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ROLE OF BIOCHEMICAL PARAMETERS IN MORTALITY PREDICTION IN PATIENTS DIAGNOSED WITH ACUTE PANCREATITIS IN A TERTIARY CARE.

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Abstract

Background: Acute pancreatitis (AP) is a common gastrointestinal emergency with significant morbidity and mortality. Early identification of patients at risk of adverse outcomes remains challenging. Biochemical parameters are readily available, cost-effective investigations that may aid in early risk stratification and mortality prediction.

Objectives: To evaluate the role of selected biochemical parameters in predicting in-hospital mortality among patients diagnosed with acute pancreatitis in a tertiary care setting.

Methods: This prospective observational study was conducted over a two-year period (August 2022–September 2023) and included 100 adult patients diagnosed with acute pancreatitis based on the Revised Atlanta Classification 2012. Demographic data, etiological factors, disease severity, and clinical outcomes were recorded. Biochemical parameters assessed at admission included hematocrit, blood urea nitrogen (BUN), serum procalcitonin, serum amylase, serum lipase, serum C-reactive protein (CRP), arterial blood gas parameters (pH, PaO_2, $PaCO_2$), and neutrophil–leukocyte ratio (NLR). The Bedside Index for Severity in Acute Pancreatitis (BISAP) score was calculated within the first 24 hours. Biochemical parameters were compared between survivors and non-survivors. ROC curve analysis was performed to identify independent predictors of mortality.P <0.05 cosidered as statistically significant.

Results: The mean age of the study population was 37.25 ± 7.3 years, with a male predominance (59%). In-hospital mortality was observed in 7% of patients. Non-survivors had significantly higher hematocrit (48.7 ± 6.4%), BUN (54.3 ± 11.6 mg/dL), serum procalcitonin (6.8 ± 3.1 ng/mL), CRP (268 ± 74 mg/L), pancreatic enzyme levels, and NLR (18.9 ± 6.5), along with lower arterial pH and PaO_2 ($p < 0.05$). ROC analysis demonstrated excellent predictive performance of NLR, serum procalcitonin, BUN, and CRP. Multivariate logistic regression identified elevated NLR, increased serum procalcitonin, raised BUN, high CRP levels, and metabolic acidosis as independent predictors of mortality.

Conclusion: Routinely available biochemical parameters play a crucial role in early prognostication and mortality prediction in acute pancreatitis. Their integration into routine clinical assessment can

aid in timely risk stratification, guide management decisions, and improve patient outcomes, particularly in resource-limited tertiary care settings.

Keywords: Bicohemical parameters, mortality prediction, Acute pancreatitis, tertiary care.

Introduction: Acute pancreatitis is an inflammatory disorder of the pancreas resulting from premature activation of pancreatic enzymes within the gland, leading to autodigestion of pancreatic tissue. This process produces parenchymal edema and areas of necrosis and, in severe forms, may progress to systemic complications, multi-organ dysfunction, or death.[1] Alcohol intake and gallstone disease are the predominant etiological factors, together accounting for nearly 80% of cases, with alcohol-related pancreatitis occurring more frequently.[2] The disease may be complicated by several local sequelae, including pancreatic pseudocyst formation, walled-off pancreatic necrosis, and disconnected pancreatic duct syndrome.[3]The clinical spectrum of acute pancreatitis ranges from a mild, self-limited illness to a severe, life-threatening condition.[4] Disease severity is a key determinant of mortality and influences the need for intensive care, nutritional support, urgent surgical intervention, and antibiotic therapy.[5] Severe acute pancreatitis is associated with substantial morbidity and mortality, largely due to pancreatic and extrapancreatic necrosis, secondary infection, and the development of multiorgan failure.[6–8]Several scoring systems, including Ranson, APACHE II, SOFA, and BISAP, are used to assess disease severity. Among these, the BISAP score is a simple bedside tool that enables early risk stratification within the first 24 hours of hospital admission and helps identify patients at increased risk of in-hospital mortality, even before organ failure develops. A BISAP score greater than three is associated with a mortality rate ranging from 5% to 20%.[9,10]Despite the availability of multiple prognostic models, accurately predicting disease progression remains challenging, as patients with similar initial clinical and radiological severity may follow markedly different clinical courses.[11] Early recognition and timely management are therefore essential to reduce morbidity and mortality. Advances in intensive care have significantly improved outcomes in acute pancreatitis; however, optimal management requires a multidisciplinary approach tailored to individual patient factors, available expertise, and dynamic risk assessment.[2,11]

