



Research Article

## Serum Enzymes and Inflammatory Mediators as Predictors of Severity in Acute Pancreatitis

Dr. Harshvardhan<sup>1</sup>, Dr. Deepa<sup>2</sup>, Dr. G.G. Kaushik<sup>3</sup>, Dr. Nitin Sharma<sup>4</sup>

<sup>1</sup> Senior Demonstrator, Department of Biochemistry, JLN Medical College, Ajmer.

<sup>2</sup> Senior Demonstrator, Department of Biochemistry, JLN Medical College, Ajmer.

<sup>3</sup> Retried Senior Professor, Department of Biochemistry, JLN Medical College, Ajmer.

<sup>4</sup> Senior Professor, Department of Biochemistry, JLN Medical College, Ajmer.

 OPEN ACCESS

### Corresponding Author:

**Dr. Nitin Sharma**

Senior Professor, Department of  
Biochemistry, JLN Medical  
College, Ajmer.

Received: 02-08-2025

Accepted: 24-08-2025

Available online: 02-10-2025

Copyright © International Journal of  
Medical and Pharmaceutical Research

### ABSTRACT

This study evaluates serum amylase, lipase, IL-10, and procalcitonin in healthy controls, mild acute pancreatitis, and severe acute pancreatitis. Results indicate significant biomarker variations across groups, highlighting diagnostic and prognostic roles, particularly for procalcitonin and IL-10 in predicting severity.

**Keywords:** Acute pancreatitis, Procalcitonin, Disease severity, Biomarkers, Prognostic indicators.

### INTRODUCTION

Acute pancreatitis (AP) is a potentially life-threatening inflammatory disorder of the pancreas characterized by autodigestion of pancreatic tissue and variable systemic complications [1,2]. The disease exhibits a broad clinical spectrum ranging from mild, self-limiting illness to severe necrotizing pancreatitis with multiorgan failure and high mortality [3]. The global incidence of AP ranges between 13 and 73 per 100,000, and the prevalence in India is approximately 7.9 per 100,000 [4,5]. Alcohol and gallstones constitute the leading etiological factors, while genetic, metabolic, and idiopathic cases account for the remainder [6,7]. The pathophysiology involves premature activation of digestive zymogens, acinar cell injury, and release of inflammatory mediators, resulting in systemic inflammatory response syndrome (SIRS) and multiorgan dysfunction [8,9]. Biochemical markers play an essential role in the early diagnosis and prognosis of AP. Serum amylase and lipase are classical diagnostic markers, though they do not always correlate with disease severity [10]. Cytokines such as interleukin-10 (IL-10), with its potent anti-inflammatory properties, and procalcitonin (PCT), a marker of systemic inflammation, are recognized as promising severity predictors [11,12]. This study was conducted to compare serum amylase, lipase, IL-10, and PCT levels in healthy controls, mild AP, and severe AP, and to explore their prognostic significance.

### METHODOLOGY

This observational case-control study was conducted at J.L.N. Medical College & Associated Group of Hospitals, Ajmer. A total of 330 participants were included: 110 healthy controls, 110 patients with mild acute pancreatitis, and 110 with severe acute pancreatitis, classified using the Revised Atlanta Classification. Acute pancreatitis diagnosis was based on clinical features, elevated amylase or lipase (>3 times the upper limit of normal), and radiological findings. Exclusion criteria included chronic pancreatitis, pancreatic malignancy, major comorbidities, and recent infections. Blood samples were collected within 48 hours of admission. Serum amylase and lipase were estimated using enzymatic methods. IL-10 levels were measured by enzyme-linked immunosorbent assay (ELISA), and procalcitonin was determined by

