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Acute pancreatitis in the critical care setting: A review of assessment and intervention strategies

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ABSTRACT

The incidence of acute pancreatitis (AP), a condition characterized by inflammation in the pancreas, has been increasing globally and is associated with several complications. This review elaborated on the etiology, clinical presentation, severity assessment, and treatment modalities of AP, mainly in the critical care setting. Patients with severe AP, as indicated by organ failure (>48 hours from onset), warrant treatment in the intensive care unit setting. The most common etiologies, biliary disease and alcohol consumption, and the advanced diagnostic tools used for the identification of the cause are highlighted. Different severity assessment tools are utilized for grading the severity of the disease, predicting patient outcomes, determining the associated risk, and guiding treatment decisions. The treatment interventions comprise various approaches, such as anti-infective therapy enteral nutrition, analgesics for pain, or minimally invasive surgical procedures, thereby demonstrating an evolving landscape of AP management. Furthermore, various complications such as necrosis, organ failure, and hemorrhage, necessitate disease monitoring and differential diagnosis and are crucial for optimal management of patients. Novel treatment modalities and advancements in multidisciplinary care emphasize the potential for reducing the burden of AP in critical care settings.

KEYWORDS: Acute pancreatitis; Critical care; Intensive care unit; Necrotizing pancreatitis; Organ failure

1. Introduction

Acute pancreatitis (AP) is a gastrointestinal condition characterized

by inflammation of the pancreas. This condition is triggered when pancreatic enzymes start to digest the pancreas, causing pancreatic tissue injury and damage[1,2]. AP imposes substantial physical, emotional, and financial burdens on patients and the healthcare system globally[3,4]. Between 1990 and 2019, the global incidence of AP surged by 62.9%, escalating from 1 727 789.3 to 2 814 972.3 cases[4]. While the mild form of the disease is associated with low mortality[5], the severe form of AP is more concerning, with a mortality rate of approximately 30%, necessitating acute hospitalization[5,6].

Patients with severe AP (SAP) are recommended for intensive care unit (ICU) admission and specialist centers for interventions by multiple guidelines[7]. SAP-associated mortality can occur in two instances: early mortality, typically within the first two weeks of hospital admission, often resulting from systemic inflammatory response syndrome (SIRS); and late mortality, which takes place after 14 days of the evolution, possibly due to complications such as infected necrosis[5]. Additionally, 25% of the patients diagnosed with AP may progress to multiple organ dysfunction syndrome or experience abdominal pain[8], thus necessitating admission to critical

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care[9]. These patients may also have comorbidities such as renal, cardiac, lung diseases, heart failure, connective tissue disorders, or cancer, thereby elevating the risk of mortality[9].

The primary etiologies of AP include biliary disease and alcohol consumption, while hypertriglyceridemia, trauma, infection, drugs, genetics, and autoimmune are less common[8,10]. Therefore, for the identification of the etiology of the disease, an initial examination comprising tests for bilirubin, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transferase, and triglycerides are vital. Additionally, in case of diagnostic ambiguity, an early abdominal computed tomography scan may be required for the patients admitted to the ICU. Monitoring of intra-abdominal pressure is also crucial in severe pancreatitis cases as it correlates to increased mortality[11]. Furthermore, various clinical scores are used to determine the severity of AP, although they may have limited clinical application[2,12]. Treatment for these patients may span over several weeks, ranging from antibiotics, analgesics, and nutritional supplementations to minor surgical interventions. Notably, in pediatric cases, patients with both mild AP and SAP are admitted to the pediatric ICU for comprehensive care[13].

This review aims to provide an in-depth overview of AP in the critical care setting by highlighting the etiologies of this disease, its clinical presentation, and initial management in the ICU setting. Further, the assessment of AP based on various severity scoring indices will be described, followed by the complications and differential diagnosis of AP. Moreover, this review will emphasize the novel treatment modalities for AP while focusing on the need for multidisciplinary care for patients with AP, thus aiming to provide insight into new treatment strategies that enhance patient outcomes and improve overall care.

