

## Original Article

# The diagnostic utility of mean platelet volume in acute pancreatitis: insights and inconsistencies

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**Abstract:** Background: Acute pancreatitis (AP) is a rapid inflammatory disorder of the pancreas that can range from moderate to severe, frequently linked to considerable morbidity and death. The prompt recognition of severe acute pancreatitis (SAP) is essential for appropriate care; yet, forecasting the severity of AP continues to be difficult. Mean Platelet Volume (MPV), a metric of platelet activity, has surfaced as a prospective biomarker for the severity of AP. This study sought to evaluate the effectiveness of MPV in forecasting illness severity in AP. Methodology: A randomized controlled trial was conducted at Yenepoya Medical College Hospital, involving 279 participants, including both healthy volunteers and AP patients. MPV levels were measured and analyzed in relation to disease severity, specifically focusing on the presence of pancreatic necrosis. Results: In comparison to healthy controls ( $8.88 \pm 0.97$  fL), the study's result showed that MPV levels were somewhat higher in AP patients ( $9.43 \pm 6.78$  fL), although there was not a significant statistical difference between the two groups ( $P > 0.05$ ). Nevertheless, it was revealed that individuals who had pancreatic necrosis had a significantly higher level of MPV ( $13.17 \pm 1.7$  fL) in comparison to those who did not have pancreatic necrosis ( $9.19 \pm 0.86$  fL), with a  $p$ -value of less than 0.05. A sub-optimal diagnostic effectiveness was established by the ROC analysis for MPV in predicting pancreatic necrosis, with an Area Under the Curve (AUC) value of 0.609. Conclusion: This study concluded that, although MPV levels were higher in AP patients, particularly those with pancreatic necrosis, the overall diagnostic performance of MPV was sub-optimal. The study highlights the need for additional research to better understand the role of MPV in assessing AP severity and to explore other potential biomarkers that could improve early risk stratification in clinical practice.

**Keywords:** Acute pancreatitis, pancreatic necrosis, mean platelet volume, diagnostic accuracy, biomarker

## Introduction

Acute pancreatitis (AP) is a disorder that causes rapid inflammation of the pancreas, resulting in considerable morbidity and mortality. AP has a wide range of clinical manifestation, ranging from moderate sickness that resolves on its own to a serious illness that poses a significant risk to the patient's life [1]. Severe acute pancreatitis (SAP) is associated with several local and systemic consequences. Complications include pancreatic necrosis, multiple organ failure, systemic inflammatory response syndrome (SIRS), and death. Early and accurate prediction of AP severity is essential for the timely initiation of appropriate therapeutic measures, including intensive care monitoring, nutritional support, and surgical intervention [1, 2].

AP is typically diagnosed based on the Revised Atlanta Criteria, which necessitates the fulfillment of at least two of the following three criteria: (1) elevated pancreatic enzymes (serum lipase or amylase exceeding three times the normal upper limit); (2) characteristic abdominal pain (sudden, severe epigastric pain potentially radiating to the back); and (3) imaging findings indicative of pancreatic inflammation or complications (as observed on CT, MRI, or ultrasound) [3, 4]. Numerous grading systems and biomarkers have been developed to predict SAP. The Ranson criteria [5], Acute Physiology and Chronic Health Evaluation (APACHE) II score [6], Bedside Index for Severity in Acute Pancreatitis (BISAP) [7], and modified Glasgow Prognostic Score (mGPS) [8]. Among these, the mGPS is commonly used and is an effective

tool for predicting SAP. It consists of eight criteria:  $\text{PaO}_2 < 8 \text{ kPa}$ , age  $> 55$  years, white blood cell count exceeding  $15 \times 10^9/\text{L}$ , serum calcium  $< 2.0 \text{ mmol/L}$ , blood urea levels greater than  $16 \text{ mmol/L}$ , lactate dehydrogenase (LDH)  $> 600 \text{ IU/L}$  or aspartate aminotransferase (AST)  $> 200 \text{ IU/L}$ , serum albumin lower than  $32 \text{ g/L}$ , and blood glucose  $> 10 \text{ mmol/L}$  [8, 9]. Each parameter was assigned one point, and a total score of three or more indicated severe AP, associated with an increased risk of complications, organ failure, and mortality [8-10]. Despite their usefulness, these scoring systems are sometimes difficult to understand, time-consuming, and may require data that are not easily accessible during the early stages of disease development. This has led to an ongoing search for simpler, more accessible biomarkers that can provide early and accurate risk stratification in patients with AP.

