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Original Article

Prognostic Factors and Outcomes in Acute Pancreatitis: A Prospective Study

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ABSTRACT

Background: Acute pancreatitis varies widely in severity, ranging from mild inflammation to life-threatening organ failure. Early identification of prognostic indicators is crucial for timely risk stratification. Objective: To assess prognostic factors and clinical outcomes in patients with acute pancreatitis admitted to a tertiary care hospital. Methods: This prospective observational study included 22 adults at Fatima Memorial Hospital, Lahore. Demographics, etiology, clinical presentation, laboratory markers, BISAP scores, Revised Atlanta severity, complications, and outcomes were recorded. Associations were analyzed using Chi-square or Fisher's exact tests with odds ratios. Results: Gallstones were the leading cause (45.5%). Mild, moderately severe, and severe disease occurred in 54.5%, 27.3%, and 18.2% of patients. Higher BISAP scores correlated with increased severity (p=0.001). Pancreatic necrosis was strongly associated with ICU admission (75.0% vs 11.1%; p=0.024) and severe disease (75.0% vs 5.6%; p=0.010). One death occurred (4.5%). Conclusion: BISAP score and pancreatic necrosis were key early predictors of severe outcomes in acute pancreatitis. Early bedside risk assessment may support timely identification and management of high-risk patients. Keywords: Acute pancreatitis, BISAP, pancreatic necrosis, prognostic factors.

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INTRODUCTION

Acute pancreatitis is an inflammatory disorder of the pancreas with a clinical spectrum ranging from mild, self-limiting disease to severe illness complicated by organ failure, systemic involvement, and significant mortality. The global incidence has risen steadily in recent decades, largely due to increasing rates of gallstone disease, metabolic disorders, and alcohol consumption. Although most patients present with mild disease and recover uneventfully, approximately 15–20% develop moderately severe or severe pancreatitis, which may result in local complications, persistent organ dysfunction, prolonged hospitalization, and death. Early identification of patients at risk for severe disease remains a major clinical challenge, as deterioration often occurs rapidly within the first few days of illness.

The underlying pathophysiology involves premature activation of pancreatic enzymes within acinar cells, leading to autodigestion, inflammation, and the development of systemic inflammatory response syndrome (SIRS). This systemic inflammation can progress to multiorgan dysfunction—including respiratory, renal, and cardiovascular failure—substantially increasing morbidity and mortality. Several etiological factors contribute to disease onset, most notably gallstones and alcohol use, along with hypertriglyceridemia, medications, metabolic disorders, and idiopathic causes.

Because the disease course is highly variable, multiple prognostic tools and biomarkers have been developed to predict severity. Traditional scoring systems such as the Ranson criteria, APACHE II, Glasgow score, and the Bedside Index of Severity in Acute Pancreatitis (BISAP) are widely used for early risk assessment. BISAP, in particular, has gained prominence due to its simplicity and reliability in predicting complications and mortality within the first 24 hours of admission. Higher BISAP scores have consistently been linked with an increased risk of severe pancreatitis, pancreatic necrosis, and death.

Beyond clinical scoring systems, numerous laboratory markers—including C-reactive protein, leukocyte count, neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio—have shown associations with disease severity. Organ dysfunction indicators such as the SOFA score also correlate strongly with in-hospital mortality, particularly at higher thresholds. Metabolic comorbidities including diabetes mellitus, chronic kidney disease, obesity, and advanced age further contribute to worse outcomes.

Large multicenter studies have highlighted the influence of patient-related factors and comorbidity burden on disease progression. Indicators such as pancreatic necrosis, organ failure, and the need for mechanical ventilation have been shown to predict infected necrosis and poor clinical outcomes. However, early prediction remains challenging across diverse clinical settings, especially in resource-limited environments where advanced diagnostic tools may not be readily available.

Variability in disease presentation across regions also complicates clinical management. Differences in etiology, comorbidity patterns, and healthcare resources may affect outcomes, underscoring the importance of locally generated data. In Pakistan, prospective evidence evaluating prognostic indicators in acute pancreatitis is limited, with most available studies being retrospective or conducted in different populations. Thus, context-specific prospective studies are necessary to better understand local disease patterns and factors influencing severity and outcomes.

