

Comparative Analysis of Biochemical Markers in Differentiating Gallstone from Non-Gallstone Acute Pancreatitis: A Prospective Observational Study

Srideepalaxmi G¹, S. Sreedevi², Riya Paul³

¹Post graduate, Department of General Surgery, Government, Mohan Kumaramangalam Medical College Hospital, Salem, Tamilnadu, India. ²Professor of General Surgery, Department of General Surgery, Government, MohanKumaramangalam Medical College Hospital, Salem, Tamilnadu, India.

Abstract

Background: Acute pancreatitis (AP) is a common gastrointestinal emergency with variable severity and etiology. Rapid differentiation between gallstone and non-gallstone pancreatitis is crucial for guiding targeted therapeutic decisions, such as urgent endoscopic retrograde cholangiopancreatography (ERCP) and same-admission cholecystectomy. The aim and objective are to evaluate the clinical utility of traditional and emerging biochemical markers in diagnosing acute pancreatitis. To establish biochemical cut-offs to differentiate gallstone-induced pancreatitis from non-gallstone etiologies. **Material and Methods:** A prospective observational study was conducted in the Department of General Surgery, GMKMC Hospital, Salem, over a 2-year period (January 2023 to January 2025). Fifty patients with clinically proven acute pancreatitis meeting the Revised Atlanta Criteria were included. Baseline demographics, clinical parameters, liver function tests (ALT, AST, ALP, bilirubin), pancreatic enzymes (amylase, lipase), and C-reactive protein (CRP) were analyzed. CECT, MRCP, and therapeutic ERCP were utilized as indicated. **Results:** Out of 50 patients, 28 (56%) were diagnosed with gallstone pancreatitis and 22 (44%) with non-gallstone pancreatitis. Female gender was significantly associated with gallstone etiology (85.7% vs. 14.3% in males, $p < 0.001$), whereas male gender and alcohol consumption were strongly associated with non-gallstone etiology (59.1% alcohol use, $p < 0.05$). Liver transaminases and cholestatic markers showed statistically significant elevations in the gallstone group: ALT (164.99 ± 44.11 vs. 122.15 ± 41.54 U/L, $p = 0.0010$), AST (137.79 ± 31.69 vs. 90.71 ± 17.82 U/L, $p < 0.001$), ALP (262.13 ± 26.35 vs. 226.71 ± 57.44 U/L, $p = 0.0122$), and Total Bilirubin (1.99 ± 0.74 vs. 0.70 ± 0.61 mg/dL, $p < 0.001$). Serum amylase, lipase, and CRP levels showed no significant intergroup differences. Among 10 patients with choledocholithiasis undergoing ERCP, successful cannulation in 8 (80%) resulted in significant post-procedure reductions in serum amylase and lipase ($p < 0.05$). **Conclusion:** Early measurement of hepatobiliary enzymes specifically ALT, AST, ALP, and total bilirubin reliably differentiates gallstone from non-gallstone acute pancreatitis at presentation. Female gender is strongly associated with gallstone etiology, while male sex and alcohol abuse correlate with non-gallstone acute pancreatitis. Integrating early biochemical profiling with selective imaging optimizes management pathways and patient outcomes.

Keywords: Acute pancreatitis, gallstone pancreatitis, alanine aminotransferase (ALT), bilirubin, biomarkers, choledocholithiasis, ERCP.

Received: 20 July 2026

Revised: 10 August 2026

Accepted: 25 August 2026

Published: 09 September 2026

INTRODUCTION

Acute pancreatitis (AP) is an increasing acute condition of the gastrointestinal tract, and one of the most common, precipitating a number of acute hospital admissions globally. It is the clinical manifestation of an acute inflammatory process of the pancreas and corresponds to a wide spectrum of disease, from self-limited and mild to fulminant and life-threatening necrotizing pancreatitis associated with multi-organ failure, and with high mortality rates. A precise etiology is the key factor in the onset of the disease as fundamental differences in the management of the disease and prognosis depend on the underlying etiology.^[1-5] In most high income countries and in many developing ones, the prevalence of gallstones is 40-70% of the cases, with chronic and acute heavy alcohol consumption being the next largest group of causes (25-35%). Other causes include hypertriglyceridemia, history of hypercalcemia, reaction to a drug, trauma, mechanical injury from an ERCP, autoimmune

diseases and idiopathic causes. Pathophysiologically, the development of gallstone pancreatitis is the result of a migrating gallstone or biliary sludge transiently blocking the ampulla of Vater which results in a sudden increase in ampullary intraluminal pressure or bile reflux into the pancreatic duct. This causes a cascade of local and systemic inflammation, due to bursts of intracellular calcium which activate trypsinogen,