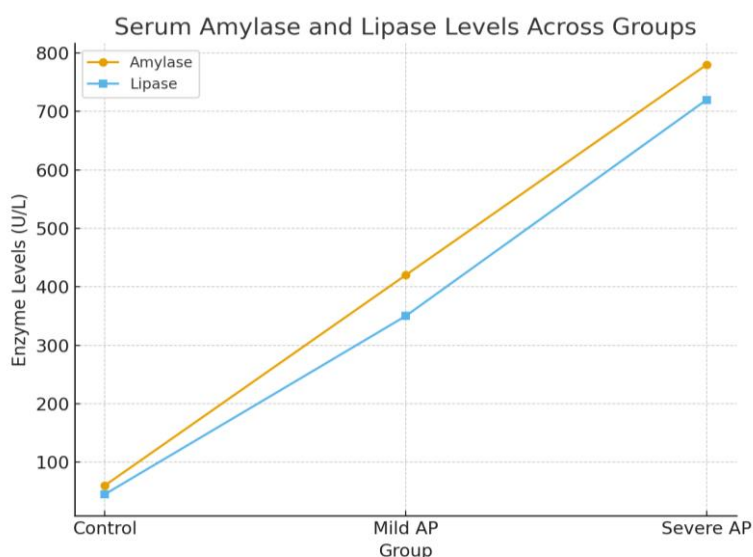
chemiluminescent assay. Statistical analysis was performed using ANOVA, independent t-test, Pearson correlation and post-hoc tests, with  $p < 0.001$  considered significant. The study acquired consent from all participants has taken also with ethical permission.

## RESULTS

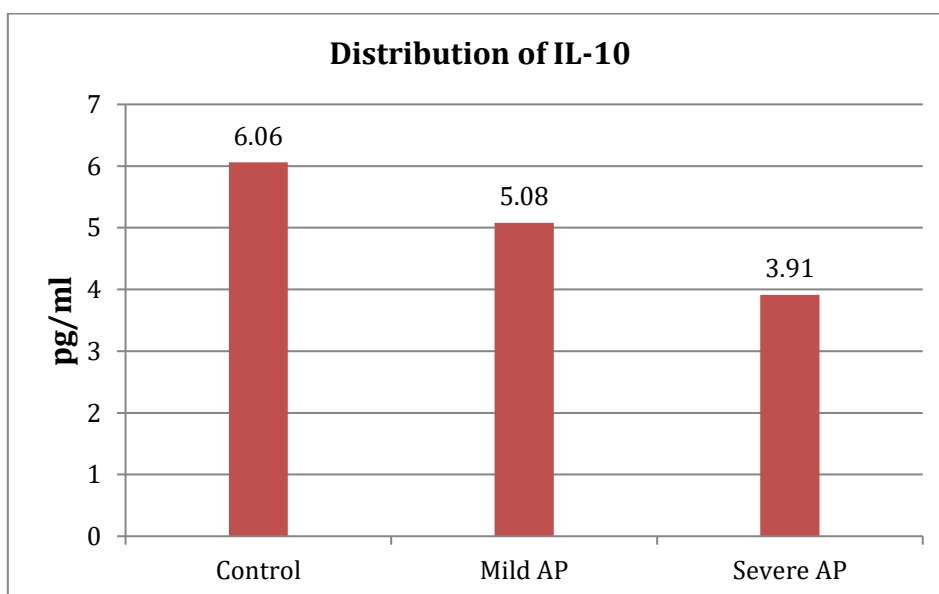
Serum amylase and lipase levels were significantly elevated in mild and severe AP compared to controls ( $p < 0.001$ ), with higher mean values in severe AP (Table 1, Figure 1). IL-10 levels showed a significantly decrease in severe AP in comparison with mild AP & healthy controls (Table 1, Figure 2). Procalcitonin levels were minimal in controls, moderately elevated in mild AP, and markedly elevated in severe AP ( $p < 0.001$ ), as shown in (Table 1 and Figure 3). These findings highlight the diagnostic value of amylase and lipase and the prognostic utility of IL-10 and procalcitonin.