MVP is a notable biomarker that has attracted considerable attention in recent years. MPV serves as a marker of platelet activation, as it is ascertained by evaluating the average size of platelets in the bloodstream, together with inflammation [11, 12]. Larger platelets exhibit heightened reactivity, possess an increased number of granules, and have a greater propensity to induce thrombosis than smaller platelets. Platelet activation is crucial in the pathophysiology of both inflammation and thrombosis [13]. During acute pancreatitis, excessive production of proinflammatory cytokines, including IL-1, IL-6, and TNF- $\alpha$ , induces platelet activation and coagulation cascades, leading to heightened thrombotic activity and ischemic damage in pancreatic tissue [14, 15]. Chronic systemic inflammation and platelet activation result in elevated MPV, indicating the generation of larger, more reactive platelets. The pathophysiological connection between MPV and AP may be predominantly facilitated by systemic inflammation, endothelial dysfunction, and microvascular abnormalities [12, 16].

There is considerable interest in the role of MPV in acute pancreatitis because of the interplay between inflammation, coagulation, and platelet activation in the etiology of the disease [13, 17]. Investigations are ongoing to ascertain the potential correlation between MPV and acute pancreatitis severity. Numerous investigations have shown contradictory findings, with

some indicating a definitive correlation between increased MPV and severe AP [1, 13], whereas others have identified no significant relationship [18]. These inconsistencies may arise from variations in study design, patient demographics, and timing of MPV assessment. The measurement of MPV is significant because of its accessibility and cost-effectiveness, as it can be obtained using standard complete blood count (CBC) assays, rendering it a desirable choice for early risk stratification in clinical practice. This study aimed to evaluate the utility of Mean Platelet Volume in predicting disease severity in Acute Pancreatitis.

### Methods and methodology

#### *Sample size*

The sample size for this study was determined based on previous research. The research conducted by Beyazit Y et al. [1] reported an area under the curve (AUC) of 0.762 for the ROC analysis of MPV in differentiating between moderate and severe acute pancreatitis based on the modified Glasgow Prognostic Score. The calculation utilized the formula  $n = Z_{\alpha/2}^2 \times V(\text{AUC})/d^2$ , where  $V(\text{AUC})$  represents the variance of the AUC and  $d$  is the margin of error, set at 0.045. The Z-score  $Z_{\alpha/2}$ , corresponding to the 95% confidence level, was 1.96. The variance  $V(\text{AUC})$  was calculated using a function that incorporated the inverse of the standard cumulative normal distribution. Substituting these values, the required sample size was 250 participants.

#### *Study population*

The study included patients admitted to Yenepoya Medical College Hospital (YMCH) with a confirmed diagnosis of acute pancreatitis. Participants were eligible if they were  $\geq 18$  years of age and met at least two of the following diagnostic criteria: characteristic abdominal pain, elevated serum amylase and/or lipase levels exceeding three times the normal upper limit, or imaging findings consistent with acute pancreatitis according to the 2012 Revised Atlanta Criteria [8, 9]. Patients who did not meet these diagnostic criteria, or those diagnosed with chronic pancreatitis (evidenced by pancreatic calcifications, ductal dilatation, atrophy, or pseudocysts), were excluded from the study.

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Healthy volunteers aged > 18 years with no significant medical or psychiatric history were included in the study. Women of childbearing potential used contraception and had negative pregnancy tests. Individuals with chronic diseases, significant infections, recent trial participation, major surgery, mental health issues, substance abuse, abnormal laboratory results, or pregnancy were excluded from the study.

## Study design

This study was designed as a randomized controlled trial and was conducted at the YMCH in Deralakatte. Patients who met the predetermined inclusion and exclusion criteria were randomly assigned to different groups: the healthy group and acute pancreatitis group.