Address for correspondence: Dr. Srideepalaxmi G,
Post graduate, Department of General Surgery, Government, Mohan
Kumaramangalam Medical College Hospital, Salem, Tamilnadu, India.
E-mail: deepalaxmi41106@gmail.com

DOI:
10.21276/amit.2026.v13.i3.1018

How to cite this article: Srideepalaxmi G, Sreedevi S, Paul R. Comparative Analysis of Biochemical Markers in Differentiating Gallstone from Non-Gallstone Acute Pancreatitis: A Prospective Observational Study. Acta Med Int. 2026;13(3):54-59.

leading to autodigestion. Small stones often pass in the duodenum without symptoms and a later imaging may not be able to detect it, so early biochemical markers are essential. In clinical practice, the early and correct differentiation of the gallstone pancreatitis from non-gallstone pancreatitis directly influences clinical decision-making. Once the biliary cause is determined, timely interventions are possible, including same admission laparoscopic cholecystectomy with sphincterotomy, which prevents further attacks, in patients with gallstones and pancreatitis, and intervention (within 24-72 hours) using endoscopic retrograde cholangiopancreatography (ERCP, with sphincterotomy) where there is an acute cholangitis or a persistent biliary obstruction. In contrast, treatment for non-gallstone pancreatitis is aimed at the treatment of the underlying cause. If inappropriate biliary-specific procedures are performed in people without gallstones, the cost of care, procedure-related complications, and length of hospital stay are all higher.^[6-15] The biochemical markers that are available are attractive for diagnosis because they are easily accessible, inexpensive, minimally invasive and provide immediate results. While pancreatic enzymes (amylase and lipase) confirm the presence of pancreatic inflammation, traditional liver function tests—alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), and total bilirubin—reflect transient biliary obstruction and hepatocellular injury. The main objective of this study is to evaluate the clinical utility of biochemical markers in diagnosing acute pancreatitis and establishing critical cut-offs to differentiate gallstone pancreatitis from other non-gallstone etiologies in a prospective clinical cohort.^[16-22]

MATERIALS AND METHODS

Study Design and Setting: This study was planned as a prospective observational clinical study in Department of General Surgery at Government Mohan Kumaramangalam Medical College Hospital (GMKMCH), Salem, Tamilnadu, India.

Study Duration & Population: The time of the study was 2 years, January 2023 – January 2025. Analysis was based on data from the study population (adult patients) admitted to the general surgical wards for clinical diagnosis along with biochemical confirmation of acute pancreatitis.

Selection Criteria: The inclusion criteria were all consecutive patients with clinically proven AP based on the criteria of the Revised Atlanta Criteria (which involve at least two of the following three characteristics: typical back and epigastric pain, elevated amylase/lipase > 3 x upper limit of normal, or typical findings on cross sectional imaging).

Exclusion Criteria: Patients with a diagnosis of chronic

pancreatitis, patients with severe complications of organ failure before the index admission, and patients who refused to sign informed consent.

Sample Size Calculation: The sample size estimation was based on data showing that 48% of patients with gallstone pancreatitis have elevated ALT levels ≥ 150 U/L, whereas only 4% of patients with non-gallstone pancreatitis do. With a conventional statistical model for independent proportions, an $\alpha = 0.05$ and a power of 80%, a sample size of at least 15 patients (30 overall) was needed. The number of patients in the study was increased from 20 to 50 to ensure sufficient numbers for analysis and to further improve the precision of the statistical results.

Study Methodology and Data Collection: Data was collected at admission, and included detailed demographic, clinical history and physical examination findings. Samples of venous blood were collected within 24 hours of the presentation for biochemical studies, such as serum amylase, serum lipase, liver function test (ALT, AST, ALP, total bilirubin), C-reactive protein (CRP), blood urea nitrogen (BUN), serum creatinine, and electrolyte panels. All patients had transabdominal ultrasonography (US) done upon admission. Contrast enhanced computed tomography (CECT) of abdomen was carried out in patients needing severity grade or in those with persistent diagnostic doubts after 48-72 hours. In patients with a suspected choledocholithiasis, magnetic resonance cholangiopancreatography (MRCP) was performed and therapeutic ERCP was attempted in both patients with proven ductal stones and in those with acute cholangitis.

