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Diagnostic and Prognostic Value of Serum Amylase, Lipase and Interleukin-6 in Healthy Individuals and Patients with Mild and Severe Acute Pancreatitis

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ABSTRACT

Background: Acute pancreatitis (AP) presents with variable severity ranging from mild self-limiting disease to severe systemic illness with organ failure. Serum amylase and lipase remain central to diagnosis, while interleukin-6 (IL-6) has emerged as a promising marker for early severity assessment. This study aimed to compare amylase, lipase, and IL-6 levels across healthy individuals, mild AP, and severe AP.

Methods: A cross-sectional analytic study was conducted in a tertiary academic hospital including 330 participants: 110 healthy controls, 110 mild AP cases, and 110 severe AP cases. Diagnosis and severity classification followed Atlanta and Ranson criteria. Fasting venous samples were analyzed for serum amylase, lipase, and plasma IL-6 using validated laboratory protocols. Statistical comparisons employed ANOVA with post-hoc testing (α =0.05).

Results: Mean serum amylase (U/L) increased from 42.88 ± 7.15 in controls to 530.32 ± 117.66 in mild AP and 1482.44 ± 437.04 in severe AP (p<0.001). Serum lipase (U/L) rose from 35.73 ± 5.73 to 256.07 ± 55.32 and 879.65 ± 252.92 , respectively (p<0.001). IL-6 (pg/mL) increased markedly from 4.84 ± 0.61 in controls to 54.94 ± 15.73 in mild AP and 300.14 ± 108.05 in severe AP (p<0.001). IL-6 showed the greatest discriminatory power between mild and severe disease.

Conclusions: While amylase and lipase remain robust diagnostic markers of AP, IL-6 demonstrates strong prognostic utility by distinguishing severity levels. Incorporating IL-6 alongside enzyme assays may enhance early triage, guide monitoring, and support clinical decision-making in acute care settings.

Keywords: Acute pancreatitis, amylase, lipase, interleukin-6, biomarkers, severity

INTRODUCTION

Acute pancreatitis (AP) is a common and potentially life-threatening inflammatory disorder of the pancreas. Its incidence has increased worldwide, with an annual rate ranging from 13 to 73 per 100,000 population [2,5,7]. Clinically, AP varies from mild, self-limiting disease to severe necrotizing pancreatitis associated with multiorgan failure and high mortality [3,12].

Diagnosis of AP has traditionally relied on serum amylase and lipase [1,13]. Amylase rises rapidly but lacks specificity, while lipase is more sensitive and remains elevated longer [1,19]. However, neither reliably predicts disease severity. Recent research has focused on inflammatory cytokines as markers of systemic involvement. Interleukin-6 (IL-6) is a multifunctional cytokine that stimulates acute-phase response proteins, correlates with systemic inflammation, and rises early in AP [9,10,16].

This study evaluates serum amylase, lipase, and IL-6 in healthy individuals and patients with mild and severe AP, aiming to determine their relative diagnostic and prognostic significance.

Methodology

This prospective observational study was conducted at a tertiary care hospital. Participants included healthy volunteers and patients diagnosed with AP. Total number of participants are 330 (110 healthy controls, 110 mild AP patients and 110 severe AP patients). Diagnosis required at least two of three criteria: (1) abdominal pain suggestive of AP, (2) \geq 3-fold elevation in serum amylase or lipase, and (3) imaging findings consistent with AP [2].

Patients were categorized as mild or severe AP according to the Revised Atlanta Classification [2]. Severity was further confirmed using Ranson's criteria [14]. Exclusion criteria included chronic pancreatitis, pancreatic malignancy, chronic kidney disease, liver dysfunction, or other systemic inflammatory disorders.

Venous blood samples were collected within 48 hours of admission. Serum amylase and lipase were measured using automated enzymatic assays. Serum IL-6 was measured using enzyme-linked immunosorbent assay (ELISA), following protocols established in previous studies [17].

Statistical analysis was performed using SPSS software. Data were expressed as mean \pm standard deviation (SD). One-way analysis of variance (ANOVA) compared the three groups, Pearson correlation with post-hoc t-tests for intergroup comparisons. A p-value <0.001 was considered statistically significant [19]. The study acquired consent from all participants has taken also with ethical permission.

