



PREDICTING SEVERE ACUTE PANCREATITIS THROUGH BLOOD UREA NITROGEN MEASUREMENT: EVALUATION OF TIMING AND DIAGNOSTIC ACCURACY

Dr. Usha Rani

Associate Professor, Geetanjali Medical College Udaipur *Corresponding Auhtor

Dr. Mahendra Kumar Jalthania

Professor, RNT Medical College Udaipur

Dr. Manjeet Sonwal

Senior Resident, RNT Medical College Udaipur

ABSTRACT

Background: Acute pancreatitis is usually self-limiting; however, 10–30% of patients develop severe disease associated with high morbidity and mortality. Early prediction of disease severity is essential for timely intervention. Blood urea nitrogen (BUN) is a simple, inexpensive, and readily available biomarker that may predict severe acute pancreatitis, although the optimal timing and cutoff values remain uncertain. **Methods:** During the study period, a total of 80 patients were included, and their clinical details were recorded in a predesigned proforma. **Results:** BUN levels at admission and after 24 hours significantly predicted severe acute pancreatitis, with the 24-hour BUN demonstrating better predictive accuracy (AUC: 0.80 vs. 0.75). Cutoff values of 6.1 mmol/L at admission and 8.3 mmol/L after 24 hours facilitated early risk stratification. Other laboratory markers showed lower predictive value, while biliary etiology and gender were not significantly associated with disease severity. **Conclusion:** BUN is a simple, cost-effective, and widely available biomarker for the early prediction of severe acute pancreatitis. Identifying high-risk patients using established cutoff values enables timely intervention, individualized management, and efficient utilization of intensive care resources, potentially improving clinical outcomes and reducing disease-related complications.

KEYWORDS : Blood Urea Nitrogen (BUN), Severe Acute Pancreatitis (SAP).

A. INTRODUCTION

Acute pancreatitis (AP) is a common and potentially life-threatening condition characterized by inflammation of the pancreas. Although the majority of cases are mild and self-limiting, approximately 10–30% of patients develop severe acute pancreatitis (SAP), which is associated with substantial morbidity and a high risk of mortality.⁽¹⁾ The in-hospital mortality rate for SAP can reach up to 30%, highlighting the critical need for early identification and intervention⁽²⁾. Mild cases usually resolve within a few days with supportive care. In contrast, SAP progresses rapidly, leading to systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), and potentially death.⁽³⁾ Early and accurate assessment of disease severity is crucial in the management of AP. Identifying patients at risk of developing SAP allows for timely therapeutic interventions, which can improve clinical outcomes and reduce mortality. Traditionally, various scoring systems and biomarkers have been used to predict the severity of AP. These include the Acute Physiology and Chronic Health Evaluation II (APACHE II), Ranson's criteria, and the Bedside Index for Severity in Acute Pancreatitis (BISAP).⁽⁴⁾ Although these scoring systems provide valuable prognostic information, they have several limitations. For instance, APACHE II is not specific to pancreatitis and requires a large number of variables, making it complex and time-consuming to calculate.⁽⁵⁾ Ranson's criteria and the BISAP score, although simpler, still require multiple clinical and laboratory parameters that may not be immediately available.⁽⁶⁾ Blood urea nitrogen (BUN), a byproduct of protein metabolism, is excreted by the kidneys and reflects renal function and intravascular volume status. Elevated BUN levels at admission and changes during the first 24 hours of hospitalization have been associated with poor outcomes in AP.⁽⁷⁾ The Revised Atlanta Classification defines SAP as AP associated with persistent organ failure lasting more than 48 hours. According to the Revised Atlanta Classification, organ failure includes respiratory, cardiovascular, and renal failure. Persistent organ failure is a critical determinant of mortality in AP, and early identification of patients at risk of persistent organ failure is essential for improving clinical outcomes.⁽⁸⁾ The utility of BUN as a prognostic marker in AP is further supported by its association with renal dysfunction and intravascular volume depletion,

