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Evaluation of Prognostic Factors to Predict the Severity of Acute Pancreatitis

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ABSTRACT

Background: Acute pancreatitis (AP) is a critical inflammatory disorder of the pancreas, commonly characterized by intense abdominal pain accompanied by nausea and vomiting. Laboratory markers that might help predict severity earlier include basic blood tests like white blood cell counts, various cell ratios, and other markers such as CRP.

Aim: To evaluate predictive factors for AP severity in cases admitted to the Emergency Hospital.

Patients and Methods: This prospective observational cohort investigation encompassed adult cases met the revised Atlanta Classification criteria for AP. Data collected included demographic information, clinical history, and laboratory tests (CBC, liver and kidney function, serum biomarkers). Cases received standardized management, including pain control, fluid resuscitation, nutritional support, and antibiotics as necessary. Clinical progression was assessed over a follow-up duration of up to six months.

Results: WBCs, neutrophil levels, and CRP were substantially elevated in severe group at both baseline and follow-up. Additionally, BUN, INR, creatinine, amylase, lipase, serum glucose, and NCI demonstrated substantially higher values in cases with severe disease. Quantitative assessment of pancreatic necrosis revealed a markedly greater incidence of necrotizing AP and necrotic involvement exceeding 30% in severe group. Occurrence of multi-organ failure (MOF) was also substantially higher among these cases. Furthermore, the duration of hospitalization was substantially prolonged in severe group. Both procalcitonin levels and NLR were significantly elevated in this cohort.

Conclusion: These results suggest that monitoring these markers, especially BUN, NCI, and NLR, could be instrumental in early identification and management of severe pancreatitis, potentially guiding more targeted and timely interventions.

Keyword: Acute Pancreatitis, Multiple Organ Failure, Blood Urea Nitrogen, Atlanta Classification.

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disorder involving the pancreatic parenchyma, commonly manifesting as severe abdominal pain accompanied by nausea and vomiting. Although most cases exhibit a mild, self-limiting trajectory, nearly 20% advance to a severe form of AP, marked by the development of multiple organ failure (MOF) and a systemic inflammatory response syndrome (SIRS). AP carries an overall mortality rate of approximately 5%, which can escalate to 25% in cases classified as severe. The development of persistent MOF and pancreatic necrosis represents the most significant prognostic indicators of poor clinical outcomes. Therefore, early identification of disease severity and progression is essential to guide timely and appropriate therapeutic interventions [1]. To aid in the early prediction of disease severity in AP, numerous prognostic scoring systems and classification models have been introduced. Among the most commonly employed are the revised Atlanta classification and the Bedside Index of Severity in Acute Pancreatitis (BISAP), both of which are extensively used to assess the clinical progression and severity of AP. The revised Atlanta criteria classify AP into three distinct forms: mild (MAP), moderately severe (MSAP), and severe (SAP) [2].

Commonly utilized prognostic tools in AP include Ranson's criteria, the Modified Glasgow Prognostic Score, the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and the Balthazar index. These scoring systems generally rely on a combination of clinical and laboratory indicators, which are assessed within the first 24 to 48 hours following hospital admission. As a result, the assessment of disease severity is often delayed until at least 48 hours post-admission, thereby limiting their applicability for early risk stratification at the time of presentation [3].

Owing to the complexity inherent in conventional prognostic scoring systems, extensive research has focused on the prognostic value of easily accessible laboratory markers and indices in evaluating disease severity and mortality risk in AP. Among the most thoroughly examined parameters are white blood cell (WBC) count, neutrophil count, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), hematocrit, blood urea nitrogen (BUN), creatinine, calcium, CRP, and procalcitonin [4]. Several of these laboratory markers have been incorporated into various prognostic scoring systems. However, neither individual laboratory parameters nor existing scoring models have demonstrated sufficient accuracy in reliably predicting the severity of AP or the development of MOF [5]. The initial 48 hours following symptom onset are critical for identifying cases at elevated risk of developing complications or mortality.