## Study procedure

Before data collection began, ethical clearance was obtained from the Institutional Ethics Committee (IEC) at the YMCH with clearance no. YEC-1/2024/379. Participants were randomly assigned to study groups in a ratio of 1:5, healthy and acute pancreatitis groups, and data collection was carried out systematically. Detailed patient information, including demographic details, clinical history, and examination findings, was obtained from the participants. The specific clinical parameters collected included the presence of icterus, mass in the left upper quadrant, and symptoms such as jaundice, fever, and vomiting. Laboratory data, particularly MPV levels, and radiological findings from ultrasound and/or contrast-enhanced CT scans were meticulously recorded for analysis. MPV was measured as part of a CBC using an automated hematology analyzer, which assesses platelet size in femtoliters (fL) from a blood sample collected in an EDTA tube. Normal MPV values typically range from 7.5 to 12 fL.

## Statistical analysis

The data were entered and encoded in MS Excel, and all statistical analyses were performed using IBM SPSS version 27. Quantitative variables were described as either the mean with standard deviation or median, depending on the data distribution, whereas categorical variables were expressed as frequencies and percentages, supplemented with graphical representations. Associations be-

tween categorical variables were assessed using the Chi-square test or Fisher's exact test, considering a  $p$ -value of < 0.05 as statistically significant. An ROC curve was computed to determine MPV cut-off values, sensitivity, and specificity in predicting severe acute pancreatitis.

## Results

### Demographic details of the participants

The study included 279 participants, of whom 267 (95.69%) were male and 12 (4.31%) were female. A significant difference was observed in the gender distribution, with 97.59% of the acute pancreatitis cohort being male compared to 80% in the healthy volunteer group ( $P < 0.001$ ). The average age of the participants was  $42.70 \pm 10.7$  years, and there was a significant difference between the acute pancreatitis patients with a mean age of  $41.98 \pm 10.56$  years and the healthy volunteers with a mean age of  $48.67 \pm 10.57$  years ( $P = 0.001$ ). Income distribution was similar across both groups, with no statistically significant differences ( $P > 0.05$ ). The clinical characteristics unique to the acute pancreatitis group included the presence of comorbidities such as diabetes (6%), hypertension (3.6%), history of jaundice (4.8%), icterus (5.2%), pancreatic mass (3.6%), nicotine dependence syndrome (2%), and alcohol dependence syndrome (9.2%). Additionally, 7.6% of the patients with acute pancreatitis exhibited pancreatic necrosis (**Table 1**).

### Comparison of mean platelet volume among healthy and acute pancreatitis patients

In healthy individuals, the mean platelet volume (MPV) was  $8.88 \pm 0.97$ , while patients with acute pancreatitis showed a slightly higher value of  $9.43 \pm 6.78$ . However, this difference was not statistically significant ( $P > 0.05$ ). When comparing patients with pancreatic necrosis to those without, the MPV was notably higher in the necrosis group at  $13.17 \pm 1.7$  than  $9.19 \pm 0.86$  in the non-necrosis group. This difference was statistically significant, with a  $p$ -value < 0.001 (**Table 2**).

### ROC analysis of mean platelet volume for acute pancreatitis and pancreatic necrosis prediction

The ROC analysis for the MPV demonstrates sub-optimal diagnostic efficacy, as indicated by

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**Table 1.** Demographic and clinical characteristics of patients with acute pancreatitis compared to healthy volunteers

Parameters		Acute pancreatitis	Healthy volunteer	p value
Gender	Male	243 (97.59%)	24 (80%)	< 0.001
	Female	6 (2.41%)	6 (20%)	
Age in year		41.98 ± 10.56	48.67 ± 10.57	0.001
Income	Low	170 (68.3%)	21 (70%)	0.957
	Low-middle	68 (27.3%)	8 (26.67%)	
	Middle	11 (4.4%)	1 (3.33%)	
Comorbidities	Diabetes	15 (6%)		
	Hypertension	9 (3.6%)		
H/o of Jaundice		12 (4.8%)		
Icterus		13 (5.2%)		
Mass		9 (3.6%)		
History of tobacco use		5 (2%)		
History of alcoholism		23 (9.2%)		
Pancreatic necrosis		19 (7.6%)		

**Table 2.** Comparison of MPV between healthy individuals, acute pancreatitis patients, and patients with/without pancreatic necrosis

Parameter	MPV	p value
Healthy voluntary (fl)	8.88 ± 0.97	0.572
Acute pancreatitis (fl)	9.43 ± 6.78	
Pancreatic necrosis (fl)	Yes 13.17 ± 1.7	< 0.001
	No 9.19 ± 0.86	

an Area Under the Curve (AUC) of 0.503. This value suggests that the mean platelet volume test does not effectively discriminate between positive (AP) and negative cases, performing marginally better than random chance (**Figure 1**). Moreover, for predicting pancreatic necrosis in AP, MPV had a poor ability to distinguish between positive and negative classes as indicated by an AUC of 0.609. At a cutoff value of 6.20, the sensitivity was 1.000, meaning that all positive cases were identified correctly, but the false-positive rate was also 100%, indicating that all negative cases were incorrectly classified as positive. As the cutoff values increased, the sensitivity declined, whereas the specificity improved only slightly, and the false-positive rates remained high (**Figure 2**).