which are common features in the early stages of SAP. Renal dysfunction in AP can result from various mechanisms, including hypovolemia, systemic inflammation, and direct renal injury caused by pancreatic enzymes and inflammatory mediators.⁽⁹⁾ In addition to its prognostic value, BUN measurement is advantageous because of its simplicity and rapid availability. However, several limitations and challenges remain. One major limitation is the variability in the cutoff values for BUN used across different studies. Standardizing these cutoff values is essential for the consistent and reliable use of BUN in clinical practice. Another challenge is the potential influence of confounding factors on BUN levels. Factors such as baseline renal function, hydration status, and comorbidities can affect BUN levels, thereby influencing its prognostic value in AP. Therefore, BUN levels should be interpreted in conjunction with other clinical and laboratory parameters. The need for the present study arises from existing gaps in the literature regarding the optimal timing and cutoff values for BUN measurement in predicting SAP.⁽¹⁰⁾

B. MATERIALS AND METHODS

A hospital-based observational study was conducted at RNT Medical College, Udaipur, involving 80 patients diagnosed with acute pancreatitis.

Inclusion Criteria

- Patients admitted with a diagnosis of acute pancreatitis.
- Patients aged 18 years or older.
- Patients willing to participate and provide informed consent.

Exclusion Criteria

- Hemodynamically unstable patients at presentation.
- Patients with pre-existing comorbid conditions, such as ischemic heart disease, angina, or coronary artery disease (CAD).
- Patients with organ failure before data collection, including a history of cirrhosis, chronic kidney disease (creatinine clearance <40 mL/min), or chronic pulmonary disease.
- Pregnant patients or those meeting any of the above exclusion criteria.

Data Collection

A pre-tested, pre-designed proforma was used to collect data from the study population.

2013 Revised Atlanta Classification for Acute Pancreatitis Severity

- **Mild:** Absence of organ failure and local or systemic complications.
- **Moderately Severe:** Presence of local and/or systemic complications and/or transient organ failure (<48 hours).
- **Severe:** Persistent organ failure (>48 hours), as assessed using the Modified Marshall Scoring System⁽¹¹⁾.

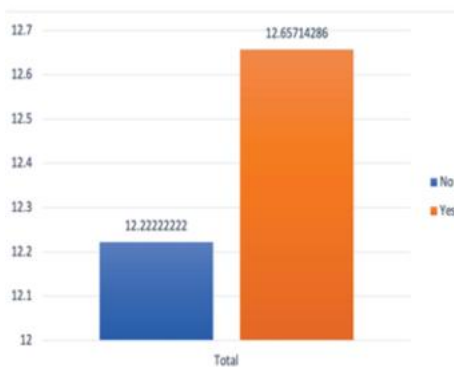
Local Complications

- Peripancreatic fluid collections.
- Pancreatic and peripancreatic necrosis (sterile or infected).
- Pancreatic pseudocyst and walled-off necrosis (sterile or infected).
- Gastric outlet obstruction.
- Splenic and portal vein thrombosis.
- Colonic necrosis⁽¹²⁾.

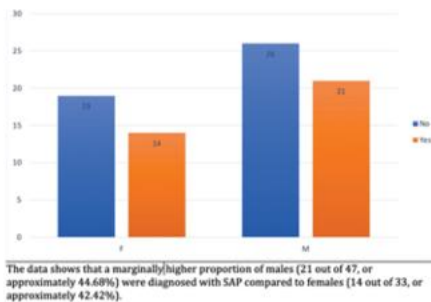
C. OBSERVATIONS AND RESULTS

During study period total of 80 patients were included and their details were filled in proforma. After study period a master chart was made and then statistical test were applied i.e. chi-square and independent t-test was applied.