This early phase is pivotal for determining the intensity of therapeutic interventions, including fluid resuscitation, analgesia, and nutritional support. Consequently, it is essential to assess at the time of admission whether the case necessitates close monitoring or escalation of care through transfer to ICU [6].

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AIM OF WORK

This investigation aimed to evaluate predictive factors for AP severity such as: Serum lipase, serum amylase, WBCs, platelets, Hb, hematocrit, AST, ALT, serum albumin, serum bilirubin, INR, serum glucose, serum calcium, LDH, CRP, procalcitonin, NCI, NLR, PLR, Age, Sex, BMI, necrosis volume in cases admitted to the Emergency Hospital.

PATIENTS AND METHODS

This prospective observational cohort investigation was conducted over a one-year period, from July 2022 to July 2023, at the Emergency Hospital, Mansoura University. The study enrolled cases presenting to and admitted through the Emergency Department (ED) with acute abdominal pain and clinical suspicion of AP. Eligible participants were those who fulfilled the diagnostic criteria of the revised Atlanta Classification for AP and were over 18 years of age, irrespective of gender.

Methods:

All enrolled cases underwent comprehensive history taking, history of prior medical or surgical interventions, hospital admission details, initial diagnosis, and relevant past medical history.

All cases received management in accordance with standardized clinical guidelines, which included thorough clinical evaluation and monitoring of vital signs (heart rate, blood pressure, temperature, and respiratory rate), as well as assessment for pallor, cyanosis, jaundice, and lymphadenopathy. Supportive care involved appropriate fluid resuscitation, oxygen therapy, and analgesics for pain control. Enteral nutrition via nasojejunal or nasogastric tube was initiated when tolerated; otherwise, parenteral nutrition was administered. Antibiotic therapy was reserved for cases with confirmed infection. Organ support measures—including mechanical ventilation, vasoactive agents, and continuous renal replacement therapy—were implemented as clinically indicated. When indicated, interventional procedures—including percutaneous, endoscopic, or surgical techniques—were employed to target the underlying etiology of AP or to manage associated local complications.

AP diagnosis, as defined by the revised Atlanta Classification, requires fulfillment of at least two out of three criteria: elevated serum amylase and/or lipase levels exceeding three times the upper normal limit, typical radiologic features on abdominal imaging, and the presence of persistent abdominal pain [7].

The revised Atlanta classification categorizes AP into two distinct clinical phases: early and late. It further stratifies disease severity into three levels—mild, moderately severe, and severe. MAP, the most frequently encountered form, is marked by the absence of organ failure and local or systemic complications, with clinical resolution generally within the first week. MSAP is characterized by transient organ failure, the presence of local complications, or exacerbation of pre-existing comorbidities. SAP is defined by persistent organ failure lasting longer than 48 hours [8].

The severity of AP was followed up in these cases during hospitalization (till the outcome of the case: discharged or dead) at least for six months starting from admission, during the period of study from July 2022 to July 2023. It was also grounded in the criteria established by the revised Atlanta Classification [7].

Investigations included serum lipase, serum amylase, WBCs, platelets, Hb, hematocrit, AST, ALT, Serum Albumin, Serum Bilirubin, INR, serum glucose, serum calcium, LDH, CRP, Procalcitonin. Radiology included abdominal ultrasonography, abdominal computed tomography.

Ethical Considerations

Ethical approval for this investigation was obtained from the Institutional Ethics Committee of Mansoura University. Written informed consent was secured from all participants following a detailed explanation of the study objectives and the procedural aspects of the intervention. Each case was adequately informed about their involvement in the clinical research. Data were solely collected by the principal investigator, with the study conducted in accordance with the Declaration of Helsinki.