2. Clinical presentation and etiology of AP

Patients with AP typically experience the abdominal region, particularly in the epigastric region or left upper quadrant. A confirmed diagnosis of AP involves meeting at least two of these three criteria, which include severe epigastric or upper abdominal pain, enhanced serum amylase and/or lipase levels (three times the upper limit of normal), or AP-correlated findings in the abdominal imaging (CT, magnetic resonance imaging)[14].

Various etiologies exist for AP, the most common one being gallstone pancreatitis, affecting 40% to 70% of patients. This acute event often necessitates a cholecystectomy to prevent recurrent attacks or the possibility of biliary sepsis. Alcohol-induced pancreatitis is considered the next most frequently observed etiology found in 25% to 35% of patients, usually emerging after >5 years of heavy alcohol consumption (>50 g/day)[3,15]. Hypertriglyceridemia, with serum triglyceride levels exceeding 1000 mg/dL, is also another notable cause for the manifestation of AP. Drug-mediated

AP is also observed in patients as a result of taking medications such as azathioprine, DDI (2', 3' - dideoxyinosine), or 6- mercaptopurine. In cases where the identification of the exact cause of AP is unclear, based on one factor[16], advanced diagnostic tools like contrast-enhanced CT scans, endoscopic ultrasound, or magnetic resonance imaging are employed for further investigation[3]. Failure to establish the etiology based on the initial laboratory and imaging tests further gives rise to idiopathic AP, where a definite cause cannot be determined. Such patients may need a referral to specialized centers for advanced diagnostic measures and a multidisciplinary approach[3].

As per the 2013 Atlanta Revision, organ failure in AP that resolves within 48 hours is categorized as moderate SAP. SAP requires critical care and is characterized by persistent organ failure lasting >48 hours and can lead to death[3,12], potentially involving a single organ or multiple organs[14]. In addition, the emergence of fluid collection or infected necrosis further increases the risk of mortality. In interstitial edematous pancreatitis, an acute peripancreatic fluid collection may develop in the early phase of pancreatitis, which may progress to form a pancreatic pseudocyst if it persists beyond 4 weeks. Alternatively, if a collection forms in the pancreas comprising fluid and necrotic tissue, it is termed an acute necrotic collection, indicative of pancreatic parenchymal necrosis. Another type of collection, termed walled-off necrosis, is a collection of pancreatic or peripancreatic necrosis with a defined wall, usually seen in patients after ≥4 weeks of the onset of necrotizing pancreatitis[14].

3. Severity assessment in SAP

Various severity assessment scoring indices are applied in clinical practice to identify patients at high risk of developing SAP (Table 1)[8]. The Modified Marshall scoring system, comprising the respiratory, cardiovascular, and renal systems, is utilized to determine organ failure in SAP. Further, several risk profiling scores are employed in SAP, such as the RANSON score, Acute Physiology and Chronic Health Evaluation II (APACHE-II) score, Sequential Organ Failure Assessment (SOFA) score, and BISAP score. The RANSON criteria evaluate 11 parameters [age, white blood cell count (WBC), blood glucose, serum aspartate transaminase, serum lactate dehydrogenase (LDH), serum calcium, fall in hematocrit, arterial oxygen (PaO₂), blood urea nitrogen (BUN), base deficit, and sequestration of fluids] for scoring alcoholic pancreatitis, while, the modified RANSON score possess 10 parameters for scoring gallbladder pancreatitis[17]. Notably, hematocrit has been underscored as a key predictor of AP severity[18].

The APACHE-II scoring system is also widely used, where a score of ≥13 indicates a greater likelihood of death, and a score of ≤9 within the first 48 hours suggests high chances of survival[19]. However, the APACHE score of ≥9 has been considered an accurate

Table 1. Severity scoring tools utilized in the assessment of acute pancreatitis.

