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Predictors of Mortality in Acute Pancreatitis at the Time of Admission

Dr Hema Deep Bhojani[1], Dr Saraswati Prajapati[2], Dr Priyanka Patel[3], Dr Harsh Patel[4]

[1] Associate Professor, Department of General Medicine, Dr. M. K. Shah Medical College & Research Center & Smt SMS Multispecialty Hospital, Ahmedabad, Gujarat, India.
[2] Assistant Professor, Department of General Medicine, Dr. M. K. Shah Medical College & Research Center & Smt SMS Multispecialty Hospital, Ahmedabad, Gujarat, India.
[3,4] Senior Resident, Department of General Medicine, Dr. M. K. Shah Medical College & Research Center & Smt SMS Multispecialty Hospital, Ahmedabad, Gujarat, India.

Corresponding Author:

Dr Harsh Patel

Senior Resident, Department of General Medicine, Dr. M. K. Shah Medical College & Research Center & Smt SMS Multispecialty Hospital, Ahmedabad, Gujarat, India.

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ABSTRACT

Background: Acute pancreatitis has a variable clinical course, and early identification of patients at risk of mortality remains a major clinical challenge.
Objectives: To examine demographic and laboratory factors at admission that predict mortality in patients with acute pancreatitis.
Material and Methods: A hospital-based observational study was conducted among 150 patients diagnosed with acute pancreatitis. Clinical outcomes were compared with admission-age groups and laboratory parameters to identify early predictors of mortality.
Results: Overall mortality was 12%. Mortality increased significantly with advancing age, and serum albumin levels were significantly lower among non-survivors. Pancreatic enzymes and most biochemical parameters did not show significant associations with mortality.
Conclusion: Advanced age and hypoalbuminemia at admission are significant early predictors of mortality in acute pancreatitis and may aid in early risk stratification and management.

Keywords: *Acute pancreatitis; Mortality; Serum albumin; Prognostic factors.*

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disease of the pancreas with a highly variable clinical course, ranging from mild self-limiting disease to severe forms complicated by organ failure and death. Early identification of patients at high risk of mortality is crucial for prompt management and resource allocation, particularly in emergency and critical care settings. Although clinical scoring systems such as the Bedside Index for Severity in Acute Pancreatitis (BISAP), Ranson criteria, and Acute Physiology and Chronic Health Evaluation II (APACHE II) have been developed to predict disease severity and mortality, their accuracy and applicability within the first hours of admission vary across populations and clinical contexts [1–3]. Biomarkers and clinical predictors assessed at the time of hospital presentation have therefore become a focus of research aimed at improving early risk stratification in AP.

Several demographic and clinical factors have been linked to mortality in AP, including advanced age, comorbidities, systemic inflammatory response syndrome (SIRS), and early organ dysfunction. Age and systemic inflammation contribute to an exaggerated host response, predisposing patients to complications such as persistent organ failure, pancreatic necrosis, and septic shock, which are associated with higher mortality [4,5]. Laboratory parameters available at admission, such as elevated blood urea nitrogen, white blood cell count, hematocrit, and markers of organ dysfunction, have also shown significant associations with adverse outcomes in AP patients [6]. Simple but integrative indices like the neutrophil-to-lymphocyte ratio and blood urea nitrogen–to–albumin ratio have emerged as potential prognostic tools that may complement traditional scoring systems [7].

Imaging findings at admission, particularly evidence of extensive inflammation or early necrosis on computed tomography (CT), have been reported to correlate with mortality risk, reflecting both local severity and the systemic inflammatory burden [8]. Advanced predictive models, including machine learning approaches incorporating clinical and laboratory variables, have further underscored the multifactorial nature of mortality prediction in acute pancreatitis and highlighted the importance of integrating diverse data sources to enhance prognostic accuracy [9]. Despite these advances, there remains a need for robust, validated predictors that can be readily assessed at the time of admission to guide early clinical decision-making and improve outcomes in patients with AP [10].