Statistical Analysis

Data were analyzed using SPSS software, version 26 (SPSS Inc., PASW Statistics for Windows, Version 26.0, Chicago, IL, USA). Categorical variables were summarized as frequencies and percentages, while continuous variables were expressed as median (minimum–maximum) for non-normally distributed data and as mean ± standard deviation (SD) for normally distributed variables. Normality of distribution was assessed using Kolmogorov–Smirnov test. Group comparisons for categorical data were conducted using Chi-square test, Monte Carlo test, or Fisher's exact test wherever appropriate. The Mann–Whitney U test was applied to compare two groups in the case of non-normally distributed variables. For normally distributed continuous data, comparisons between two independent groups were made using the Student's t-test. ROC curve analysis was employed to evaluate the diagnostic performance of continuous variables, including the determination of sensitivity, specificity, and optimal cutoff values. Statistical significance was considered at a p-value < 0.05.

RESULTS

The investigation enrolled 100 cases, 82 non-severe and 18 severe. Mean age was higher in severe cases. Sex distribution and BMI were similar across groups. Biliary causes were most common in both groups. No substantial variations were observed between groups in terms of age, sex, BMI, or etiology **(Table 1)**.

Table (1): Comparison of demographic characteristics and etiology between severe and non-severe cases

	Non-severe N=82	**Severe N=18**	**Test of significance**
Age/years Mean ±SD	38.57±13.14	44.06±13.95	t=1.58 p=0.116
Sex Male Female	39(47.6) 43(52.4)	10(55.6) 8(44.4)	χ^2=0.377 P=0.539
BMI (kg/m²) Mean ±SD	25.69±2.96	25.39±3.07	t=0.382 p=0.704
Etiology			
Biliary	56(68.3)	13(72.2)	χ2=0.107 P=0.744
Nonbiliary	10(12.2)	2(11.1)	χ2=0.016 P=0.898
Idiopathic	16(19.5)	3(16.7)	χ2=0.078 P=1.0

t: Student t test, χ^2=Chi-Square test

Table **(2)** shows that WBCs, neutrophils, and CRP were substantially higher in severe cases at both baseline and follow-up. Lymphocyte counts were markedly lower in the severe group at baseline and at follow-up. Hemoglobin and hematocrit levels were also elevated in severe cases. Platelet counts showed no substantial variation between groups at any time point. ALT levels were substantially lower in severe group relative to non-severe group. BUN, creatinine, and INR were markedly elevated in severe cases. No substantial variations were detected in AST, albumin, total bilirubin, LDH, or calcium levels. Amylase and lipase levels were substantially elevated in severe group relative to non-severe group. Serum glucose was also elevated in severe cases. The Necrosis, Complication, and Infection (NCI) score was substantially higher in severe cases.