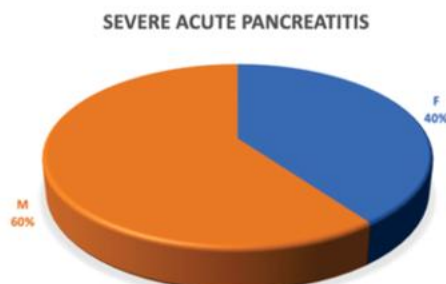
Average length of hospital stay



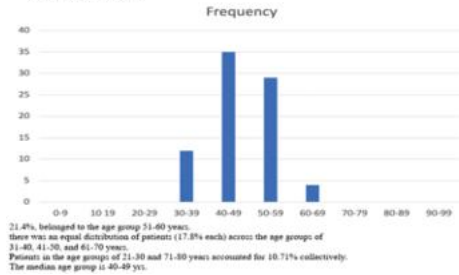
Gender distribution



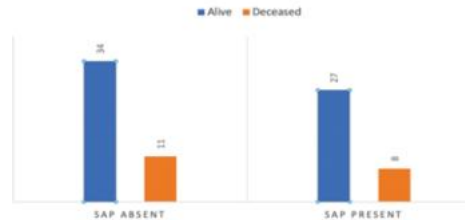
Gender distribution



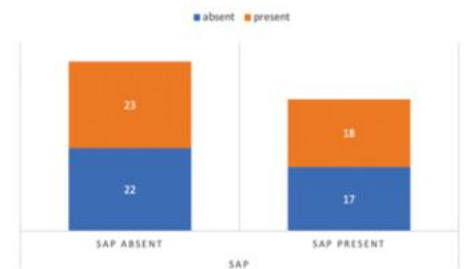
Age Distribution



MORTALITY IN SAP



BILIARY ETIOLOGY

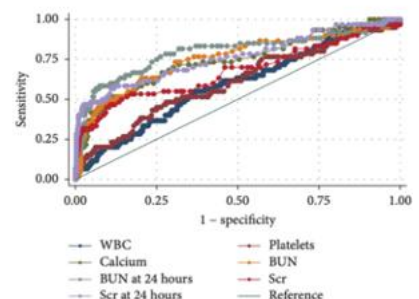


The data indicates a similar distribution of SAP presence between patients with and without biliary etiology. Approximately 44% of patients with biliary etiology developed SAP, which is almost equivalent to the 43.6% in patients without biliary etiology, suggesting that biliary etiology does not significantly influence the development of SAP in this cohort.

Table 1: Univariate analysis of predictive factors of SAP in 80 patients

	No SAP	SAP	P-Value
WBC (10 ³ /l)	11.04 (9 - 13)	11.04 (9 - 13)	0.072
Hemoglobin (g/l)	15.49 (13 - 17)	15.23 (13 - 17)	0.3869
Platelet (10 ³ /l)	221.33 (180 - 250)	214.57 (180 - 250)	0.1211
Potassium (mmol/l)	4.45 (4.2 - 4.7)	4.53 (4.4 - 4.7)	0.0097
Sodium (mmol/l)	139.16 (136 - 142)	140.06 (137 - 142)	0.0339
Calcium (mmol/l)	9.08 (8.8 - 9.4)	9.06 (8.8 - 9.4)	0.7239
BU/N (mmol/l)	15.13 (12 - 18)	14.2 (12 - 18)	0.0142
BUN (24 h) (mmol/l)	25.1 (20-30)	24.03 (20 - 30)	0.0131
Scr (umol/L)	1.01 (0.7 - 1.3)	1.194 (0.95 - 1.3)	0.2696
Scr (24 h) (umol/L)	1.19 (0.95 - 1.71)	1.16 (0.95 - 1.71)	0.6146
S. Amylase (U/L)	373.5 (243-650)	369.31 (243 - 650)	0.8856
S. Lipase (U/L)	482.98 (294 - 800)	461.03 (294 - 800)	0.5278