Severity assessment tool	Parameters	Condition	Scoring	References
Ranson criteria	Age, WBC count, blood glucose, serum AST, serum LDH, serum calcium, fall in hematocrit, PaO ₂ , BUN, base deficit, and sequestration of fluids	Alcoholic pancreatitis	Score of 0 to 2: Mortality 0% to 3%; 3 to 4: Mortality 15%; 5 to 6: Mortality 40%; 7 to 11: Nearly 100% mortality	[17]
Modified Ranson criteria	Age, WBC count, blood glucose, serum AST, serum LDH, serum calcium, fall in hematocrit, arterial oxygen PaO ₂ , BUN, base deficit, and sequestration of fluids	Gallstone pancreatitis	Score of 0 to 2: Mortality 0% to 3%; 3 to 4: Mortality 15%; 5 to 6: Mortality 40%; 7 to 11: Nearly 100% mortality	[17]
Glasgow criteria	Age, WBC count, calcium, urea, serum LDH, albumin, glucose, BUN	Stratification of severity of AP	Score ≥ 3 indicates SAP in the initial 48 hours of hospitalization	[35]
BISAP	BUN >25 mg/dL, mental status of patient (Glasgow coma scale score), SIRS, age >60-year, pleural effusion (on CT scan/chest X-ray/USG)	Prediction of high risk and mortality in AP	A score of 0-5; BISAP scores of ≥ 3 will indicate early organ failure or pancreatic necrosis	[36]
APACHE II	Body temperature, mean arterial pressure, heart rate, respiratory rate, oxygenation, pH, Na, K, creatinine, hematocrit, total leucocyte count, and the Glasgow coma score	Monitoring the patients' response to therapy on admission	A score ≥ 10 predicts a complicated outcome	[37]
PASS	Organ failure (Modified Marshall Score >2), SIRS, intravenous morphine equivalents, abdominal pain (0-10 scale), tolerating solid diet (no=1, yes=0)	Prediction of moderately severe or SAP	>145 Score predicts the development of moderately severe and severe AP and associated adverse clinical outcomes	[38]
SOFA	PaO ₂ /FiO ₂ ratio, Glasgow coma scale, mean arterial pressure or administration of vasopressors, bilirubin levels, and platelet levels	Organ failure	Graded from 0 (normal) to 4 (severe)	[21]
MMSS	Organ system; PaO ₂ / FiO ₂ ratio; renal (serum creatinine in mg/dL); cardiovascular (SBP in mmHG)	Organ failure	Score of ≥ 2 for more than 48 hours was considered persistent organ failure, whereas a score of ≥ 2 for less than 48 hours was considered transient organ failure	[39][40]

BISAP: Bedside Index for Severity in Acute Pancreatitis; APACHE II : Acute Physiology and Chronic Health Evaluation II ; PASS: Pancreatitis Activity Index Scoring System; SOFA: Sequential organ failure assessment; MMSS: Modified Marshall Scoring System; AP: acute pancreatitis; AST: aspartate transaminase; BUN: blood urea nitrogen; CT: computed tomography; LDH: lactate dehydrogenase; SIRS: systemic inflammatory response syndrome; SAP: severe acute pancreatitis; SBP: systemic blood pressure; USG: ultrasonography; WBC: white blood cell.

marker for AP severity in another study, highlighting different interpretations based on APACHE II scores[20]. Moreover, the SOFA score has been claimed to be effective in accurately stratifying disease severity and appropriately predicting ICU admission and mortality in AP patients. The grading of the disease depends on scores ranging between 0 to 4, based on markers PaO₂/FiO₂ ratio, Glasgow coma scale, mean arterial pressure or administration of vasopressors, bilirubin levels, and platelet levels[21,22].

According to Sundararajan *et al.*, the Bedside Index for Severity in Acute Pancreatitis (BISAP) score is effective in identifying ICU patients with SAP who are at greater risk of mortality[8]. This score

evaluates five variables (BUN >25 mg/dL; impaired mental status; SIRS; age >60 years, and presence of pleural effusion) within 24 hours of hospitalization to predict the in-hospital mortality[8]. The Pancreatitis Activity Scoring System (PASS), utilized for predicting the severity of AP, considers multiple factors such as organ failure, intolerance to a solid diet, pain, SIRS, and morphine equivalent dose by relevant weight[2]. Moreover, a US-based retrospective study reported the development of a new prediction model, ABL (age, BUN, lactate), which efficiently predicted in-hospital mortality in patients with AP[23].