Against this backdrop, the present prospective observational study was undertaken to evaluate the clinical characteristics, laboratory parameters, and prognostic indicators associated with disease severity and outcomes in patients with acute pancreatitis admitted to a tertiary care hospital in Lahore, Pakistan. The study specifically aimed to describe etiological patterns, assess the prognostic performance of BISAP scoring and laboratory markers, and examine their associations with disease severity, complications, and clinical outcomes.

MATERIALS AND METHODS

This prospective observational study was conducted at the Department of Internal Medicine and Gastroenterology, Fatima Memorial Hospital, Lahore, to evaluate clinical characteristics, prognostic indicators, and outcomes in patients with acute pancreatitis. Eligible adults admitted with acute pancreatitis were consecutively enrolled and followed from admission to discharge or in-hospital death.

Diagnosis was based on at least two standard criteria: typical abdominal pain, serum amylase or lipase levels more than three times the upper limit of normal, or imaging evidence of pancreatic inflammation. Patients with chronic pancreatitis, pancreatic malignancy, traumatic injury, incomplete data, or those who declined participation or were transferred before evaluation were excluded. Consecutive enrollment minimized selection bias.

At admission, demographic data, comorbidities, BMI, smoking status, etiology, and clinical presentation were recorded using structured forms. Laboratory investigations included serum amylase, lipase, complete blood count, blood urea nitrogen, creatinine, calcium, and C-reactive protein. SIRS was assessed using standard criteria. Imaging was performed when clinically indicated to assess pleural effusion or pancreatic necrosis.

Disease severity was classified using the Revised Atlanta Classification. Prognostic assessment was performed using the BISAP score within 24 hours of admission. Outcomes evaluated included ICU admission, vasopressor or ventilatory support, ERCP, interventions for collections or necrosis, hospital stay, and mortality.

All laboratory tests followed standardized protocols in the hospital's accredited laboratory. Data entry was independently verified, and discrepancies were resolved by reviewing medical records. Statistical analysis was conducted using SPSS version 26. Descriptive statistics summarized clinical variables;

associations were assessed using Chi-square or Fisher's exact tests, with odds ratios and 95% confidence intervals. A p-value <0.05 was considered statistically significant.

Ethical approval was obtained from the institutional review committee, and written informed consent was secured from all participants. Patient confidentiality was maintained throughout the study.

RESULTS

All 22 consecutively enrolled patients met the eligibility criteria and were included in the final analysis, with no missing data. The cohort represented a wide adult age range, most commonly 31–45 years (31.8%), followed by 46–60 years (27.3%), >60 years (22.7%), and 18–30 years (18.2%). Males comprised 59.1% of the sample. Overweight and obesity were prevalent, collectively affecting 63.6% of patients. Diabetes mellitus and hypertension were present in 27.3% and 36.4%, respectively, while 31.8% were smokers and 22.7% had a previous episode of pancreatitis. Gallstones were the most common cause of acute pancreatitis (45.5%), followed by alcohol use (22.7%), hypertriglyceridemia (13.6%), idiopathic etiology (13.6%), and drug-induced disease (4.5%). Epigastric pain was reported by all patients, with nausea or vomiting in 81.8% and pain radiating to the back in 72.7%. On admission, tachycardia was noted in 50.0%, dehydration in 40.9%, and hypotension in 22.7% of cases.