### *Correlation of mean platelet volume and CT Severity Score in acute pancreatitis*

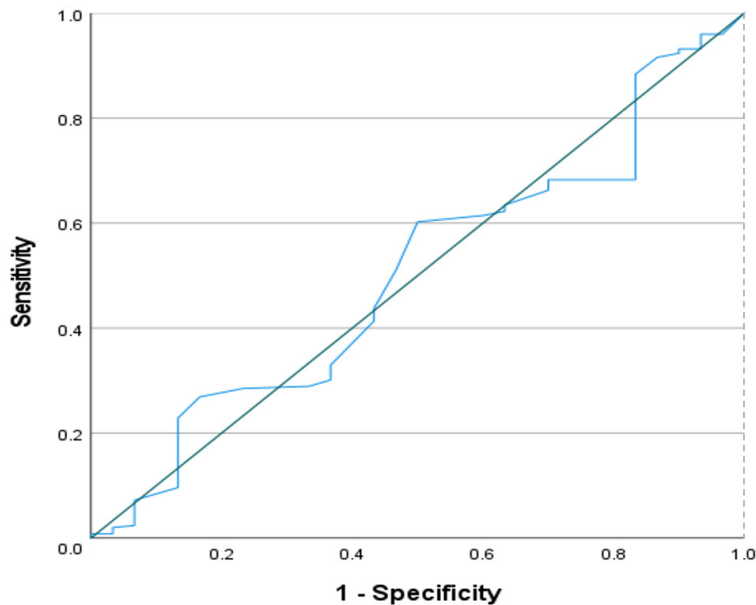
The MPV in the AP group was  $9.43 \pm 6.78$ , reflecting substantial variability in the data. In

contrast, the CT severity score had a mean of  $6.26 \pm 1.96$ , indicating that the values were more tightly distributed around the mean value. Correlation analysis revealed a very weak negative Pearson correlation coefficient of -0.041 between MPV and CT severity score. However, this association was not statistically significant, as denoted by a *p*-value of 0.524. Therefore, no meaningful linear relationship was observed between MPV and CT severity, suggesting that variations in MPV did not predict changes in CT severity (**Table 3**).

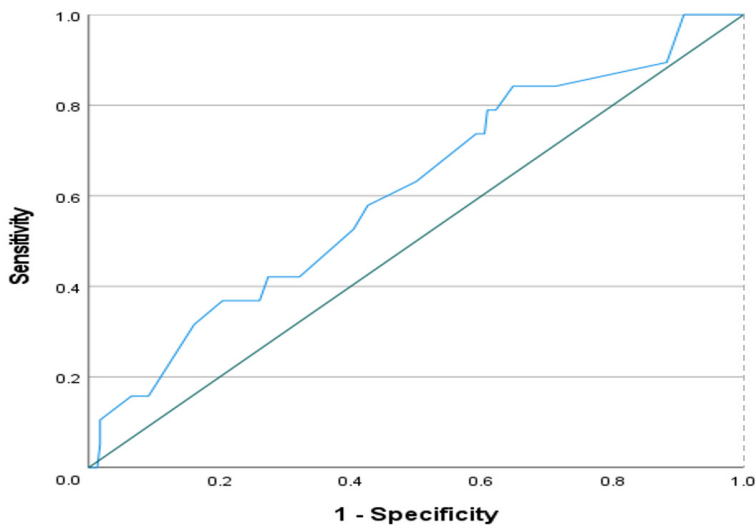
### **Discussion**

This study investigated the correlation between MPV and acute pancreatitis, revealing that MPV levels in patients with acute pancreatitis were marginally higher than those in healthy individuals ( $9.43 \pm 6.78$  fL vs.  $8.88 \pm 0.97$  fL). However, the observed difference was not statistically significant ( $P > 0.05$ ), with an AUC value of 0.503. These findings align with the research conducted by Kefeli A et al. [19], although their reported MPV level in acute pancreatitis patients was lower ( $7.8 \pm 1.6$  fL). In contrast, several other studies have demonstrated significant variations in MPV levels among patient with acute pancreatitis. Erdem A et al. [20] noted a markedly elevated MPV at admission in individuals with acute pancreatitis ( $8.6 \pm 1.4$  fL vs.  $7.6 \pm 0.7$  fL), indicating an early elevation of MPV during the acute phase. In contrast, studies conducted by Beyazit Y et al.