Results

Serum Amylase

Mean serum amylase was 42.88 ± 7.15 U/L in healthy controls, 530.32 ± 117.66 U/L in mild AP, and 1482.44 ± 437.04 U/L in severe AP (Table 1, Figure 1). ANOVA showed significant intergroup differences (p<0.001). Post-hoc comparisons confirmed significance between each pair (p<0.001).

Serum Lipase

Mean lipase levels were 35.73 ± 5.73 U/L in healthy controls, 256.07 ± 55.32 U/L in mild AP, and 879.65 ± 252.92 U/L in severe AP(Table 2, Figure 2). Differences were statistically significant (ANOVA p<0.001; all post-hoc p<0.001).

Serum IL-6

IL-6 levels were 4.84 ± 0.61 pg/ml in controls, 54.94 ± 15.73 pg/ml in mild AP, and 300.14 ± 108.05 pg/ml in severe AP(Table 3, Figure 3). Intergroup differences were highly significant (ANOVA p<0.001; all post-hoc p<0.001).

Statistical Summary

All three biomarkers were significantly elevated in AP compared to controls and increased further with disease severity. IL-6 demonstrated the greatest discriminatory power between mild and severe AP.

Tables

Table 1. Serum Amylase Levels (U/L)

Groups	Healthy control N=110	Mild Acute Pancreatitis N=110	Severe Acute Pancreatitis N=110	p-Value
Mean±(SD) Amylase(U/L)	42.88 ±7.15	530.32 ± 117.66	1482.44 ±437.04	<0.001*

Table 2. Serum Lipase Levels (U/L)

Groups	Healthy control N=110	Mild Acute Pancreattits N=110	Severe Acute Pancreattits N=110	p-Value
Mean±(SD) Lipase(U/L)	35.73 ±5.73	256.07 ± 55.32	879.65 ±52.92	<0.001*

Table 3. Serum IL-6 Levels (pg/ml)

Groups	Healthy control N=110	Mild Acute Pancreattits N=110	Severe Acute Pancreattits N=110	p-Value
Mean±(SD) IL-6 (pg/ml)	4.84 (0.61)	54.94 (15.73)	300.14 (108.05)	<0.001*

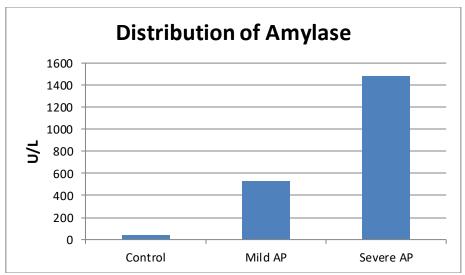


Figure 1. Serum Amylase Levels

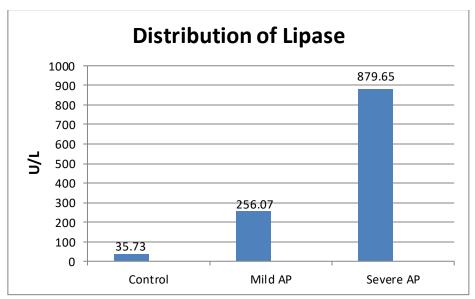


Figure 2. Serum Lipase Levels

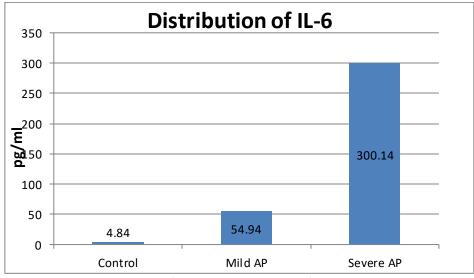


Figure 3. Serum IL-6 Levels

Discussion

This study confirms that amylase, lipase, and IL-6 are significantly elevated in AP compared with healthy individuals, with the highest levels seen in severe disease.

Amylase remains widely used for initial diagnosis, but its lack of specificity and rapid clearance limit prognostic value [1,7]. Lipase is more sensitive and remains elevated longer, offering greater diagnostic reliability [1,19]. However, neither enzyme effectively distinguishes mild from severe disease in early stages [13].

IL-6 showed the strongest correlation with disease severity, supporting previous studies that highlight its role as an early prognostic biomarker [9,10,16,17]. Elevated IL-6 reflects activation of the systemic inflammatory cascade, which drives complications such as systemic inflammatory response syndrome (SIRS) and multiorgan failure [8,11,15]. Studies have consistently shown IL-6 to outperform CRP, TNF-α, and other markers in early prediction of severe AP [16,17,18].