Table 1. Baseline characteristics, etiology, and clinical presentation of patients with acute pancreatitis (n = 22)

Variable	Category	n	%
Age group (years)	18–30	4	18.2
	31–45	7	31.8
	46–60	6	27.3
	>60	5	22.7
Sex	Male	13	59.1
	Female	9	40.9
BMI category	Normal	8	36.4
	Overweight	9	40.9
	Obese	5	22.7
Smoking status	Yes	7	31.8
	No	15	68.2
Diabetes mellitus	Yes	6	27.3
	No	16	72.7
Hypertension	Yes	8	36.4
	No	14	63.6
Previous pancreatitis	Yes	5	22.7
	No	17	77.3
Etiology	Gallstone-related	10	45.5
	Alcohol-related	5	22.7
	Hypertriglyceridemia	3	13.6
	Drug-induced	1	4.5
	Idiopathic	3	13.6
Presenting symptoms	Epigastric pain	22	100.0
	Pain radiating to back	16	72.7
	Nausea/vomiting	18	81.8
	Fever	7	31.8
	Abdominal distension	6	27.3
Hemodynamic/clinical status at admission	Hypotension	5	22.7
	Tachycardia	11	50.0
	Dehydration	9	40.9
	Altered mental status	2	9.1

Admission laboratory findings showed that serum lipase was more than three times the upper limit of normal in 90.9% of patients and serum amylase in 77.3%. Elevated white blood cell count was seen in 54.5%, elevated C-reactive protein in 59.1%, elevated blood urea nitrogen in 27.3%, elevated creatinine in 18.2%, and low serum calcium in 22.7%. Pleural effusion was present in 18.2% and systemic inflammatory response syndrome in 36.4% of cases. BISAP scoring demonstrated that half of the patients were categorized as low risk with scores of 0–1, whereas 27.3% had scores ≥3, indicating a clinically important subgroup with higher predicted risk of severe disease and adverse outcomes (Table 2).

Table 2. Admission laboratory profile and BISAP score distribution (n = 22)

Variable	Category	n	%
Serum lipase	>3 times upper limit	20	90.9
Serum amylase	>3 times upper limit	17	77.3
White blood cell count	Elevated	12	54.5

Variable	Category	n	%
Blood urea nitrogen	>25 mg/dL	6	27.3
Serum creatinine	Elevated	4	18.2
Serum calcium	Low	5	22.7
CRP	Elevated	13	59.1
Pleural effusion	Present	4	18.2
SIRS	Present	8	36.4
BISAP score	0–1	11	50.0
	2	5	22.7
	3	4	18.2
	4–5	2	9.1

According to the Revised Atlanta Classification, 54.5% of patients had mild acute pancreatitis, 27.3% had moderately severe disease, and 18.2% had severe acute pancreatitis. Local and systemic complications were not uncommon. Acute peripancreatic fluid collection was documented in 27.3%, pancreatic necrosis in 18.2%, pleural effusion in 18.2%, acute kidney injury in 13.6%, and respiratory failure in 13.6%. Cardiovascular failure or shock occurred in 9.1%, while infected necrosis and multiorgan failure were each observed in 4.5% of the cohort (Table 3). These data indicate that although most patients had mild disease, a clinically meaningful minority progressed to severe disease with organ dysfunction and complex local complications.

Table 3. Disease severity and complications among patients with acute pancreatitis (n = 22)

Variable	Category	n	%
Severity (Revised Atlanta Classification)	Mild acute pancreatitis	12	54.5
	Moderately severe acute pancreatitis	6	27.3
	Severe acute pancreatitis	4	18.2
Local and systemic complications	Pancreatic necrosis	4	18.2
	Acute peripancreatic fluid collection	6	27.3
	Pleural effusion	4	18.2
	Acute kidney injury	3	13.6
	Respiratory failure	3	13.6
	Cardiovascular failure/shock	2	9.1
	Infected necrosis	1	4.5
	Multiorgan failure	1	4.5

With respect to hospital course, 22.7% of patients required ICU admission, 9.1% required vasopressor support, and 9.1% required mechanical ventilation. ERCP was performed in 18.2%, reflecting the substantial proportion of gallstone-related pancreatitis, while intervention for collections or necrosis was required in 9.1%. Most patients (59.1%) had a hospital stay of 7 days or less, but 40.9% required hospitalization beyond 7 days. Overall survival was favorable, with 21 of 22 patients discharged alive and one in-hospital death, yielding a mortality rate of 4.5% (Table 4).