Table (2): Laboratory parameters between studied groups

		Non-severe (N=82)	**Severe (N=18)**	**Test of significance**
WBC (×103/µL)	Baseline	12.98±3.23	18.87±3.73	t=5.45, p=0.001*
	At follow up	9.04±2.98	15.41±2.82	t=8.29, p=0.001*
Neutrophil (×103/µL)	Baseline	9.45±2.54	15.85±3.15	t=7.08, p=0.001*
	At follow up	6.60±1.73	12.34±1.83	t=8.47, p=0.001*
Lymphocyte (×103/µL)	Baseline	1.62±0.29	1.19±0.20	t=2.15, p=0.034*
	At follow up	1.264±0.23	0.904±0.25	t=3.37, p=0.001*
Platelet (×103/µL)	Baseline	248.09±65.85	245.81±61.14	t=0.135, p=0.893
	At follow up	198.89±7.41	188.5±49.39	t=0.712, p=0.478
Hemoglobin (g/dL)	Baseline	12.92±1.39	14.35±1.85	t=3.68, p<0.001*
Hematocrit (%)	Baseline	39.83±4.84	42.88±5.19	t=2.37, p=0.019*
CRP (mg/L)	Baseline	37.51±4.28	42.89±5.36	t=4.61, p<0.001*
	At follow up	92.42±21.04	151.40±18.76	t=10.96, p<0.001*
ALT (UL)		184.57±15.29	168.42±13.76	t=4.12, p<0.001*
AST (UL)		188.55±14.19	185.43±17.46	t=0.810, p=0.420
Albumin (g/dL)		3.88±0.65	3.63±0.61	t=1.54, p=0.128
Total bilirubin (mg/dL)		2.38±0.38	2.87±0.27	t=1.25, p=0.216
BUN (mg/dL)		14.88±1.03	17.49±1.21	t=9.44, p<0.001*
Creatinine (mg/dL)		0.81±0.04	1.09±0.19	t=12.47, p<0.001*
Calcium (mg/dL)		9.19±0.69	9.20±0.52	t=0.059, p=0.953
LDH (IUL)		409.21±213.11	493.29±70.92	t=1.44, p=0.153
INR		1.55±0.45	2.85±0.41	t=6.93, p=0.001*
Amylase (UL)		1358.69±8.46	1490.59±8.78	t=6.41, p<0.001*
Lipase (UL)		1440.96±41.45	1751.66±95.38	t=8.86, p<0.001*
Serum glucose (mg/dL)		126.25±27.73	144.24±22.78	t=2.56, p=0.012*
NCI		11.69±2.41	14.66±3.25	t=2.68, p=0.008*

t: Student t test, χ^2=Chi-Square test, Data expressed as mean±SD, *: Statistically significant.

Table **(3)** shows that local pancreatic complications differed substantially between non-severe and severe groups. Walled-off pancreatic necrosis and pseudocysts were observed only in severe cases. In contrast, non-severe cases showed various forms of fluid collections and necrosis, but not pseudocysts or walled-off necrosis. These findings highlight a higher frequency and severity of local complications in the severe group.

Table (3): Comparison of local complications among studied groups

Local complications	**Non-severe N=82 (%)**	**Severe N=18 (%)**	**Test of significance**
No	51(62.2)	0	MC=100
Walled off pancreatic necrosis	0	12(66.7)	P<0.001*
Pseudocyst	0	6(33.3)	
Fluid collection without necrosis	7(8.5)	0	
Fluid collection with necrosis	15(18.3)	0	
Acute necrotic collection	5(6.1)	0	
Acute fluid collection	4(4.9)	0	

Mc: Monte Carlo test *: Statistically significant

Table (4) shows that quantification of pancreatic necrosis by CT showed significantly higher rates in the severe group. Necrosis volume >30% was also more frequent in severe cases. MOF occurred exclusively in the severe group, with 38.9% of cases affected. None of the non-severe cases developed MOF, resulting in a highly significant difference. The length of hospital stay was substantially prolonged in severe group relative to non-severe group.

Table (4): Comparison of quantification of pancreatic necrosis by CT, presence of MOF and hospital stay among studied groups

Quantification pancreatic necrosis by CT	**Non-severe N=82**	**Severe N=18**	**Test of significance**
Necrotizing AP	21(25.6)	12(66.7)	$\chi2$=11.25, P<0.001*
Necrosis volume > 30%	8(9.8)	7(38.9)	$\chi2$=9.83, P=0.002*
Presence of MOF	0	7(38.9)	$\chi2$=34.28, P<0.001*
Hospital stay (days)	8.32±2.41	17.52±2.65	t=14.39, p=0.001*

χ^2=Chi-Square test *: Statistically significant

Both procalcitonin and NLR levels were substantially elevated at baseline and follow-up in severe group relative to non-severe group. While the baseline PLR did not differ markedly between groups, follow-up measurements revealed a notable increase in PLR within the severe group **(Table 5)**.