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**Figure 1.** Receiver Operating Characteristic (ROC) curve for the classification model, indicating the trade-off between sensitivity and specificity of MPV in diagnosing AP.



**Figure 2.** Receiver Operating Characteristic (ROC) curve for the classification model, indicating the trade-off between sensitivity and specificity of MPV in predicting pancreatic necrosis among AP.

**Table 3.** Comparison of MPV and CT severity score in acute pancreatitis and their correlation

Parameter	Mean $\pm$ Standard Deviation	Correlation (r)	p-value
Mean Platelet Volume (MPV)	9.43 $\pm$ 6.78	-0.041	0.524
CT Severity Score	6.26 $\pm$ 1.96		

[1] and Pençe HH et al. [21] reported a notable and statistically significant reduction in MPV levels in patients with acute pancreatitis compared to healthy controls, with values reported as  $8.06 \pm 0.71$  fL versus  $8.63 \pm 0.62$  fL, and  $7.9 \pm 1.1$  fL versus  $8.4 \pm 0.9$  fL, respectively. As AP progresses, MPV levels fluctuate in response to systemic inflammation. During the early inflammatory phase, cytokine release and platelet activation lead to an increase in the MPV. However, with sustained inflammation, excessive platelet consumption results in decreased MPV levels in the plasma, which is compensated for by an immediate bone marrow response [18]. The inconsistencies in MPV results across these studies may be due to timing of MPV measurements and variations in patient populations, such as the severity of pancreatitis, comorbid conditions, and demographic characteristics. Additionally, variations in laboratory methods and equipment may have contributed to discrepancies in MPV measurements.

This study highlighted the potential of Mean Platelet Volume (MPV) as an indicator of the severity of acute pancreatitis. The study revealed that patients with pancreatic necrosis had significantly higher MPV levels ( $13.17 \pm 1.7$ ) than those with acute pancreatitis ( $9.19 \pm 0.86$ ), with a *p*-value of less than 0.05 and an area under the curve (AUC) of 0.609. Huang P et al., [22] similarly reported that patients with persistent organ failure had elevated MPV values on admission, suggesting a link be-



tween higher MPV and severe forms of acute pancreatitis. However, contrasting findings from Lei JJ et al. [23] and Affarah L et al. [24] revealed significantly lower MPV levels in the SAP group than in the non-SAP group. These discrepancies raise questions about the reliability of MPV as a standalone marker for detecting severe acute pancreatitis, highlighting the need for further research to clarify its role and improve its diagnostic accuracy. Future studies should focus on standardizing MPV thresholds, integrating it with other inflammatory markers and determining its predictive value in different AP subtypes to enhance its clinical utility.

This study has several limitations that affect the quality and reliability of its findings. First, there was a lack of standardization in MPV measurement protocols, including variations in laboratory techniques and equipment, which could have contributed to inconsistent results. Moreover, the study did not adequately control for potential confounding variables such as patient demographics, comorbid conditions, and pancreatitis severity, which may influence MPV levels. Furthermore, the timing of MPV measurements relative to the onset of symptoms was not standardized, potentially affecting data accuracy. Multicenter collaborations are lacking, which would have included a more diverse population and enhanced generalizability. Addressing these limitations in future studies is crucial for improving the robustness and reliability of findings related to MPV and acute pancreatitis.

## Conclusion

The study revealed that MPV levels were marginally increased in patients with acute pancreatitis compared to healthy controls; however, the difference was not statistically significant. Despite MPV's promise of differentiating severe acute pancreatitis, inconsistencies among studies underscore the necessity for further research to elucidate its reliability and function in evaluating disease severity.

## Disclosure of conflict of interest

All authors affirm that they have no competing interests that could potentially interfere with the impartiality of the research, analysis or conclusions presented in this manuscript.

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