approach for the management of complex walled-off pancreatic necrosis. *Minerva Surg* 2024; **79**: 183-196 [RCA] [PMID: 38127434 DOI: 10.23736/S2724-5691.23.10132-8] [FullText]
- 33 **Verma S**, Rana SS. Disconnected pancreatic duct syndrome: Updated review on clinical implications and management. *Pancreatology* 2020; **20**: 1035-1044 [RCA] [PMID: 32800651 DOI: 10.1016/j.pan.2020.07.402] [FullText]
- 34 **Bang JY**, Wilcox CM, Navaneethan U, Hasan MK, Peter S, Christein J, Hawes R, Varadarajulu S. Impact of Disconnected Pancreatic Duct Syndrome on the Endoscopic Management of Pancreatic Fluid Collections. *Ann Surg* 2018; **267**: 561-568 [RCA] [PMID: 27849658 DOI: 10.1097/SLA.0000000000002082] [FullText]
- 35 **Zerem E**, Pavlović-Čalić N, Sušić A, Haračić B. Percutaneous management of pancreatic abscesses: long term results in a single center. *Eur J Intern Med* 2011; **22**: e50-e54 [RCA] [PMID: 21925043 DOI: 10.1016/j.ejim.2011.01.015] [FullText]
- 36 **Huang J**, Li L, Chen Y, Mao E, Qu H. Early short-term abdominal paracentesis drainage in moderately severe and severe acute pancreatitis with pelvic ascites. *BMC Surg* 2023; **23**: 363 [RCA] [PMID: 38012699 DOI: 10.1186/s12893-023-02269-z] [FullText]
- 37 **Takenaka M**, Saito T, Hamada T, Omoto S, Shiomi H, Iwashita T, Masuda A, Matsubara S, Maruta A, Iwata K, Mukai T, Isayama H, Yasuda I, Nakai Y. Disconnected pancreatic duct syndrome: diagnostic and therapeutic challenges and future directions. *Expert Rev Gastroenterol Hepatol* 2024; **18**: 631-645 [RCA] [PMID: 39420546 DOI: 10.1080/17474124.2024.2419056] [FullText]
- 38 **Beger HG**, Rau BM. Severe acute pancreatitis: Clinical course and management. *World J Gastroenterol* 2007; **13**: 5043-5051 [RCA] [PMID: 17876868 DOI: 10.3748/wjg.v13.i38.5043] [FullText] [Full Text(PDF)]
- 39 **Wu BU**, Johannes RS, Sun X, Tabak Y, Conwell DL, Banks PA. The early prediction of mortality in acute pancreatitis: a large population-based study. *Gut* 2008; **57**: 1698-1703 [RCA] [PMID: 18519429 DOI: 10.1136/gut.2008.152702] [FullText]
- 40 **Mofidi R**, Duff MD, Wigmore SJ, Madhavan KK, Garden OJ, Parks RW. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. *Br J Surg* 2006; **93**: 738-744 [RCA] [PMID: 16671062 DOI: 10.1002/bjs.5290] [Full Text]
- 41 **Schepers NJ**, Bakker OJ, Besselink MG, Ahmed Ali U, Bollen TL, Gooszen HG, van Santvoort HC, Bruno MJ; Dutch Pancreatitis Study Group. Impact of characteristics of organ failure and infected necrosis on mortality in necrotizing pancreatitis. *Gut* 2019; **68**: 1044-1051 [RCA] [PMID: 29950344 DOI: 10.1136/gutjnl-2017-314657] [FullText]
- 42 **Nieminen A**, Maksimov M, Mentula P, Kyhälä L, Kylänpää L, Puolakkainen P, Kempainen E, Repo H, Salmi M. Circulating cytokines in predicting development of severe acute pancreatitis. *Crit Care* 2014; **18**: R104 [RCA] [PMID: 24886762 DOI: 10.1186/cc13885] [FullText] [Full Text(PDF)]
- 43 **Samanta J**, Rana A, Dhaka N, Agarwala R, Gupta P, Sinha SK, Gupta V, Yadav TD, Kochhar R. Ascites in acute pancreatitis: not a silent bystander. *Pancreatology* 2019; **19**: 646-652 [RCA] [PMID: 31301995 DOI: 10.1016/j.pan.2019.06.004] [FullText]