**Table 1: Comparison of serum biomarkers among study groups.**

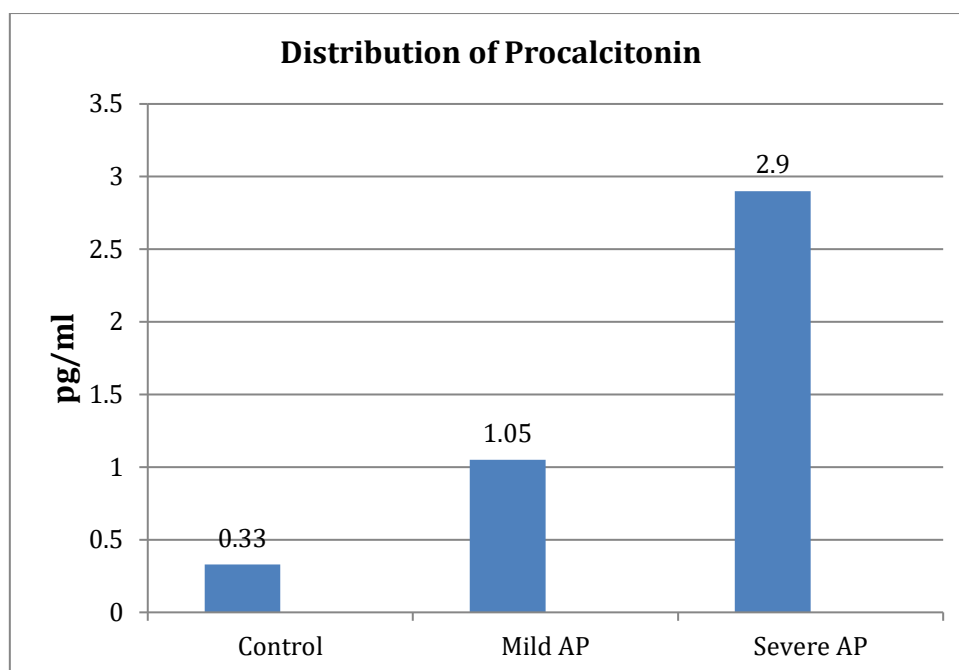
Parameter	Control (n=110)	Mild AP (n=110)	Severe AP (n=110)	p-value
<b>Amylase (U/L)</b>	42.88 $\pm$ 7.15	530.32 $\pm$ 117.66	1482.44 $\pm$ 437.04	<0.001*
<b>Lipase (U/L)</b>	35.73 $\pm$ 5.73	256.07 $\pm$ 55.32	879.65 $\pm$ 52.92	<0.001*
<b>IL-10 (pg/mL)</b>	6.06 $\pm$ 0.31	5.08 $\pm$ 0.30	3.91 $\pm$ 0.31	<0.001*
<b>Procalcitonin (ng/mL)</b>	0.33 $\pm$ 0.06	1.05 $\pm$ 0.27	2.90 $\pm$ 0.92	<0.001*



**Figure 1: Serum amylase and lipase levels across groups.**



**Figure 2: Serum IL-10 levels across groups.**



**Figure 3: Serum procalcitonin levels across groups.**

## DISCUSSION

The present study demonstrates that while serum amylase and lipase are useful for diagnosing AP, they lack specificity in predicting severity. Our findings corroborate previous studies indicating that procalcitonin is a highly sensitive and specific marker for identifying severe cases [13,14]. Our study is consistent with the study conducted by Fisie E et al (2006) [15], Berney T et al (1999) [16], Rongione AJ et al (1997) and various studies which reported a significant decrease in the level of IL-10 in patients with mild AP & severe AP subjects in comparison with health controls. Berney T et al (1999) study showed IL-10 on day 0 to be an extremely good marker for predicting the progression to severity AP. IL-10 is an anti-inflammatory cytokine which inhibits the release of pro-inflammatory cytokines (i.e., IL-1  $\beta$ , IL-6, and tumor necrosis factor- $\alpha$ ) from monocytes/macrophages thus preventing subsequent tissue damage. Studies have correlated lower levels of IL-10 with a higher severity of pancreatitis. [16] Procalcitonin, by contrast, rises proportionally with severity and correlates well with clinical scoring systems such as Ranson and APACHE II [17,18]. Thus, combining classical enzymatic markers with cytokine and inflammatory biomarkers may improve early risk stratification.

## Limitations

The sample size was modest, and the study was conducted at a single center, which may limit generalizability. Serial measurements of biomarkers were not performed, which could provide better insights into dynamic changes. Advanced imaging correlations with biomarkers were limited.

## CONCLUSION

Serum amylase and lipase remain diagnostic cornerstones of acute pancreatitis, but their prognostic utility is limited. IL-10 and procalcitonin provide additional insights into disease severity, with procalcitonin emerging as a reliable biomarker for early identification of severe acute pancreatitis. IL-10 can be also used therapeutically in patients of AP.