Biochemical parameters are simple, objective, readily available, and cost-effective investigations that are routinely performed at admission and during hospital stay in patients with acute pancreatitis. Markers such as haematocrit (HCT), C-reactive protein (CRP), blood urea nitrogen (BUN), arterial blood gas parametrs (ABG), procalcitonin and neutrophil and leucocyte ration (NLR) have been shown to reflect disease severity, systemic inflammation, metabolic derangements, and organ dysfunction. These parameters may offer valuable prognostic information for early risk stratification and mortality prediction.

However, data regarding the predictive value of individual and combined biochemical parameters for mortality in acute pancreatitis in the Indian population are limited and inconsistent. Hence this study was done to understand the biochemical predictors in patients with acute pancreatitis which can be used by the clinicians in assessing patients' condition for better management to improve outcome thus decreasing the mortality.

Methodology:A prospective observational study was conducted over a two-year period from August 2022–September 2023 in a tertiary care hospital and included 100 adult patients of either sex diagnosed with acute pancreatitis. Approval for the study was obtained from the Institutional Ethics Committee prior to enrolment.Patients presenting to the surgical outpatient department or emergency services and subsequently admitted for inpatient care were screened for inclusion. Diagnosis was established when at least two of the following three criteria were fulfilled, in accordance with the Revised Atlanta Classification 2012:[12] (i) abdominal pain consistent with acute pancreatitis, (ii) serum amylase and/or lipase levels exceeding three times the upper limit of normal, and (iii) imaging findings suggestive of acute pancreatitis on abdominal ultrasonography,

computed tomography, or magnetic resonance imaging. Based on this classification, cases were categorized as mild, moderately severe, or severe. Mild acute pancreatitis was defined by the absence of organ failure and local or systemic complications, with resolution typically within one week. Moderately severe acute pancreatitis was characterized by transient organ failure, local complications, or worsening of pre-existing comorbidities. Severe acute pancreatitis was defined by persistent organ failure lasting more than 48 hours. Local complications included peripancreatic fluid collections, pancreatic or peripancreatic necrosis (sterile or infected), pseudocyst formation, and walled-off necrosis.Patients with a prior diagnosis of chronic pancreatitis, evidenced by hospital records or radiological features such as pancreatic calcifications, ductal dilatation, parenchymal atrophy, or pseudocyst formation, as well as those with pancreatic malignancy or unwillingness to participate, were excluded from the study.A comprehensive clinical evaluation was performed for all participants, including detailed medical and surgical history and physical examination. Baseline laboratory investigations obtained at admission included arterial blood gas analysis, serum procalcitonin, C-reactive protein (CRP), hematocrit, renal and liver function tests, serum electrolytes, serum amylase, serum lipase, and complete blood counts. Abdominal ultrasonography was carried out at presentation, while contrast-enhanced computed tomography of the pancreas was performed after 72 hours of hospitalization.The Bedside Index for Severity in Acute Pancreatitis (BISAP) score was calculated within the first 24 hours of admission to assess early mortality risk prior to the development of organ failure.[13] One point was assigned for each of the following parameters: blood urea nitrogen level greater than 8.9 mmol/L, impaired mental status, presence of systemic inflammatory response syndrome, age over 60 years, and pleural effusion on imaging. A BISAP score exceeding three was considered indicative of an increased risk of mortality.[8,13]Statistical analysis was performed using IBM SPSS software version 20.0. Continuous variables were expressed as mean ± standard deviation with 95% confidence intervals, while categorical variables were presented as proportions. Univariate analysis was conducted using one-way analysis of variance and Pearson's chi-square test to determine associations between variables. A two-tailed p-value of less than 0.05 was considered statistically significant.