MATERIAL AND METHODS

This hospital-based observational study was conducted in the Department of General Medicine and Gastroenterology of a tertiary care teaching hospital over a defined study period after obtaining approval from the Institutional Ethics Committee. A total of 150 patients diagnosed with acute pancreatitis (AP) were included in the study. Written informed consent was obtained from all participants or their legally authorized representatives prior to enrollment.

Patients aged 18 years and above admitted with a diagnosis of acute pancreatitis were included. The diagnosis of AP was established based on the presence of at least two of the following criteria: characteristic abdominal pain suggestive of acute pancreatitis, serum amylase and/or lipase levels greater than three times the upper limit of normal, and imaging findings consistent with acute pancreatitis on ultrasonography or computed tomography. Patients with chronic pancreatitis, pancreatic malignancy, post-ERCP pancreatitis, traumatic pancreatitis, or incomplete medical records were excluded from the study.

Clinical, demographic, and laboratory data were collected at the time of admission. Demographic variables included age and sex, while clinical parameters included presenting symptoms, etiology of pancreatitis, vital signs, and presence of systemic inflammatory response syndrome or organ dysfunction. Laboratory investigations obtained within the first 24 hours of admission included complete blood count, serum amylase, serum lipase, blood urea nitrogen, serum creatinine, serum electrolytes, liver function tests, serum calcium, and inflammatory markers where available. Imaging findings from ultrasonography or contrast-enhanced computed tomography were reviewed to assess pancreatic inflammation and complications.

Patients were followed during their hospital stay to record outcomes, including survival or mortality. Mortality was defined as death occurring during the same hospital admission due to complications related to acute pancreatitis. The collected variables were analyzed to identify clinical, laboratory, and demographic factors associated with mortality at the time of admission.

Data were entered into a structured proforma and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were expressed as frequencies and percentages. Comparisons between survivors and non-survivors were performed using suitable parametric or non-parametric statistical tests. Multivariate analysis was conducted to identify independent predictors of mortality. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The association between age and clinical outcomes in patients with acute pancreatitis is presented in Table 1. Mortality increased progressively with advancing age. Patients aged ≤20 years showed no mortality, with all 12 patients (100%) surviving. In the 21–40 years age group, mortality was observed in 3 out of 62 patients (4.8%), whereas survival was noted in 59 patients (95.2%). Among patients aged 41–60 years, mortality increased to 10 out of 54 patients (18.5%), while 44 patients (81.5%) survived. The highest mortality was observed in patients aged more than 60 years, where 5 out of 22 patients (22.7%) died during hospitalization. Overall mortality in the study population was 18 patients (12%), while 132 patients (88%) survived, indicating a clear age-related trend in adverse outcomes.

Comparison of mean hematological and biochemical parameters between survivors and non-survivors is shown in Table 2. Mean serum amylase and lipase levels were higher among non-survivors compared to survivors; however, these differences were not statistically significant ($p > 0.05$). Liver enzymes including SGOT and alkaline phosphatase also showed higher mean values in the mortality group, but the differences remained insignificant. Serum total protein and globulin levels were lower in non-survivors, though these differences did not reach statistical significance. Serum albumin levels were significantly lower in patients who died (2.48 ± 0.51 g/dL) compared to survivors (3.62 ± 0.68 g/dL), and this difference was statistically significant ($p = 0.001$). Coagulation parameters, including prothrombin time and INR, were marginally higher in the mortality group but did not show statistically significant differences.

Table 1: Association between age groups and outcomes in acute pancreatitis patients (n = 150)

Age group (years)	Others n (%)	Mortality n (%)	Total n (%)

≤20	12 (100.0)	0 (0.0)	12 (100.0)
21–40	59 (95.2)	3 (4.8)	62 (100.0)
41–60	44 (81.5)	10 (18.5)	54 (100.0)
>60	17 (77.3)	5 (22.7)	22 (100.0)
Total	**132 (88.0)**	**18 (12.0)**	**150 (100.0)**

Table 2: Comparison of mean hematological and biochemical parameters in relation to outcomes