Table (5): Comparison of procalcitonin, NLR and PLR between studied groups

		Non-severe (N=82)	**Severe (N=18)**	**Test of significance**
PCT (ng/mL)	Baseline	1.52(0.07-18)	2.35(1.96-35)	Z=3.42, P=0.001*
	At follow up	1.4(0.07-23)	4.25(2.4-34)	Z=4.74, P=0.001*
NLR	Baseline	6.38(1.247-38.6)	12.34(6.29-53.71)	Z=4.29, P=0.001*
	At follow up	5.56(1.2-24.09)	13.83(6.78-32.04)	Z=5.86, P=0.001*
PLR	Baseline	158.85(34.8-733.9)	180.89(89.47-769.06)	Z=1.49, P=0.136
	At follow up	165.92(56.66-331.4)	207.86(109.91-559.18)	Z=2.15, P=0.03*

Data expressed as median (min-max), Z: Mann Whitney U test, *: Statistically significant.

Table **(6)** shows that BUN, NCI, and NLR showed significant validity in differentiating severe from non-severe pancreatitis cases. BUN had the highest AUC (0.953), with excellent sensitivity and specificity at a cutoff of 15.89. NLR also demonstrated strong diagnostic performance (AUC 0.943) with high specificity and good sensitivity at a cutoff of 10.77. NCI had moderate predictive value (AUC 0.702), with lower specificity and sensitivity at a cutoff of 12.25. Overall, BUN and NLR were the most reliable markers for predicting severe pancreatitis.

Table (6): Validity of BUN, NCI and NLR in differentiating severe from non-severe cases

Test Result Variable(s)	**Area under the curve (AUC)**	**P value**	**Asymptotic 95% Confidence Interval**		**Cut off point**	**Sensitivity %**	**Specificity %**
			Lower Bound	**Upper Bound**			
BUN (mg/dL)	**0.953**	**0.001***	**0.899**	**1.006**	**15.89**	**94.4**	**85.4**
NCI	**0.702**	**0.008***	**0.593**	**0.810**	**12.25**	**72.2**	**57.3**
NLR	**0.943**	**0.001***	**0.893**	**0.993**	**10.77**	**83.3**	**95.1**

DISCUSSION

AP is a dynamic and evolving condition, wherein cases initially presenting without organ failure may subsequently develop organ dysfunction during hospitalization [9]. Moreover, predicting the prognosis of more severe forms of AP (i.e., MSAP and SAP) at the time of admission remains challenging. This is largely due to the inability to accurately detect local complications through imaging in the early phase, as well as the frequent emergence of systemic complications—characteristic of MSAP and SAP—which often result from the deterioration of pre-existing comorbid conditions following hospital admission [10]. The factors most strongly associated with unfavorable outcomes include the development of pancreatic necrosis and pancreatic infection in conjunction with MOF, a combination that carries a mortality rate approaching 50% [11].

So, this investigation aimed to evaluate predictive factors for the severity of AP such as: Serum lipase, serum amylase, WBCs, platelets, Hb, hematocrit, AST, ALT, serum albumin, serum bilirubin, INR, serum glucose, serum calcium, LDH, CRP, procalcitonin, NCI, NLR, PLR, age, sex, BMI, necrosis volume in cases admitted to the Emergency Hospital. This was a prospective observational cohort study conducted at Emergency Hospital, Mansoura University, over the course of one year (July 2022 to July 2023), focusing on cases with suspected AP. The study included adults aged 18–60 years who met the criteria for AP according to the revised Atlanta Classification, while excluding those outside this age range. Data collected consisted of demographic details, clinical history, and laboratory results (such as CBC, liver and kidney function, and serum biomarkers). Cases received standardized care, including pain management, fluid resuscitation, nutritional support, and antibiotics when needed. AP severity was monitored using the revised Atlanta Classification, and the study evaluated outcomes, including complications and mortality. Diagnostic imaging, including abdominal ultrasound and CT scans, were performed. Cases were followed throughout their hospitalization for up to six months to monitor their clinical progression.