- 44 **Leppäniemi A**, Tolonen M, Tarasconi A, Segovia-Lohse H, Gamberini E, Kirkpatrick AW, Ball CG, Parry N, Sartelli M, Wolbrink D, van Goor H, Baiocchi G, Ansaloni L, Biffl W, Coccolini F, Di Saverio S, Kluger Y, Moore E, Catena F. 2019 WSES guidelines for the management of severe acute pancreatitis. *World J Emerg Surg* 2019; **14**: 27 [RCA] [PMID: 31210778 DOI: 10.1186/s13017-019-0247-0] [FullText] [FullText(PDF)]
- 45 **Bender F**, König T, Hecker M, Fritzenwanker M, Braun J, Pons-Kühnemann J, Wolff M, Hecker A, Reichert M. Impact of invasive infections on clinical outcomes in acute pancreatitis: early predictive factors and implications for prophylactic anti-infective therapy. *Gut Pathog* 2025; **17**: 5 [RCA] [PMID: 39828733 DOI: 10.1186/s13099-024-00671-3] [FullText]
- 46 **Silva-Vaz P**, Abrantes AM, Castelo-Branco M, Gouveia A, Botelho MF, Tralhão JG. Multifactorial Scores and Biomarkers of Prognosis of Acute Pancreatitis: Applications to Research and Practice. *Int J Mol Sci* 2020; **21**: 338 [RCA] [PMID: 31947993 DOI: 10.3390/ijms21010338] [FullText] [FullText(PDF)]
- 47 **Cui J**, Xiong J, Zhang Y, Peng T, Huang M, Lin Y, Guo Y, Wu H, Wang C. Serum lactate dehydrogenase is predictive of persistent organ failure in acute pancreatitis. *J Crit Care* 2017; **41**: 161-165 [RCA] [PMID: 28554094 DOI: 10.1016/j.jcrc.2017.05.001] [FullText]
- 48 **Tian F**, Li H, Wang L, Li B, Aibibula M, Zhao H, Feng N, Lv J, Zhang G, Ma X. The diagnostic value of serum C-reactive protein, procalcitonin, interleukin-6 and lactate dehydrogenase in patients with severe acute pancreatitis. *Clin Chim Acta* 2020; **510**: 665-670 [RCA] [PMID: 32828732 DOI: 10.1016/j.cca.2020.08.029] [FullText]
- 49 **Stutee I**, Midha NK, Chaudhary M, Kumar D, Banerjee M, Garg P, Soni S, Garg MK. Role of Inflammatory Markers and Radiological Profile in Predicting Acute Pancreatitis Severity: A Prospective Analysis. *Cureus* 2025; **17**: e89033 [RCA] [PMID: 40895844 DOI: 10.7759/cureus.89033] [FullText] [FullText(PDF)]
- 50 **Xinyu X**, Jiang Z, Qing A, Lihua L, Xiehong L, Lin Z. Clinical significance of PCT, CRP, IL-6, NLR, and TyG Index in early diagnosis and severity assessment of acute pancreatitis: A retrospective analysis. *Sci Rep* 2025; **15**: 2924 [RCA] [PMID: 39849025 DOI: 10.1038/s41598-025-86664-x] [FullText] [FullText(PDF)]
- 51 **Yuan H**, Liang W, You S, Zhou J, Chen S, Tan X, Luo Q, Ma Y, Lu L, Luo Z, Wang J. Analysis of the Application of Laparoscopic Peritoneal Lavage and Drainage in the Early Treatment of Severe Acute Pancreatitis. *Pancreas* 2025; **54**: e596-e603 [RCA] [PMID: 39933051 DOI: 10.1097/MPA.0000000000002478] [FullText]
- 52 **Al-Bahrani AZ**, Abid GH, Holt A, McCloy RF, Benson J, Eddleston J, Ammori BJ. Clinical relevance of intra-abdominal hypertension in patients with severe acute pancreatitis. *Pancreas* 2008; **36**: 39-43 [RCA] [PMID: 18192879 DOI: 10.1097/mpa.0b013e318149f5bf] [FullText]



Published by **Baishideng Publishing Group Inc**  
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA  
**Telephone:** +1-925-3991568  
**E-mail:** [office@baishideng.com](mailto:office@baishideng.com)  
**Help Desk:** <https://www.f6publishing.com/helpdesk>  
<https://www.wjgnet.com>