Ethical Principles and Statistical Analysis: The study protocol was approved by the Institutional Ethics Committee of GMKMCH. Written informed consent was obtained from all participants. Data were analyzed using SPSS Version 25.0. Categorical variables were compared using Chi-square or Fisher's exact tests. Continuous variables were presented as mean $\pm$ SD and compared using Student's t-test or paired t-tests where appropriate ($p < 0.05$ considered statistically significant).

RESULTS

A total of 50 patients with acute pancreatitis were enrolled. Based on clinical evaluation, laboratory investigations, and imaging findings, 28 patients (56.0%) were categorized into the gallstone pancreatitis group and 22 patients (44.0%) into the non-gallstone pancreatitis group.

Demographic and Clinical Profile: The age distribution of the study cohort revealed that the majority of patients belonged to the middle age groups: 41-50 years (28.0%) and 51-60 years (28.0%), followed by 31-40 years (26.0%), ≤ 30 years (10.0%), and > 60 years (8.0%) [Table 1]. Overall, females comprised 52.0% ($n = 26$) and males 48.0% ($n = 24$) of the study population [Table 2].

Table 1: Distribution of Study Participants According to Age Group and Gender

Age Group (Years)	Frequency (n)	Percentage (%)
≤ 30	5	10.0%
31-40	13	26.0%
41-50	14	28.0%
51-60	14	28.0%
> 60	4	8.0%

Gender	Frequency (n)	Percentage (%)
Female	26	52.0%
Male	24	48.0%

Association of Gender and Lifestyle Factors with Etiology: A statistically significant association was observed between gender and the etiology of acute pancreatitis ($p < 0.001$). Among female patients, 24 out of 26 (92.3%) suffered from gallstone pancreatitis. In contrast, among male patients, non-gallstone pancreatitis was predominant,

accounting for 20 out of 24 cases (83.3%) [Table 3, Figure 1]. Assessment of alcohol usage revealed that 59.1% ($n = 13$) of patients in the non-gallstone group had a documented history of alcohol intake, compared to only 21.4% ($n = 6$) in the gallstone pancreatitis group [Table 4].

Table 2: Distribution of Patients According to Gender and Etiology of Pancreatitis

Gender	Gallstone (n=28)	Non-Gallstone (n=22)	Total (N=50)	p-value
Female	24 (92.3%)	2 (7.7%)	26 (100%)	< 0.001
Male	4 (16.7%)	20 (83.3%)	24 (100%)	
Etiology Group	Alcohol Use: Yes (%)	Alcohol Use: No (%)	Total	
Gallstone Pancreatitis	6 (21.4%)	22 (78.6%)	28	
Non-Gallstone Pancreatitis	13 (59.1%)	9 (40.9%)	22	

Disease Severity and CT Imaging Findings: According to the Revised Atlanta Criteria, disease severity was stratified across both groups. Mild pancreatitis was present in 53.6% of gallstone and 54.5% of non-gallstone cases. Moderately severe cases constituted 25.0% and 31.8%, while severe

pancreatitis occurred in 21.4% of gallstone cases compared to 13.6% in non-gallstone cases [Table 5, Figure 3]. Mean hospital stay was 5.68 ± 1.89 days for gallstone cases and 6.50 ± 2.35 days for non-gallstone cases.

Table 3: Distribution of Patients According to Etiology and Severity of Acute Pancreatitis

Etiology Group	Mild (%)	Moderately Severe (%)	Severe (%)
Gallstone Pancreatitis (n=28)	15 (53.6%)	7 (25.0%)	6 (21.4%)
Non-Gallstone Pancreatitis (n=22)	12 (54.5%)	7 (31.8%)	3 (13.6%)

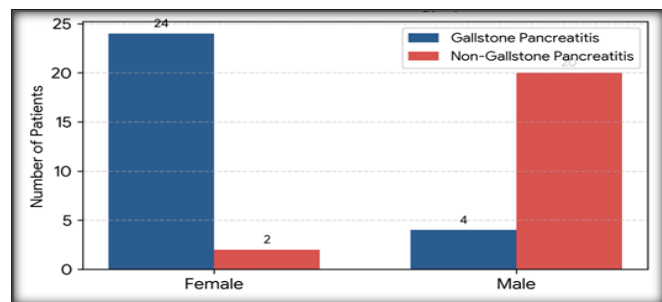


Figure 1: Distribution of Etiology by Gender

Comparison of Admission Biochemical Markers: Comparison of biochemical parameters drawn at admission demonstrated marked, statistically significant differences in hepatobiliary and liver transaminase enzymes between the two etiologies [Table 6, Figure 2]. Alanine aminotransferase (ALT) was significantly elevated in the gallstone group compared to non-gallstone cases (164.99 ± 44.11 U/L vs. 122.15 ± 41.54 U/L, $p = 0.0010$). Similarly, aspartate aminotransferase (AST) was markedly higher in gallstone