Results:

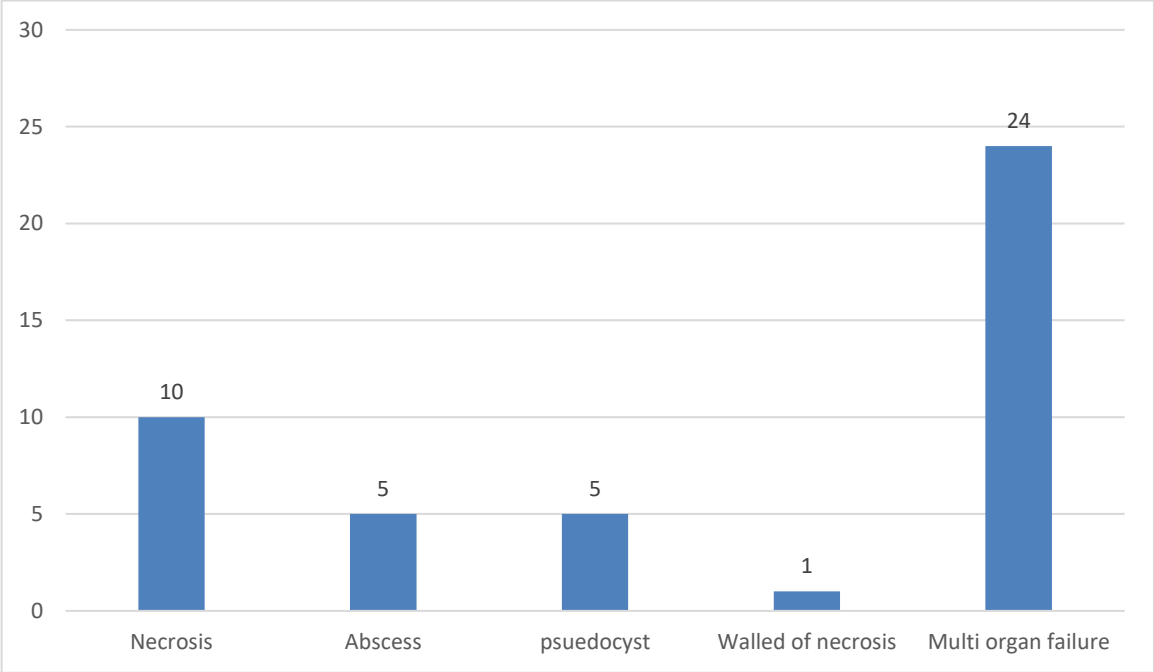
The study population had a mean age of 37.25 ± 7.3 years, with most patients (45%) belonging to the 31–40-year age group. Males constituted a higher proportion of cases (59%) compared to females (41%). Gallstone disease was the most common etiological factor (41%), followed by alcohol consumption (31%), while hypertriglyceridemia accounted for 12% of cases; in 26% of patients, the cause remained unidentified. Based on the Revised Atlanta Classification, 46% of patients had mild acute pancreatitis, whereas moderately severe and severe disease was observed in 27% each. A BISAP score greater than 3 was noted in 38% of patients. Overall in-hospital mortality was 7%, with survival observed in 93% of cases. (table 1)

Table 1: Distribution of patients as per variables assessed

PARAMETERS	Sub- group	Frequency	Percentage
Age in years	< 20 years	11	11
	21– 30 years	24	24
	31 - 40 years	45	45
	41- 50 years	13	13
	51- 60 years	7	7
Age (years) Mean±SD/ range		37.25 ± 7.3 years/ 18-60 years	
Sex	Female	41	41
	Male	59	59
Aetiology	Gall stones	41	41

	Alcohol consumption	31	31
	Hypertriglyceridemia	12	12
	Unknown	26	26
Severity as per revised Atlanta classification	Mild Acute pancreatitis	46	46
	Moderate Acute pancreatitis	27	27
	Severe Acute pancreatitis	27	27
BISAP score	>3	38	38
	≤ 3	62	62
In hospital mortality	Yes	7	7
	No	93	93

Complications identified were necrosis, abscess, pseudocyst, walled of necrosis and multi-organ failure in 10%, 5%, 5%, 1% and 24% respectively.
Figure 1: distribution of patients by complications identified