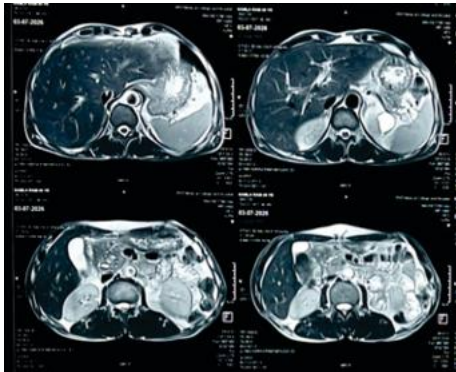
ROC curves of univariate variables



The predictive accuracy of various laboratory parameters for severe acute pancreatitis (SAP) was assessed using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) was calculated for each parameter. The AUC values for the different parameters at admission and after 24 hours were as follows:

- **WBC at Admission:** The AUC for WBC at admission was 0.59 ± 0.08 , indicating poor predictive accuracy for SAP.
- **Platelet Count at Admission:** The AUC for platelet count at admission was 0.61 ± 0.08 , also indicating limited predictive value.
- **Calcium at Admission:** The AUC for serum calcium at admission was 0.74 ± 0.08 , demonstrating moderate predictive accuracy for SAP.
- **BUN at Admission:** The AUC for BUN at admission was 0.75 ± 0.08 , indicating good predictive accuracy.
- **BUN After 24 Hours:** The AUC for BUN measured 24 hours after admission was 0.80 ± 0.07 , indicating excellent diagnostic accuracy among the individual predictors.
- **Serum Creatinine (SCr) at Admission:** The AUC for SCr at admission was 0.68 ± 0.09 , indicating moderate predictive value.
- **SCr After 24 Hours:** The AUC for SCr measured 24 hours after admission was 0.76 ± 0.08 , also indicating good predictive accuracy.

Both BUN at admission and BUN measured 24 hours after admission were identified as useful predictors of SAP, with AUC values greater than 0.70. Notably, BUN measured 24 hours after admission demonstrated the highest predictive performance, with an AUC of 0.80, indicating excellent diagnostic accuracy among the individual laboratory parameters.



Severe acute pancreatitis with early stage of pseudocyst formation

D. DISCUSSION

Our findings revealed that BUN levels measured both at admission and 24 hours after admission were significant predictors of severe acute pancreatitis (SAP), with the 24-hour BUN demonstrating superior diagnostic accuracy.

The AUC for BUN at admission was 0.75 ± 0.08 , whereas the AUC for BUN measured 24 hours after admission was 0.80 ± 0.07 . These findings suggest that BUN measurements can reliably predict the severity of acute pancreatitis.

The optimal BUN cutoff values identified were 6.1 mmol/L (17.1 mg/dL) at admission and 8.3 mmol/L (23.2 mg/dL) at 24 hours. These thresholds may facilitate the early identification and management of patients with SAP, potentially improving clinical outcomes and reducing mortality.

Other laboratory parameters, including WBC, platelet count, and serum creatinine (SCr), demonstrated moderate predictive value but did not achieve the predictive accuracy of BUN.

The study also demonstrated that biliary etiology and gender were not significantly associated with the development of SAP, emphasizing the importance of biochemical markers in the early prediction and prognostic assessment of disease severity.

Strengths

- **Comprehensive Analysis:** Multiple laboratory parameters (WBC, platelet count, calcium, BUN, and SCr) were evaluated at admission and 24 hours after admission. BUN was identified as a reliable predictor with high diagnostic accuracy.
- **Clinical Relevance:** The findings provide practical insights for the early risk stratification and management of patients with SAP using simple, cost-effective, and widely available laboratory tests.
- **Statistical Rigor:** The high AUC values observed for BUN indicate strong predictive performance.

Limitations

- **Sample Size:** The relatively small sample size may limit the generalizability of the findings to the broader population.
- **Observational Design:** The observational design limits the ability to establish a causal relationship between laboratory parameters and SAP outcomes. Larger prospective studies and randomized controlled trials are needed to validate these findings.
- **Variability and Confounding Factors:** Variability in laboratory measurements and the potential influence of underlying comorbidities, medication use, and nutritional status were not fully accounted for.
- **Focus on Individual Predictors:** Although BUN demonstrated excellent predictive value, combining multiple clinical and biochemical markers may further improve predictive accuracy. Future research should focus on developing comprehensive prognostic models.