Table 4. Hospital course and final outcomes of patients with acute pancreatitis (n = 22)

Variable	Category	n	%
ICU admission	Yes	5	22.7
	No	17	77.3
Vasopressor support	Yes	2	9.1
	No	20	90.9
Mechanical ventilation	Yes	2	9.1
	No	20	90.9
ERCP performed	Yes	4	18.2
	No	18	81.8
Intervention for collections/necrosis	Yes	2	9.1
	No	20	90.9
Length of stay	≤7 days	13	59.1
	>7 days	9	40.9
Final status	Discharged alive	21	95.5
	Died	1	4.5

A strong association was observed between BISAP score category and disease severity. Among patients with BISAP scores of 0–1, 90.9% had mild pancreatitis and none developed severe disease. In contrast, among those with BISAP scores of 4–5, all patients developed severe acute pancreatitis. Patients with BISAP score 3 also showed marked risk escalation, with 50.0% classified as severe and the remaining 50.0% as moderately severe. Overall, the distribution of disease severity differed significantly across BISAP categories (Pearson chi-square = 23.23, df = 6, p = 0.001), supporting BISAP as a clinically useful early prognostic indicator in this cohort (Table 5A).

Pancreatic necrosis was also strongly associated with worse clinical outcomes. Patients with necrosis had substantially higher ICU admission rates than those without necrosis (75.0% vs 11.1%; OR 24.00, 95% CI

1.62–356.65; Fisher's exact p = 0.024). Severe acute pancreatitis was present in 75.0% of patients with necrosis compared with only 5.6% of those without necrosis (OR 51.00, 95% CI 2.46–1057.09; Fisher's exact p = 0.010). Mortality occurred only among patients with necrosis (25.0% vs 0.0%); however, this association did not reach statistical significance, likely due to the small sample size and low event count (Fisher's exact p = 0.182) (Table 5B).

Table 5A. Association of BISAP score with severity of acute pancreatitis (n = 22)

BISAP score	Mild n (%)	Moderately severe n (%)	Severe n (%)	Total	p-value
0–1	10 (90.9)	1 (9.1)	0 (0.0)	11	
2	2 (40.0)	3 (60.0)	0 (0.0)	5	
3	0 (0.0)	2 (50.0)	2 (50.0)	4	
4–5	0 (0.0)	0 (0.0)	2 (100.0)	2	
Overall association				22	0.001*

*Pearson chi-square test, $\chi^2 = 23.23$, df = 6.

Table 5B. Association of pancreatic necrosis with major outcomes (n = 22)

Outcome	Necrosis present (n = 4)	Necrosis absent (n = 18)	Odds ratio (95% CI)	p-value
ICU admission	3 (75.0)	2 (11.1)	24.00 (1.62–356.65)	0.024†
Severe pancreatitis	3 (75.0)	1 (5.6)	51.00 (2.46–1057.09)	0.010†
Mortality	1 (25.0)	0 (0.0)	15.86 (0.53–474.41)‡	0.182†

† Fisher's exact test. ‡ Odds ratio estimated with continuity correction because of a zero cell.

All 22 enrolled patients were included in the final analysis, with no missing observations in the analyzed variables. The study population was predominantly male (59.1%), and the most represented age group was 31–45 years (31.8%). Overweight and obesity together were observed in 63.6% of patients, while diabetes mellitus and hypertension were present in 27.3% and 36.4%, respectively. Gallstones were the leading etiology of acute pancreatitis (45.5%), followed by alcohol use (22.7%), indicating that biliary and metabolic risk profiles were dominant in this cohort. Clinically, all patients presented with epigastric pain, and high frequencies of nausea or vomiting (81.8%) and pain radiating to the back (72.7%) further reflected the typical symptomatic pattern of acute pancreatitis.

Laboratory findings at presentation showed that elevated pancreatic enzymes were common, with serum lipase exceeding three times the upper limit of normal in 90.9% and serum amylase in 77.3% of patients. Inflammatory and severity-related markers were also notable: white blood cell count was elevated in 54.5%, CRP in 59.1%, and SIRS was present in 36.4%. Half of the patients had a BISAP score of 0–1, whereas 27.3% had a BISAP score of 3 or higher, suggesting a smaller but clinically relevant subgroup at increased early risk of severe disease.