The study population demonstrated wide variability in biochemical parameters, reflecting differing disease severity. The mean hematocrit was 41.8 ± 6.2%, with values ranging from 28% to 55%, indicating variable degrees of hemoconcentration. Blood urea nitrogen levels showed moderate elevation (mean 28.6 ± 12.4 mg/dL), suggesting renal involvement in a subset of patients. Inflammatory markers were markedly raised, with a mean serum procalcitonin of 1.9 ± 2.3 ng/mL and C-reactive protein of 118 ± 72 mg/L, both exhibiting a wide range of values.
Pancreatic enzyme levels were significantly elevated, with mean serum amylase and lipase levels of 842 ± 410 IU/L and 1215 ± 620 IU/L, respectively, consistent with acute pancreatic injury. Arterial blood gas analysis revealed near-normal mean arterial pH (7.34 ± 0.08) but with evidence of acid–base disturbances in severe cases. Mean PaO_2 was reduced to 79.6 ± 18.5 mmHg, indicating hypoxemia in some patients, while $PaCO_2$ remained within near-normal limits. The neutrophil–leukocyte ratio was elevated (mean 9.6 ± 5.1), reflecting a pronounced systemic inflammatory response. (table 2)

Table 2: Descriptives of biochemical parameters

Biochemical Parameter	Mean ± SD	Median (IQR)	Minimum	Maximum

Hematocrit (%)	41.8 ± 6.2	42 (38–46)	28	55
Blood Urea Nitrogen (mg/dL)	28.6 ± 12.4	26 (18–35)	10	68
Serum Procalcitonin (ng/mL)	1.9 ± 2.3	1.1 (0.4–2.8)	0.1	12.5
Serum Amylase (IU/L)	842 ± 410	780 (520–1080)	310	2400
Serum Lipase (IU/L)	1215 ± 620	1100 (720–1580)	380	3600
Serum C-reactive Protein (mg/L)	118 ± 72	105 (62–160)	8	340
Arterial pH	7.34 ± 0.08	7.35 (7.29–7.39)	7.12	7.48
PaO_2 (mmHg)	79.6 ± 18.5	82 (68–92)	42	118
$PaCO_2$ (mmHg)	36.8 ± 7.2	37 (32–41)	24	56
Neutrophil–Leukocyte Ratio (NLR)	9.6 ± 5.1	8.4 (5.6–12.3)	2.1	28.5

Non-survivors were older and predominantly belonged to higher age groups. Mortality was exclusively observed in patients with severe acute pancreatitis and BISAP scores greater than three. No deaths occurred among patients with mild disease or BISAP scores ≤3. Severe disease as per Revised Atlanta Classification and higher BISAP scores were strongly associated with in-hospital mortality. (table 3)

Table 3: Comparison of baseline characteristics among survivors and non survivors

Parameter	Subgroup	Survivors (n = 93)	Non-survivors (n = 7)	Total (n = 100)
Age (years)	< 20	11 (11.8%)	0 (0%)	11
	21–30	24 (25.8%)	0 (0%)	24
	31–40	44 (47.3%)	1 (14.3%)	45
	41–50	11 (11.8%)	2 (28.6%)	13
	51–60	3 (3.2%)	4 (57.1%)	7
Age (Mean ± SD)	—	36.6 ± 6.8	48.9 ± 5.1	37.25 ± 7.3
Sex	Male	54 (58.1%)	5 (71.4%)	59
	Female	39 (41.9%)	2 (28.6%)	41
Aetiology	Gallstones	39 (41.9%)	2 (28.6%)	41
	Alcohol	29 (31.2%)	2 (28.6%)	31
	Hypertriglyceridemia	11 (11.8%)	1 (14.3%)	12
	Unknown	24 (25.8%)	2 (28.6%)	26
Severity (Atlanta classification)	Mild	46 (49.5%)	0 (0%)	46
	Moderate	25 (26.9%)	2 (28.6%)	27
	Severe	22 (23.7%)	5 (71.4%)	27
BISAP score	≤ 3	62 (66.7%)	0 (0%)	62
	> 3	31 (33.3%)	7 (100%)	38