4. Treatment interventions for SAP

Upon ICU admission, patients undergo diagnostic tests and severity assessment before treatment initiation. Generally, anti-infective therapy is recommended during their stay in the ICU, though the specific regimen varies and may include beta-lactams such as carbapenems, aminoglycosides, antifungal therapy, and anti-Gram positive agents[24]. For SAP patients experiencing vomiting, respiratory losses, and reduced oral intake, fluid resuscitation is considered crucial. Crystalloid fluids, such as Ringer lactate, are beneficial as a replacement fluid, as they possess anti-inflammatory effects in patients with AP and also reduce the risk of persistent SIRS. Monitoring the response to resuscitation involves maintaining a hematocrit >47%, reducing BUN levels, and urine output >0.5 mL/kg of body weight/hour[25].

Further, enteral nutrition is suggested to be administered within 48 hours of hospital admission for SAP patients to reduce the risk of pancreatic infections and multiple organ failure[25]. It can be administered via nasogastric tube, nasojejunal tube, feeding jejunostomy tube, or a combination of these three[7]. Furthermore, antibiotics such as metronidazole, ciprofloxacin, clindamycin, meropenem, and imipenem are recommended for SAP patients who develop sepsis, pancreatic infections, or infected necrosis systemic inflammatory responses. Moreover, pain management is also crucial, often involving analgesics such as fentanyl, non-steroidal anti-inflammatory drugs[25], and sometimes even combined with opioids[2]. Additionally, China-based consensus guidelines recommend using anti-inflammatory peptides such as somatostatin or octreotide to treat moderate AP and SAP in hospitalized patients[26].

Recent trends involved in the management of AP in the ICU setting also include interventions such as primary percutaneous drainage of intra-abdominal collections, percutaneous nephroscopic necrosectomy, and videoscopic-assisted retroperitoneal debridement[7]. Minimally invasive endoscopic treatment modalities have also become popular, reducing the need for surgical procedures, and leading to lower costs and fewer complications[2]. Furthermore, for patients with biliary pancreatitis with acute cholangitis, an endoscopic retrograde cholangiography can be considered to treat bile duct obstruction[6,27]. Advances in intensive care and surgical interventions have paved the way to decreased mortality rates, shorter hospital stays, and reduced hospital costs[26]. Figure 1 illustrates the treatment and management pathway for AP, encompassing ICU admission, diagnosis, severity assessment, and treatment outcome monitoring.

5. Complications associated with the disease and differential diagnosis

AP is often accompanied by local complications such as infected necrosis (gas in peripancreatic tissue), abdominal hemorrhage (liquid

collections observed in CT), and intestinal perforation (presence of wall thickening, or gas bubbles next to the wall of the intestinal loop)[5]. A Saudi Arabia-based study confirmed peripancreatic fluid collection as one of the most frequently occurring complications in the study cohort[1]. According to a study by Kumar *et al.*, increased incidences of thromboembolic complications (55.6% compared with 12.5%) and necrotizing pancreatitis (11.1%) were observed in patients who were primarily admitted due to COVID-19 and later developed AP[28]. Moreover, a Korea-based retrospective study, reported a higher prevalence of AP in critically ill patients (≥ 18 years) with COVID-19 compared to non-COVID patients. This high prevalence of AP among COVID-19-infected patients (odds ratio = 5.42, 95% confidence interval: 2.35-6.58, $P < 0.01$) indicated COVID-19 infection to be a cause of acute pancreatic damage, specifically in critically ill patients[16].