Our findings align with Dambrauskas et al. [16] who reported higher IL-6 expression in severe compared to mild AP, and Chen et al. [17] who demonstrated its predictive role for prognosis. Mayer et al. [18] further emphasized its role in the inflammatory network of AP. The discriminatory capacity of IL-6 seen in our study highlights its potential clinical utility, especially when combined with amylase and lipase.

Early identification of patients likely to progress to severe AP is essential for appropriate triage, intensive monitoring, and intervention [3,12,20]. Incorporating IL-6 into standard diagnostic panels could improve accuracy and enable timely escalation of care.

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 valuable diagnostic tools for AP but lack prognostic utility. IL-6 demonstrated superior correlation with disease severity, supporting its role as a reliable early prognostic biomarker. Routine IL-6 testing a longside classical enzyme assays may enhance early risk stratification, guide management, and improve patient outcomes [6].

References

- 1. Matull WR, Pereira SP, O'Donohue JW. Biochemical markers of acute pancreatitis. J ClinPathol. 2006;59(4):340-344.
- 2. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification. Gut. 2013;62(1):102-111.
- 3. Wu BU, Banks PA. Clinical management of patients with a cute pancreatitis. Gastroenterology. 2013;144(6):1272-1281.
- 4. Tenner S, Baillie J, DeWitt J, Vege SS. ACG guideline: management of acute pancreatitis. Am J Gastroenterol. 2013;108(9):1400-1415.
- 5. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. Gastroenterology. 2013;144(6):1252-1261.
- 6. Andriulli A, Loperfido S, Napolitano G, Niro G, Valvano MR, Spirito F, et al. Incidence rates of post-ERCP complications. Am J Gastroenterol. 2007;102(8):1781-1788.
- 7. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. Lancet. 2015;386(9988):85-96.
- 8. Singh VK, Wu BU, Bollen TL, Repas K, Maurer R, Johannes RS, et al. Early SIRS is associated with severe acute pancreatitis. ClinGastroenterolHepatol. 2009;7(11):1247-1251.
- 9. Nieminen A, Maksimow M, Mentula P, Kyhälä L, Kylänpää ML, Puolakkainen P, et al. Circulating cytokines in predicting severity of AP. Crit Care. 2014;18(3):R104.
- 10. Cho IR, Chung MJ, Park JY, Bang S, Park SW, Song SY. Clinical significance of IL-6 in acute pancreatitis. Pancreatology. 2023;23(2):228-236.
- 11. Frossard JL, Steer ML, Pastor CM. Acute pancreatitis. Lancet. 2008;371(9607):143-152.
- 12. Petrov MS, Shanbhag S, Chakraborty M, Phillips AR, Windsor JA. Organ failure and infection of necrosis as determinants of mortality. Gastroenterology. 2010;139(3):813-820.
- 13. Whitcomb DC. Clinical practice. Acute pancreatitis. N Engl J Med. 2006;354(20):2142-2150.
- 14. Forsmark CE, Vege SS, Wilcox CM. Acute pancreatitis. N Engl J Med. 2016;375(20):1972-1981.
- 15. Bhatia M. Inflammatory response in pancreatic acinar injury. Scand J Surg. 2005;94(2):97-102.
- 16. Dambrauskas Z, Giese N, Gulbinas A, Barauskas G, Poch B, et al. Cytokine expression in mild vs severe AP. World J Gastroenterol. 2010;16(15):1845-1853.
- 17. Chen CC, Wang SS, Lee FY. Proinflammatory cytokines in prognosis of AP. Am J Gastroenterol. 1999;94(1):213-218.
- 18. Mayer J, Rau B, Gansauge F, Beger HG. Inflammatory mediators in human AP. Gut. 2000;47(4):546-552.
- 19. Staubli SM, Oertli D, Nebiker CA. Laboratory markers predicting severity of AP. Crit Rev Clin Lab Sci. 2015;52(6):273-283.
- 20. Working Group IAP/APA Guidelines. IAP/APA evidence-based guidelines for management of AP. Pancreatology. 2013;13(4 Suppl 2):e1-e15.