## REFERENCES

1. Andris A. Pancreatitis: understanding the disease and implications for care. AACN Advanced Critical Care. 2010 Apr 1;21(2):195-204.
2. 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 Dec 1;57(12):1698-703.
3. Frossard JL, Hadengue A, Pastor CM. New serum markers for the detection of severe acute pancreatitis in humans. American journal of respiratory and critical care medicine. 2001 Jul 1;164(1):162-70..
4. Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Official journal of the American College of Gastroenterology| ACG. 2006 Oct 1;101(10):2379-400.
5. Yadav D, Vege SS, Chari ST. Epidemiology of pancreatitis. GI Epidemiology: Diseases and Clinical Methodology. 2014 Feb 6;306-12.
6. Lankisch PG, Breuer N, Bruns A, Weber-Dany B, Lowenfels AB, Maisonneuve P. Natural history of acute pancreatitis: a long-term population-based study. Official journal of the American College of Gastroenterology| ACG. 2009 Nov 1;104(11):2797-805..

7. Whitcomb DC. Genetic risk factors for pancreatic disorders. *Gastroenterology*. 2013 May 1;144(6):1292-302..
8. Balakrishnan V, Nair P, Radhakrishnan L, Narayanan VA. Tropical pancreatitis-a distinct entity, or merely a type of chronic pancreatitis?. *Indian Journal of Gastroenterology*. 2006 Mar 1;25(2):74.
9. Takeda K, Yokoe M, Takada T, Kataoka K, Yoshida M, Gabata T, Hirota M, Mayumi T, Kadoya M, Yamanouchi E, Hattori T. Assessment of severity of acute pancreatitis according to new prognostic factors and CT grading. *Journal of hepato-biliary-pancreatic sciences*. 2010 Jan;17(1):37-44.
10. Mareninova OA, Hermann K, French SW, O’Konski MS, Pandol SJ, Webster P, Erickson AH, Katunuma N, Gorelick FS, Gukovsky I, Gukovskaya AS. Impaired autophagic flux mediates acinar cell vacuole formation and trypsinogen activation in rodent models of acute pancreatitis. *The Journal of clinical investigation*. 2009 Nov 2;119(11):3340-55.
11. Thrower EC, et al. Calcium and acinar cell injury in pancreatitis. *Gastroenterology*. 2006.
12. Bhatia M et al. microvascular thrombosis in pancreatitis. *Gut*. 2003
13. ELIII B. A clinically based classification system for acute pancreatitis. *Arch Surg*.. 1993;128:586-90.
14. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013 Jan 1;62(1):102-11.
15. Fisic E, Poropat G, Bilic-Zulle L, Licul V, Milic S, Stimac D. The Role of IL-6, 8, and 10, sTNF $\alpha$ , CRP, and pancreatic elastase in the prediction of systemic complications in patients with acute pancreatitis. *Gastroenterology research and practice*. 2013;2013(1):282645.
16. Berney T, Gasche Y, Robert J, Jenny A, Mensi N, Grau G, Vermeulen B, Morel P. Serum profiles of interleukin-6, interleukin-8, and interleukin-10 in patients with severe and mild acute pancreatitis. *Pancreas*. 1999 May 1;18(4):371-7.
17. Granger J, Remick D. Acute pancreatitis: models, markers, and mediators. *Shock*. 2005 Dec 1;24:45-51.
18. Dinarello CA. Proinflammatory and anti-inflammatory cytokines as mediators in the pathogenesis of septic shock. *Chest*. 1997 Dec 1;112(6):321S-9S.
19. Schölmerich J. Interleukins in acute pancreatitis. *Scandinavian Journal of Gastroenterology*. 1996 Jan 1;31(sup219):37-42..
20. Pastor CM, et al. Pathophysiology of pancreatitis. *Best Pract Res Clin Gastroenterol*. 2004;):321 S-329S. doi: 10.1378/chest.112.6 supplement.321. PMID: 9400897.
21. Bhatia M. Inflammatory response on the pancreatic acinar cell injury. *Scandinavian Journal of Surgery*. 2005 Jun;94(2):97-102.
22. Mayer J, Rau B, Gansauge F, Beger HG. Inflammatory mediators in human acute pancreatitis: clinical and pathophysiological implications. *Gut*. 2000 Oct 1;47(4):546-52.
23. Norman J. The role of cytokines in the pathogenesis of acute pancreatitis. *The American Journal of Surgery*. 1998 Jan 1;175(1):76-83.
24. Mayer J, Rau B, Gansauge F, Beger HG. Inflammatory mediators in human acute pancreatitis: clinical and pathophysiological implications. *Gut*. 2000 Oct 1;47(4):546-52.