Moreover, necrotizing pancreatitis is found to be correlated with serious complications such as endocrine and exocrine pancreatic insufficiency, organ failure, fistulae, bleeding, and death. A retrospective cohort study conducted by Kao *et al.* reported complications such as abdominal compartment syndrome (ACS), sepsis (100%), septic shock (72%), renal failure (76%), and hepatic dysfunction (65%) in 47 patients with necrotizing pancreatitis[29]. Another study found that patients with SAP who underwent surgical management for ACS within the first 4 weeks of hospital admission faced an elevated mortality risk[12]. Further, SAP patients with ACS may also experience intra-abdominal hypertension, indicative of a worse prognosis as it leads to pancreatic perfusion[30]. Moreover, the abdominal pain in these patients can be differential for various other conditions such as cholangitis, cholecystitis, bowel perforation, peptic ulcer disease, or mesenteric ischemia. Therefore, an ultrasound or CT scan may be warranted for such differential diagnosis[31].

6. Novel treatment strategies

In recent years, a multidisciplinary approach to the management of AP has gained prominence, with pain management becoming crucial for improving the patient's quality of life. Analgesics are typically used in the management of pain. The Epidural Analgesia for Pancreatitis trial was conducted to assess the effectiveness of epidural analgesia for AP. Patients were randomized into two groups: one received usual care with epidural analgesia while the other group received usual care alone. Although the hypothesized benefit of epidural analgesia was not observed, a reduction in opioid requirement was evident in patients who received epidural analgesia, suggesting potential advantages that merit further investigations[32]. Furthermore, SAP is often accompanied by sepsis, and presepsin may play a vital role as an early indicator of sepsis. A study examined the correlation between the likelihood of sepsis in patients with SAP based on the presepsin levels, evaluating its predictive