Severity classification demonstrated that 54.5% of patients had mild disease, 27.3% had moderately severe disease, and 18.2% had severe acute pancreatitis. Acute peripancreatic fluid collection was the most frequent complication (27.3%), followed by pancreatic necrosis (18.2%), pleural effusion (18.2%), acute kidney injury (13.6%), and respiratory failure (13.6%). During hospitalization, 22.7% of patients required ICU admission, 9.1% required vasopressors, and 9.1% required mechanical ventilation. Although most patients were discharged alive (95.5%), one patient died, resulting in an in-hospital mortality rate of 4.5%.

A statistically significant association was observed between BISAP score and disease severity. Among patients with BISAP scores of 0–1, 90.9% had mild disease and none developed severe pancreatitis, whereas all patients with BISAP scores of 4–5 developed severe pancreatitis. This gradient across risk strata was significant on chi-square testing (p = 0.001), supporting the discriminatory value of BISAP in early severity assessment. Pancreatic necrosis also showed strong adverse prognostic significance. Patients with necrosis had markedly higher ICU admission rates (75.0% vs 11.1%; p = 0.024) and much greater frequency of severe pancreatitis (75.0% vs 5.6%; p = 0.010) than those without necrosis. Mortality occurred only in the necrosis group (25.0% vs 0.0%), but this did not achieve statistical significance (p = 0.182), most likely because only one death occurred in the study population.

DISCUSSION

The present prospective observational study evaluated prognostic factors and clinical outcomes among patients admitted with acute pancreatitis at a tertiary care hospital in Lahore, Pakistan, and demonstrated that most patients had mild disease, while a smaller but clinically important subgroup developed moderately severe or severe pancreatitis with local and systemic complications. Gallstone-related pancreatitis was the most frequent etiology, followed by alcohol-related disease, which is consistent with the etiologic pattern reported in contemporary clinical literature describing biliary and alcohol-related causes as the dominant contributors to acute pancreatitis worldwide (7). The predominance of epigastric pain, nausea or vomiting, and back-radiating pain in the present cohort also reflects the classic symptomatic profile described in standard clinical reviews and cohort studies of acute pancreatitis (7,15).

A key finding of this study was the strong association between BISAP score and disease severity. Patients with low BISAP scores of 0–1 were overwhelmingly represented in the mild disease category, whereas all patients with BISAP scores of 4–5 developed severe acute pancreatitis. This graded relationship supports the clinical utility of BISAP as an early bedside prognostic tool in acute pancreatitis, particularly in settings where rapid risk stratification is necessary. These findings are in agreement with prior prospective and observational studies showing that BISAP performs well in identifying patients at increased risk of severe disease, organ failure, and mortality by integrating simple variables available within the first 24 hours of admission (3,12). The practical value of BISAP is especially relevant in resource-constrained hospital environments because it requires no advanced imaging or complex calculations, thereby facilitating timely clinical decision-making.

The present data also highlighted the adverse prognostic significance of pancreatic necrosis. Patients with necrosis had markedly higher rates of ICU admission and severe pancreatitis than those without necrosis, and the only death in the cohort occurred in a patient with necrosis. This pattern is clinically plausible and consistent with prior evidence showing that necrosis, particularly when extensive or infected, is strongly associated with organ dysfunction, prolonged hospitalization, and mortality in acute pancreatitis (16). Although the mortality association in this study did not reach statistical significance, this was most likely due to the limited sample size and the occurrence of only one death event. The direction of effect nevertheless supports the broader literature indicating that pancreatic necrosis represents an important marker of poor prognosis and a potential driver of clinical deterioration (16).