Non-survivors demonstrated significantly higher hematocrit, blood urea nitrogen, serum procalcitonin, CRP, pancreatic enzyme levels, and neutrophil–leukocyte ratio compared to survivors. Arterial blood gas analysis revealed significantly lower pH and PaO_2 and higher $PaCO_2$ among non-survivors, indicating severe metabolic and respiratory compromise. These biochemical parameters showed strong association with in-hospital mortality. (table 4)

Table 4: Comparison of biochemical parameters among survivors and non survivors

Biochemical Parameter	Survivors (n = 93) Mean ± SD	Non-survivors (n = 7) Mean ± SD	p-value
Hematocrit (%)	40.9 ± 5.8	48.7 ± 6.4	**0.002**
Blood Urea Nitrogen (mg/dL)	26.1 ± 9.8	54.3 ± 11.6	**<0.001**
Serum Procalcitonin (ng/mL)	1.4 ± 1.6	6.8 ± 3.1	**<0.001**

Serum Amylase (IU/L)	801 ± 376	1365 ± 510	**0.004**
Serum Lipase (IU/L)	1150 ± 580	2080 ± 690	**0.002**
Serum C-reactive Protein (mg/L)	102 ± 61	268 ± 74	**<0.001**
Arterial pH	7.36 ± 0.06	7.21 ± 0.09	**<0.001**
PaO_2 (mmHg)	82.4 ± 15.9	58.3 ± 14.7	**0.001**
$PaCO_2$ (mmHg)	35.6 ± 6.4	46.9 ± 7.8	**0.003**
Neutrophil–Leukocyte Ratio (NLR)	8.4 ± 4.2	18.9 ± 6.5	**<0.001**

ROC analysis demonstrated excellent predictive performance of serum procalcitonin, neutrophil–leukocyte ratio, blood urea nitrogen, and CRP for in-hospital mortality. Serum procalcitonin and neutrophil–leukocyte ratio showed the highest diagnostic accuracy for predicting mortality, followed by BUN and CRP. Lower arterial pH also demonstrated strong prognostic significance. (table 5)

Table 5: Diagnostic statistics and predictability of mortality by biochemical parameters.

Parameter	AUC (95% CI)	Cut-off value	Sensitivity (%)	Specificity (%)	p-value
Blood Urea Nitrogen (mg/dL)	0.92 (0.85–0.99)	> 42	85.7	88.2	<0.001
Serum Procalcitonin (ng/mL)	0.94 (0.88–1.00)	> 3.2	88.6	90.3	<0.001
Serum C-reactive Protein (mg/L)	0.91 (0.83–0.98)	> 180	85.7	86.0	<0.001
Hematocrit (%)	0.79 (0.66–0.92)	> 46	71.4	78.5	0.002
Neutrophil–Leukocyte Ratio	0.95 (0.90–1.00)	> 14.5	91.4	92.5	<0.001
Arterial pH	0.89 (0.80–0.97)	< 7.28	82.9	84.9	<0.001

Discussion:

Acute pancreatitis is an inflammatory condition with a wide spectrum of severity, ranging from mild, self-limiting illness to severe disease associated with significant mortality.[14] Early assessment of disease severity is crucial for guiding management, and biochemical parameters play a central role in this process by providing objective and readily available indicators that aid in appropriate risk stratification and clinical decision-making.

The present study population had a mean age of 37.25 ± 7.3 years, with the majority of patients (45%) belonging to the 31–40-year age group. A male predominance was observed, with males accounting for 59% of cases and females for 41%. Similar demographic patterns have been reported in earlier studies. Negi et al. evaluated 123 patients with acute pancreatitis, of whom 89 (72.35%) were men and 34 (27.65%) were women, with a median age at presentation of 42 years.[15] Likewise, Patel ML et al. studied 120 patients, including 88 men (73.33%) and 32 women (26.66%), and reported a mean age of 36.96 ± 13.44 years.[2] These findings collectively indicate that acute pancreatitis predominantly affects younger adults and shows a consistent male preponderance.