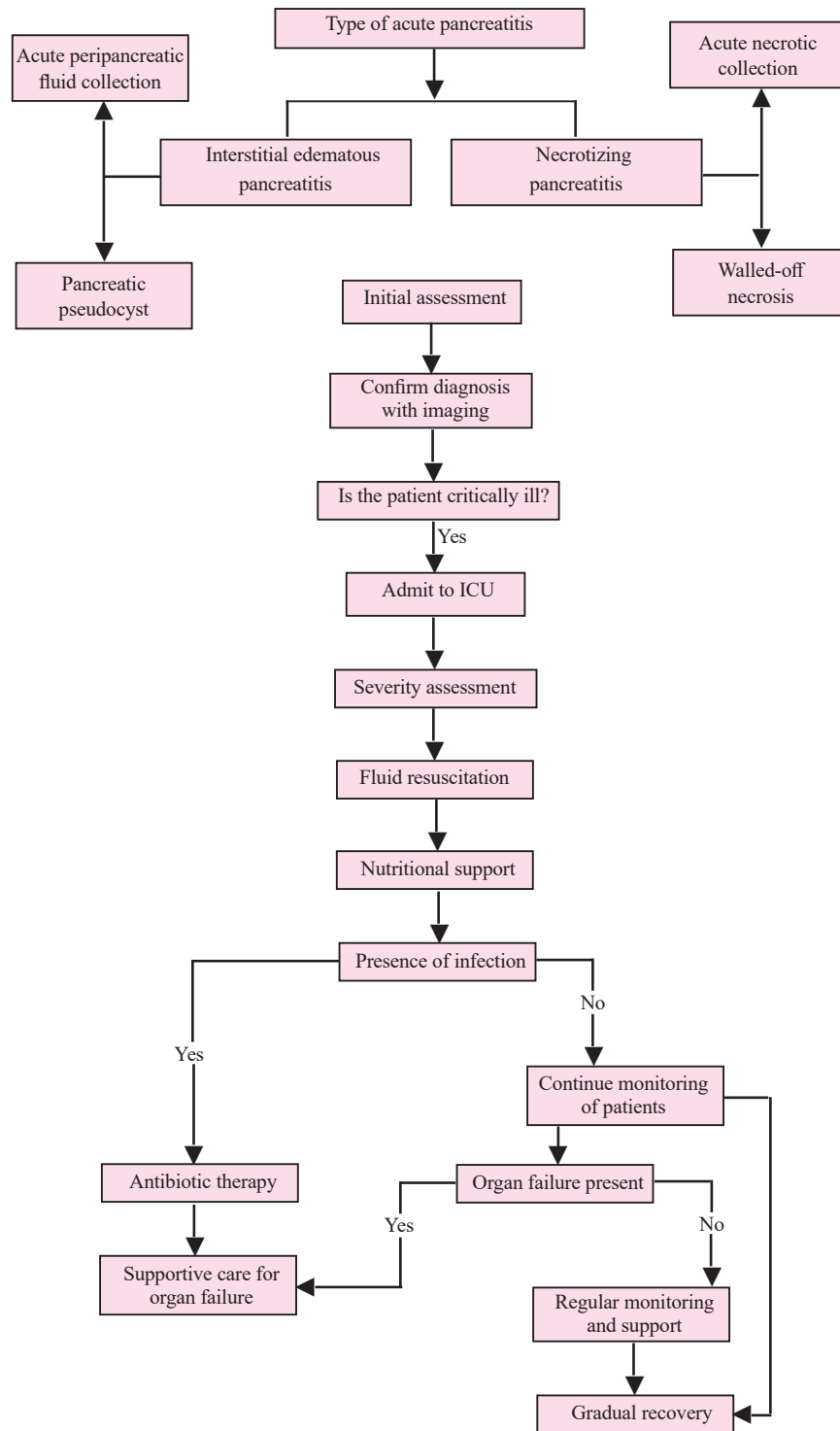


Figure 1. Treatment and management pathway for acute pancreatitis.

power for secondary sepsis and patient outcomes in the ICU setting. The study reported presepsin as the only biomarker independently associated with secondary sepsis and mortality at ICU discharge. Therefore, presepsin demonstrated potential as an early indicator of secondary sepsis and mortality in ICU-admitted SAP patients and provided an option for early risk assessment^[33].

Moreover, ulinastatin, an intrinsic trypsin inhibitor, has shown

promise as a treatment option for AP in patients with organ failure. A study involving 48 patients (up to 60 years old) admitted to the ICU with AP and organ failure underwent standard care. Of these, 25 patients also received a 5-day course of ulinastatin along with standard care. The overall in-hospital mortality was higher in the group that received only standard care (69.6%) than in the ulinastatin group (16%; $P < 0.01$). Furthermore, the hospital stay was reduced

in the ulinastatin group (15.3 days *vs.* 19 days) and greater resolution of organ dysfunction (indicated by improved renal, respiratory, cardiovascular, hepatic function, and coagulation) was also observed in the ulinastatin group. These outcomes suggest that incorporating ulinastatin into the management of SAP can significantly benefit patients^[34].

7. Conclusion

Understanding the etiology, clinical presentation, and severity assessment of AP is paramount for effective management, particularly in the ICU setting. Tailored approaches for diagnosis and treatment of AP based on the various etiologies may be helpful due to their association with organ failure and high mortality rates. Additionally, the adoption of validated severity assessment tools in clinical practice can aid in risk stratification and guiding treatment decisions, thereby improving patient outcomes. Further, advancements in minimally invasive procedures and multidisciplinary care can enhance the management of AP, leading to reduced mortality rates, hospital stays, and costs.

However, AP remains associated with various complications, and differential diagnosis is essential to rule out other conditions and ensure appropriate management. A comprehensive understanding of AP, along with multidisciplinary care and innovative treatment strategies focused on biomarker development, improved imaging, and diagnosis, may reduce the burden associated with this disease in the critical care setting.

Conflict of interest statement

The authors report no conflict of interest.

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Data availability statement

The data supporting the findings of this study are available from the corresponding authors upon request.

Authors' contributions

AHZ: concept, literature search, data acquisition, manuscript

editing and manuscript review, guarantor. MFK: concept, data acquisition, literature search, manuscript preparation, manuscript drafting, editing, and manuscript review.

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