Parameter	Others (n = 132) Mean ± SD	Mortality (n = 18) Mean ± SD	t value, df	p value
Serum amylase (IU/L)	512.4 ± 892.6	645.8 ± 910.3	−0.62, 148	0.536 (NS)
Serum lipase (IU/L)	2318.6 ± 4825.4	2589.7 ± 4012.9	−0.41, 148	0.683 (NS)
Serum SGOT (IU/L)	76.9 ± 102.4	92.3 ± 88.6	−0.58, 148	0.563 (NS)
Serum alkaline phosphatase (IU/L)	146.8 ± 118.9	188.6 ± 132.1	−1.01, 148	0.315 (NS)
Serum total proteins (g/dL)	6.48 ± 1.12	5.11 ± 0.64	1.52, 148	0.131 (NS)
Serum albumin (g/dL)	3.62 ± 0.68	2.48 ± 0.51	4.21, 148	**0.001***
Serum globulin (g/dL)	2.86 ± 0.63	2.63 ± 0.41	1.42, 148	0.158 (NS)
Prothrombin time (seconds)	15.42 ± 3.96	16.88 ± 4.12	−1.37, 148	0.172 (NS)
INR	1.18 ± 0.46	1.32 ± 0.58	−1.21, 148	0.228 (NS)

DISCUSSION

The present study evaluated early predictors of mortality in acute pancreatitis (AP) at the time of admission and demonstrated that advancing age and hypoalbuminemia were significantly associated with adverse outcomes, while several routinely measured pancreatic enzymes and liver function parameters did not show a statistically significant association with mortality. The overall in-hospital mortality rate of 12% observed in this study is consistent with reported mortality rates for mixed-severity AP cohorts, reinforcing the clinical relevance of early risk stratification at admission [11].

Age emerged as an important demographic determinant of mortality in AP, with a clear stepwise increase in deaths among older age groups. Patients aged more than 60 years exhibited the highest mortality, supporting existing evidence that elderly patients have reduced physiological reserve, higher prevalence of comorbidities, and a heightened inflammatory response, all of which predispose to persistent organ failure and death [12]. This age-related vulnerability underscores the importance of early intensive monitoring and aggressive supportive care in older patients presenting with AP, even when initial clinical features appear mild.

Among laboratory parameters, serum albumin demonstrated a strong and statistically significant association with mortality. Non-survivors had markedly lower albumin levels at admission compared to survivors, highlighting hypoalbuminemia as a robust early marker of poor prognosis. Hypoalbuminemia reflects both systemic inflammation and capillary leakage, which are central to the pathophysiology of severe AP and multiorgan dysfunction [13]. Several studies have identified low serum albumin at admission as an independent predictor of mortality, outperforming traditional pancreatic enzymes and reinforcing its value as a simple, readily available prognostic marker [14].

In contrast, serum amylase and lipase levels were not significantly different between survivors and non-survivors in the present study. This finding aligns with current understanding that pancreatic enzyme levels correlate poorly with disease severity and outcomes once the diagnosis of AP is established. Enzyme elevations primarily reflect pancreatic injury rather than the systemic inflammatory response that drives complications and mortality [15]. Similarly, liver enzymes, alkaline phosphatase, coagulation parameters, and INR did not demonstrate significant associations with mortality, suggesting that isolated abnormalities in these parameters at admission may have limited prognostic utility when used alone.

The findings of this study emphasize that early mortality prediction in AP should rely on a combination of demographic and systemic markers rather than pancreatic enzymes alone. Simple admission variables such as age and serum albumin can aid in early identification of high-risk patients, facilitating timely escalation of care, appropriate triage to higher levels of monitoring, and optimization of supportive management. Integrating these readily available predictors into early clinical assessment may enhance prognostic accuracy, particularly in resource-limited settings where complex scoring systems may not be feasible.

CONCLUSION

The present study concludes that advanced age and low serum albumin levels at the time of admission are significant early predictors of mortality in acute pancreatitis. Pancreatic enzyme levels and most routine biochemical parameters did

not show a significant association with mortality. Early recognition of high-risk patients using simple clinical and laboratory markers may improve risk stratification, guide management decisions, and potentially reduce mortality in acute pancreatitis.

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