In the present study, an equal distribution between male and female cases was observed, yielding a male-to-female ratio of 1:1. In contrast, **Ghiță et al.** [12] conducted a study aimed at estimating the incidence, healthcare costs, and tobacco use among hospitalized AP cases in southern Romania, while also providing updated insights into the etiology, severity, outcomes, morphological features, and local complications of the disease. Their analysis, based on electronic health records of cases treated for AP at the Emergency University Hospital of Bucharest from 2015 to 2022, revealed that 68.88% (n = 652) of cases occurred in male cases.

While the incidence of AP is comparable between males and females, male sex has been associated with increased mortality. Moreover, men are more prone to experience recurrent episodes of AP, which may progress to chronic pancreatitis due to the replacement of necrotic areas with fibrotic tissue. As a result, the incidence of chronic pancreatitis is notably higher in men, with an estimated 12 cases per 100,000 compared to 6 cases per 100,000 in women. The most common causes of AP are gallstone-related pancreatitis and alcohol-induced pancreatitis, while the leading cause of chronic pancreatitis is alcohol-related [13].

In our study, WBC counts were notably higher in SAP group relative to non-severe group. This finding aligns with the results of a prospective study conducted by **Popa** [14] which aimed to identify biological markers predictive of AP severity. Over a four-year period (2007–2010), the study included 103 cases diagnosed with SAP and treated at a surgical clinic in Bucharest. The analysis revealed that cases with severe forms of the disease exhibited significantly elevated WBC counts compared to those with MAP, confirming the association between leukocytosis and disease severity.

Our study demonstrated that CRP levels were substantially elevated in SAP group compared to the non-severe group. This finding is in agreement with results reported by **Stirling *et al.*** [15] who investigated whether changes in CRP levels over time offer comparable or superior prognostic value relative to absolute CRP measurements in AP. Their study compared the interval change (ΔCRP) from admission to days 1, 2, and 3 against static CRP values. They observed that the mean ΔCRP was markedly greater in cases with SAP compared to those with mild disease at all time points, with the most pronounced difference occurring at 48 hours (29.5 mg/dL vs. 318.9 mg/dL), reflecting a 10.8-fold increase.

Furthermore, a study was led by **Imamura *et al*.** [16] to measure serum CRP levels in cases with AP, using a high-sensitivity CRP (hs-CRP) assay method. In their study, serum hs-CRP levels were measured in 20 cases diagnosed with AP. The findings revealed a significantly elevated mean hs-CRP level in cases with SAP (222,760 ± 32,197 ng/mL) compared to those with mild to moderate forms of the disease (22,798 ± 8,216 ng/mL), indicating a strong association between elevated hs-CRP and disease severity.

Our study demonstrated that serum glucose levels were markedly higher in cases with SAP relative to those in non-severe group. This observation is in harmony with findings of **Sun et al.** [17] who examined the relationship between serum glucose levels and the progression of AP. Their analysis included 153 cases, categorized as having mild (MAP, n = 130), moderately severe (MSAP, n = 4), or SAP (SAP, n = 19). The study concluded that both serum glucose concentrations and APACHE II scores were markedly elevated in the MSAP and SAP groups relative to MAP group,

supporting the association between hyperglycemia and increased disease severity.

During the early phase of SAP, numerous pro-inflammatory cytokines and mediators are activated, initiating a SIRS [18]. This response involves a complex network of factors operating through positive feedback mechanisms, which progressively amplify the inflammatory cascade. When this inflammatory burden exceeds the host's compensatory capacity, it may disseminate systemically, leading to widespread inflammatory injury and multi-organ dysfunction. **Bank et al.** [19] reported that elevated glucose levels can stimulate the release of inflammatory cytokines, thereby promoting a cascade of inflammatory events that, in some cases, may rapidly progress to multi-organ damage.