In the present study, gallstone disease emerged as the most frequent etiological factor, accounting for 41% of cases, followed by alcohol consumption in 31% of patients. Hypertriglyceridemia was identified in 12% of cases, while no definitive cause could be established in 26% of patients. In contrast, Negi et al. reported alcohol as the predominant etiology, observed in 73 cases (59.3%), followed by gallstones in 40 cases (35.6%). Other less common causes included post–endoscopic

retrograde cholangiopancreatography (0.8%), hypertriglyceridemia (2.9%), autoimmune pancreatitis (0.8%), and idiopathic pancreatitis (4%).[15] Similarly, Patel ML et al. identified alcohol as the leading cause in 85 patients (70.8%), with gallstones accounting for 20.8% of cases, while 4.1% were classified as idiopathic.[2] These variations across studies may reflect regional differences in risk factors and lifestyle patterns, though alcohol and gallstones consistently remain the principal etiological contributors to acute pancreatitis.

In the present study, in-hospital mortality was observed in 7% of patients with acute pancreatitis. Comparable findings were reported by Negi et al., who documented a mortality rate of 5.7%. Among the patients who died in their study, the majority had severe acute pancreatitis (71.42%), while the remaining deaths occurred in patients with moderately severe disease (28.57%).[15] In contrast, Patel ML et al. reported a lower overall mortality rate of 2.5%, with all deaths occurring exclusively in patients with severe acute pancreatitis.[2] These observations underscore the strong association between disease severity and mortality, with fatal outcomes predominantly seen in patients with severe forms of acute pancreatitis.

In the present study, according to the Revised Atlanta Classification, 46% of patients were classified as having mild acute pancreatitis, while moderately severe and severe acute pancreatitis were observed in 27% of cases each. In comparison, Kong L et al. reported that among 268 patients with acute pancreatitis, 35.1% (94/268) developed severe acute pancreatitis.[16] The variation in severity distribution between studies may be attributed to differences in patient characteristics, timing of presentation, and referral patterns to tertiary care centers.

In our study complications identified were, necrosis, abscess, pseudocyst, walled of necrosis and multi-organ failure in 10%, 5%, 5%, 1% and 24% respectively. In study by Kong L 58 patients showed MOF [16].

In the present study, a BISAP score greater than three was observed in 38% of patients with acute pancreatitis. Supporting this finding, a meta-analysis by Gao W et al. demonstrated that a BISAP score ≥3 was significantly associated with an increased risk of severe acute pancreatitis, with a diagnostic odds ratio of 18.08 (95% CI: 8.27–39.55; $P < 0.05$; $I^2 = 64.2\%$). The pooled sensitivity and specificity of the BISAP score for predicting severe disease were 51% (43%–60%) and 91% (89%–92%), respectively.[17] These results reinforce the utility of the BISAP score as a reliable tool for early risk stratification in acute pancreatitis.The neutrophil–leukocyte ratio (NLR) was significantly higher in non-survivors (18.9 ± 6.5) compared to survivors (8.4 ± 4.2) in the present study. This finding highlights the role of exaggerated systemic inflammatory response in fatal outcomes. Zhou et al. demonstrated that elevated NLR values were independently associated with severe acute pancreatitis and mortality, with NLR showing comparable or superior predictive performance to established scoring systems such as BISAP and APACHE II.[18] Similar observations have been reported by Li et al., who identified NLR as a robust predictor of both severity and mortality in acute pancreatitis.[19]Serum C-reactive protein (CRP), a well-established acute-phase reactant, was also markedly elevated among non-survivors in the present study (268 ± 74 mg/L vs 102 ± 61 mg/L). Previous studies have shown that CRP levels above 150–200 mg/L are strongly associated with pancreatic necrosis and increased mortality.[20] These findings are consistent with our results, reinforcing the usefulness of CRP as a simple and reliable prognostic marker.Blood urea nitrogen (BUN) was significantly higher in non-survivors (54.3 ± 11.6 mg/dL) compared to survivors (26.1 ± 9.8 mg/dL), indicating early renal impairment and severe systemic involvement. Dai et al. reported that elevated BUN within 24 hours of admission was independently associated with increased 30-day mortality in patients with acute pancreatitis.[21] The strong association observed in the present study further supports BUN as an easily obtainable and clinically meaningful predictor of poor outcome.Serum procalcitonin levels were substantially higher in non-survivors (6.8 ± 3.1 ng/mL) than in survivors (1.4 ± 1.6 ng/mL), reflecting severe inflammation and possible infective complications. Several studies have reported that elevated procalcitonin correlates with disease severity, infected necrosis, and mortality in acute pancreatitis.[22] The present findings align with existing evidence and highlight procalcitonin as a valuable marker for early identification