Inflammatory and biochemical abnormalities at admission further reinforce the concept that early systemic response influences disease progression. Elevated white blood cell count, elevated CRP, SIRS, pleural effusion, and derangements in renal parameters were present in a substantial proportion of patients. Previous research has shown that inflammatory markers, including leukocyte count, neutrophil-based ratios, and CRP, correlate with severity and may complement formal scoring systems in early prognostic assessment (2,12). Similarly, organ dysfunction-related indicators such as creatinine and blood urea nitrogen have been linked to worse outcomes and are often incorporated into prognostic models because they reflect evolving systemic compromise (5,9). The present results do not establish causality, but they support clinical relevance of combining bedside scoring with routine laboratory evaluation to improve early identification of high-risk patients.

The observed frequency of obesity, obesity, diabetes mellitus, and hypertension in this cohort also deserves attention. More than half of the study population was overweight or obese, and over one-quarter had diabetes mellitus. These findings are relevant because metabolic comorbidities are increasingly recognized as modifiers of disease severity in acute pancreatitis. Prior large prospective studies have shown that obesity and comorbidity burden are associated with persistent organ failure and mortality, while narrative evidence has suggested that diabetes and chronic kidney disease may worsen inflammatory response, delay recovery, and prolong hospitalization (6,8,11). Although the current sample was too small to support robust multivariable adjustment for these comorbidities, their

frequency within the cohort suggests that they may meaningfully contribute to adverse outcomes and should be explored in larger local studies.

The hospital-course data in the present study showed that nearly one-quarter of patients required ICU admission, while smaller proportions required vasopressor support, mechanical ventilation, ERCP, or intervention for collections or necrosis. These findings indicate that a non-negligible subset of patients progressed beyond uncomplicated disease and required advanced supportive care or procedure-based management. Such observations are consistent with tertiary-care studies demonstrating that even when most acute pancreatitis cases are mild, severe presentations consume disproportionately greater critical care resources and are associated with more complicated clinical trajectories (14,15). The relatively low mortality observed in the present study may reflect timely hospital-based management, the predominance of mild disease, and the limited sample size rather than absence of prognostic risk.

This study has several strengths. It used prospective observational design, consecutive patient inclusion, standardized diagnostic criteria, and clinically relevant severity classification based on the Revised Atlanta framework. In addition, the study evaluated a combination of demographic, clinical, laboratory, and outcome variables within a real-world tertiary-care setting, which improves its practical clinical relevance. At the same time, several limitations should be acknowledged. The study was conducted at a single center and included only 22 patients, which limits external validity and statistical power. The small number of severe cases and the occurrence of only one death restricted the precision of effect estimates and widened confidence intervals for several associations. The modest sample also limited the ability to perform adjusted multivariable analyses to account for potential confounding by age, comorbidity burden, etiology, or obesity. In addition, some laboratory and imaging markers reported in broader prognostic literature were not evaluated in sufficient depth for comparative modeling. These limitations should be considered when interpreting the results, and the present findings should be viewed as clinically informative but preliminary.

Despite these constraints, the study provides useful local evidence indicating that BISAP score and pancreatic necrosis are important early indicators of severe disease and adverse hospital outcomes in patients with acute pancreatitis. The findings support the incorporation of simple bedside prognostic assessment into early routine care and underscore the importance of close monitoring in patients presenting with higher BISAP scores, necrosis, inflammatory response, or comorbid metabolic disease. Future multicenter prospective studies with larger sample sizes, longer follow-up, and multivariable prognostic modeling are needed to validate these findings and develop context-specific risk stratification strategies for acute pancreatitis in Pakistani tertiary-care settings (13,18-20).

CONCLUSION

In this prospective observational study of patients with acute pancreatitis at Fatima Memorial Hospital, Lahore, most patients had mild disease, but a clinically important proportion developed moderately severe or severe pancreatitis with significant complications and need for critical care support. Higher BISAP scores showed a clear association with increasing disease severity, while pancreatic necrosis was strongly associated with ICU admission and severe pancreatitis. Gallstone disease was the leading etiology, and inflammatory, renal, and systemic response markers were frequently abnormal at presentation. These findings suggest that early bedside risk stratification using BISAP, combined with careful assessment of complications such as pancreatic necrosis, may improve identification of high-risk patients and support timely clinical management in tertiary-care practice.

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