E. CONCLUSION

Blood urea nitrogen (BUN) is a simple, cost-effective, and widely available biomarker for the early risk stratification of patients with acute pancreatitis. Early prediction of severe acute pancreatitis (SAP) enables timely intervention and appropriate clinical management.

By applying the identified cutoff values, clinicians can accurately identify high-risk patients and tailor management strategies accordingly. This approach may improve resource allocation in hospital settings by ensuring that intensive care resources are directed toward patients who are most likely to benefit, thereby potentially improving clinical outcomes.

F. REFERENCES

1. Mallo, G., Mazzone, E., Siriwardena, A. K., & Cuzzocrea, S. (2007). Role of tumor necrosis factor- α in acute pancreatitis: from biological basis to clinical evidence. *Shock*, 28(2), 130-140.
2. Talukdar, R., Nechutova, H., Clemens, M., & Vege, S. S. (2013). Could rising BUN predict the future development of infected pancreatic necrosis? *Pancreatology*, 13(4), 355-359.
3. Granger, J., & Remick, D. (2005). Acute pancreatitis: models, markers, and mediators. *Shock*, 24(Suppl 1), 45-51.
4. Chen, Z.-Q., Tang, Y.-Q., Zhang, Y., et al. (2004). Adenoviral transfer of human interleukin-10 gene in lethal pancreatitis. *World Journal of Gastroenterology*, 10(20), 3021-3025.
5. Rustgi, A. K. (2013). A historical perspective on clinical advances in pancreatic diseases. *Gastroenterology*, 144(6), 1249-1251.
6. Faisst, M., Wellner, U. F., Utzolino, S., Hopt, U. T., & Keck, T. (2010). Elevated blood urea nitrogen is an independent risk factor of prolonged intensive care unit stay due to acute necrotizing pancreatitis. *Journal of Critical Care*, 25(1), 105-111.
7. Wu, B. U., Johannes, R. S., Sun, X., Conwell, D. L., & Banks, P. A. (2009). Early changes in blood urea nitrogen predict mortality in acute pancreatitis. *Gastroenterology*, 137(1), 129-135.
8. Wu, B. U., Bakker, O. J., Papachristou, G. I., et al. (2011). Blood urea nitrogen in the early assessment of acute pancreatitis: an international validation study. *Archives of Internal Medicine*, 171(7), 669-676.
9. Kaya, E., Dervisoglu, A., & Polat, C. (2007). Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. *World Journal of Gastroenterology*, 13(22), 3090-3094.

10. Banks, P. A., Bollen, T. L., Dervenis, C., et al. (2013). Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut*, 62(1), 102-111.
11. Wu, B. U., Hwang, J. Q., Gardner, T. H., et al. (2011). Lactated Ringer's solution reduces systemic inflammation compared with saline in patients with acute pancreatitis. *Clinical Gastroenterology and Hepatology*, 9(8), 710.e1-717.e1.
12. Kaya, E., Dervisoglu, A., & Polat, C. (2007). Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. *World Journal of Gastroenterology*, 13(22), 3090-3094.
13. Wu, B. U., Johannes, R. S., Sun, X., Tabak, Y., Conwell, D. L., & Banks, P. A. (2008). The early prediction of mortality in acute pancreatitis: a large population-based study. *Gut*, 57(12), 1698-1703.
14. Wu, B. U., Papachristou, G. I., & Repas, K. (2014). Blood urea nitrogen in the early assessment of acute pancreatitis: an international validation study. *Archives of Internal Medicine*, 174(8), 1321-1329.
15. Altman, D. G., & Bland, J. M. (1994). Diagnostic tests 2: predictive values. *BMJ*, 309(6947), 102.
16. Dickerson, R. N., Vehe, K. L., Mullen, J. L., & Feurer, I. D. (1991). Resting energy expenditure in patients with pancreatitis. *Critical Care Medicine*, 19(4), 484-490.