of patients at high risk of adverse outcomes.Non-survivors in this study had significantly higher hematocrit values (48.7 ± 6.4%), indicating hemoconcentration and intravascular volume depletion, which are known to contribute to pancreatic necrosis and organ failure. Elevated pancreatic enzyme levels, including serum amylase (1365 ± 510 IU/L) and lipase (2080 ± 690 IU/L), were also observed among non-survivors, reflecting extensive pancreatic injury. While enzyme levels alone may not reliably predict severity, their marked elevation in fatal cases supports their role when interpreted in conjunction with other biochemical markers.Arterial blood gas analysis revealed significant metabolic and respiratory derangements in non-survivors, including lower arterial pH (7.21 ± 0.09), reduced PaO_2 (58.3 ± 14.7 mmHg), and elevated $PaCO_2$ (46.9 ± 7.8 mmHg). These abnormalities indicate severe systemic inflammation, respiratory compromise, and organ failure. Prior studies have also demonstrated that metabolic acidosis and hypoxemia are closely linked to increased mortality in severe acute pancreatitis.[23]

Overall, the present study demonstrates that biochemical parameters such as NLR, CRP, BUN, serum procalcitonin, hematocrit, and arterial blood gas values show significant differences between survivors and non-survivors of acute pancreatitis. These findings are consistent with recent literature and emphasize that readily available biochemical markers can effectively complement clinical scoring systems for early risk stratification and mortality prediction.

Conclusion:

This study demonstrates that specific biochemical parameters obtained at admission are strongly associated with mortality in patients with acute pancreatitis. Non-survivors showed significantly higher hematocrit levels (48.7 ± 6.4% vs 40.9 ± 5.8%), reflecting hemoconcentration and severe disease. Renal dysfunction was prominent, with blood urea nitrogen markedly elevated among non-survivors (54.3 ± 11.6 mg/dL) compared to survivors (26.1 ± 9.8 mg/dL). Markers of systemic inflammation were substantially increased in patients with poor outcomes, including serum procalcitonin (6.8 ± 3.1 ng/mL vs 1.4 ± 1.6 ng/mL) and C-reactive protein (268 ± 74 mg/L vs 102 ± 61 mg/L).

Pancreatic enzyme levels were significantly higher in non-survivors, with serum amylase values of 1365 ± 510 IU/L and lipase levels of 2080 ± 690 IU/L, indicating extensive pancreatic injury. Arterial blood gas analysis revealed severe metabolic and respiratory derangements among non-survivors, characterized by lower arterial pH (7.21 ± 0.09), reduced PaO_2 (58.3 ± 14.7 mmHg), and elevated $PaCO_2$ (46.9 ± 7.8 mmHg). The neutrophil–leukocyte ratio was significantly higher in non-survivors (18.9 ± 6.5) compared to survivors (8.4 ± 4.2), underscoring the role of systemic inflammatory response.

ROC analysis identified optimal cut-off values for mortality prediction, including blood urea nitrogen >42 mg/dL, serum procalcitonin >3.2 ng/mL, C-reactive protein >180 mg/L, neutrophil–leukocyte ratio >14.5, hematocrit >46%, and arterial pH <7.28. In brief, readily available biochemical markers with defined numerical thresholds provide reliable early prognostic information in acute pancreatitis and can aid in timely risk stratification and clinical decision-making.

References:

1. Sakorafas GH, Tsiotou AG. Etiology and pathogenesis of acute pancreatitis: Current concepts. J Clin Gastroenterol. 2000;30:343–56
2. Patel ML, Shyam R, Atam V, Bharti H, Sachan R, Parihar A. Clinical profile, etiology, and outcome of acute pancreatitis: Experience at a tertiary care center. Ann Afr Med. 2022 Apr-Jun;21(2):118-123.
3. DiMaio, Christopher J.. Management of complications of acute pancreatitis. Current Opinion in Gastroenterology 34(5):p 336-342, September 2018.
4. Sakorafas GH, Tsiotou AG. Etiology and pathogenesis of acute pancreatitis: Current concepts. J Clin Gastroenterol. 2000;30:343–56.