Previous literature has examined the association between serum glucose levels and clinical outcomes such as single-organ failure, MOF, ICU admission rates, and mortality. Findings suggest that elevated glucose levels may serve as a critical marker for multi-organ dysfunction and adverse prognosis. This underscores the strong association between hyperglycemia and the inflammatory response in AP, wherein heightened glucose levels may exacerbate disease progression by amplifying systemic inflammation. Therefore, hyperglycemia can be considered a valuable clinical indicator for assessing disease severity in cases with AP [20].

Our study demonstrated that BUN levels were significantly elevated in SAP group relative to non-severe group. This evidence is concordant with the results reported by **Dai *et al.*** [21] who aimed to identify early and reliable prognostic markers for SAP. Their study included cases diagnosed with SAP at Xiangya Hospital, Central South University, from April 2017 to May 2021, serving as the training cohort. An external validation cohort was established from June 2021 to February 2022. The authors observed that BUN levels were substantially higher among non-survivors relative to survivors, highlighting its prognostic value in assessing SAP severity and outcomes.

Beyond its association with pancreatic pathology, BUN has demonstrated prognostic utility as both an independent and integrated biomarker in various clinical contexts, including the incidence of acute cardiovascular and cerebrovascular events, mortality among critically ill cases, and outcomes in individuals with coronavirus disease 2019 (COVID-19). Despite this, the predictive capacity of BUN outside its conventional role in assessing renal function remains insufficiently understood. Several mechanisms may account for elevated BUN levels in these settings, including renal hypoperfusion resulting from inflammation-induced vascular permeability and interstitial fluid extravasation—hallmarks of the systemic inflammatory response—as well as direct chemical injury to renal tissue mediated by circulating activated enzymes, cytokines, and inflammatory mediators [22].

Our study identified a significantly higher incidence of MOF in cases with SAP compared to those with non-severe disease. This finding aligns with the results of a study conducted by ***Zhu et al.*** [23] , which examined the association between SAP and organ failure. In this retrospective analysis, clinical data from 74 SAP cases managed between January 1993 and December 2002 were evaluated. The investigators analyzed the relationship between organ failure and various clinical variables, including age, sex, etiology, extent of pancreatic necrosis, necrotic tissue infection, and mortality. Among the cohort, 47 cases (63.5%) developed organ failure, with 20 (27.0%) exhibiting MOF and 27 (36.5%) presenting with failure of a single organ system.

Organ failure is the leading cause of mortality in SAP. In the early stage of the disease, dysfunction of vital organs is predominantly mediated by SIRS, even in the absence of infection. In contrast, the later phase is often characterized by septic complications, wherein organ failure is typically induced by sepsis. As a result, organ failure is a common and serious complication in SAP. Reported incidence rates in prior studies range from 72% to 90.3%, with single organ failure documented in 24.7% to 37% of cases, and MOF in 35% to 65.6%. Among isolated organ failures, pulmonary dysfunction is most frequently encountered (39.1%–63%), followed by cardiovascular compromise (23%–37.7%), hepatic failure (20.7%), and renal failure (8.5%–13%) [24].

CONCLUSION

The findings of our study show that several clinical markers, including WBCs, neutrophils, CRP, procalcitonin, NLR, and BUN, are significantly elevated in cases with severe pancreatitis relative to those with non-severe cases. Severe pancreatitis was also associated with lower lymphocyte counts, increased local complications, MOF, and longer hospital stays. Key independent predictors of severe pancreatitis were identified, with BUN, NCI, and NLR showing high sensitivity and specificity for distinguishing severe cases. These results suggest that monitoring these markers, especially BUN, NCI, and NLR, could be instrumental in early identification and management of severe pancreatitis, potentially guiding more targeted and timely interventions.

LIMITATIONS

The limited sample size of 100 cases, in conjunction with the study's single-center design, may constrain the extrapolation of findings to broader and more diverse populations.

RECOMMENDATIONS

Future research should incorporate larger sample sizes to improve the external validity and generalizability of the findings. Conduct multi-center studies involving diverse geographical locations and populations to improve the generalizability of findings.

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