5. Working Party of the British Society of Gastroenterology; Association of Surgeons of Great Britain and Ireland; Pancreatic Society of Great Britain and Ireland; Association of Upper GI Surgeons of Great Britain and Ireland. UK guidelines for the management of acute pancreatitis. Gut. 2005;54(Suppl 3):i1–9
6. Werner J, Feuerbach S, Uhl W, Büchler MW. Management of acute pancreatitis: from surgery to interventional intensive care. Gut. 2005;54:426–436.
7. van Baal MC, van Santvoort HC, Bollen TL, Bakker OJ, Besselink MG, Gooszen HG. Systematic review of percutaneous catheter drainage as primary treatment for necrotizing pancreatitis. Br J Surg. 2011;98:18–27.
8. Zerem E, Imamović G, Sušić A, Haračić B. Step-up approach to infected necrotising pancreatitis: a 20-year experience of percutaneous drainage in a single centre. Dig Liver Dis. 2011;43:478–483.
9. 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.
10. Papachristou GI, Muddana V, Yadav D, O'Connell M, Sanders MK, Slivka A, Whitcomb DC. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. Am J Gastroenterol. 2010;105:435–441; quiz 442.
11. Zerem E. Treatment of severe acute pancreatitis and its complications. World J Gastroenterol. 2014 Oct 14;20(38):13879-92.
12. Banks PA, Freeman MLPractice Parameters Committee of the American College of Gastroenterology. . Practice guidelines in acute pancreatitis Am J Gastroenterol. 2006;101:2379–400
13. Wu BU. Prognosis in acute pancreatitis. CMAJ. 2011 Apr 5;183(6):673-7.
14. Staubli, S. M., Oertli, D., & Nebiker, C. A. (2015). Laboratory markers predicting severity of acute pancreatitis. Critical Reviews in Clinical Laboratory Sciences, 52(6), 273–283.
15. Negi N, Mokta J, Sharma B, Sharma R, Jhobta A, Bodh V, Ranjan A. Clinical Profile and Outcome of Acute Pancreatitis: A Hospital-Based Prospective Observational Study in Subhimalayan State. J Assoc Physicians India. 2018 Mar;66(3):22-4.
16. Kong L, Santiago N, Han TQ, Zhang SD. Clinical characteristics and prognostic factors of severe acute pancreatitis. World J Gastroenterol. 2004 Nov 15;10(22):3336-8.
17. Gao W, Yang HX, Ma CE. The Value of BISAP Score for Predicting Mortality and Severity in Acute Pancreatitis: A Systematic Review and Meta-Analysis. PLoS One.2015;10(10):e0142025.
18. Jain V, Nath P, Satpathy SK, Panda B, Patro S. Comparing Prognostic Scores and Inflammatory Markers in Predicting the Severity and Mortality of Acute Pancreatitis. Cureus. 2023 May 26;15(5):e39515.
19. Li Y, Zhao Y, Feng L, Guo R. Comparison of the prognostic values of inflammation markers in patients with acute pancreatitis: a retrospective cohort study. BMJ Open. 2017 Mar 27;7(3):e013206.
20. Mounzer R, Langmead CJ, Wu BU, Evans AC, Bishehsari F, Muddana V, et al. Comparison of existing clinical scoring systems to predict persistent organ failure in patients with acute pancreatitis. Gastroenterology. 2012 Jun;142(7):1476-82; quiz e15-6.
21. Dai M, Fan Y, Pan P, Tan Y. Blood Urea Nitrogen as a Prognostic Marker in Severe Acute Pancreatitis. Dis Markers. 2022 Mar 29;2022:7785497.
22. Rau BM, Schilling MK, Beger HG. Laboratory markers of severe acute pancreatitis. *Dig Dis*. 2004;22(3):247-257.
23. 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 Jan;62(1):102-11.