cases (137.79 ± 31.69 U/L vs. 90.71 ± 17.82 U/L, $p < 0.001$). Cholestatic markers also showed significant elevations: Alkaline Phosphatase (ALP) was 262.13 ± 26.35 U/L in gallstone vs. 226.71 ± 57.44 U/L in non-gallstone ($p = 0.0122$), and Total Bilirubin was 1.99 ± 0.74 mg/dL vs. 0.70 ± 0.61 mg/dL ($p < 0.001$). Conversely, serum amylase, lipase, and CRP showed no significant intergroup differences.

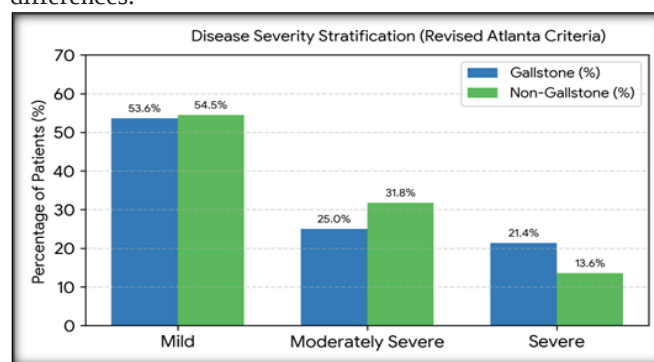


Figure 2: Disease Severity Stratification by Etiology

Table 4: Comparison of Biochemical and Inflammatory Biomarkers Between Gallstone and Non-Gallstone Pancreatitis

Biomarker	Gallstone (Mean $\pm$ SD)	Non-Gallstone (Mean $\pm$ SD)	t-statistic	p-value
Serum Amylase (U/L)	783.28 $\pm$ 231.34	744.45 $\pm$ 240.44	0.576	0.5673
Serum Lipase (U/L)	990.83 $\pm$ 312.97	972.25 $\pm$ 249.73	0.228	0.2100
ALT / SGPT (U/L)	164.99 $\pm$ 44.11	122.15 $\pm$ 41.54	3.472	0.0010
AST / SGOT (U/L)	137.79 $\pm$ 31.69	90.71 $\pm$ 17.82	6.284	< 0.001
ALP (U/L)	262.13 $\pm$ 26.35	226.71 $\pm$ 57.44	2.603	0.0122
Total Bilirubin (mg/dL)	1.99 $\pm$ 0.74	0.70 $\pm$ 0.61	6.541	< 0.001
CRP (mg/L)	139.61 $\pm$ 38.44	134.96 $\pm$ 32.15	0.465	0.6438

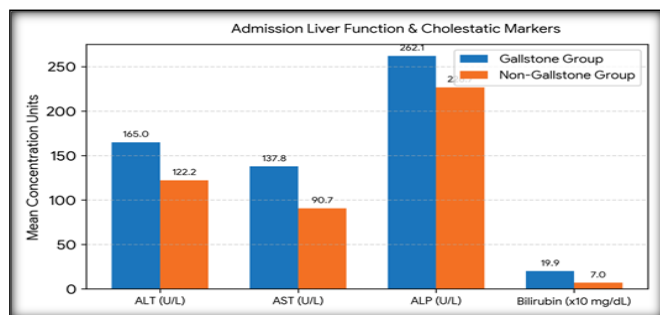


Figure 3: Admission Liver Function & Cholestatic Markers Comparison

Interventional Outcomes: MRCP and ERCP

Among the 28 patients with CT findings of acute pancreatitis with cholelithiasis, MRCP was performed, confirming choledocholithiasis in 10 patients (35.7%). These 10 patients underwent therapeutic ERCP, achieving successful bile duct cannulation and stone extraction in 8 cases (80.0%) [Table 7]. In these 8 patients, serial pancreatic enzyme levels showed dramatic and statistically significant reductions ($p < 0.05$). Mean serum amylase dropped from 775.25 U/L pre-procedure to 167.00 U/L post-procedure, and mean serum lipase decreased from 927.63 U/L to 233.75 U/L [Table 8, Figure 4].

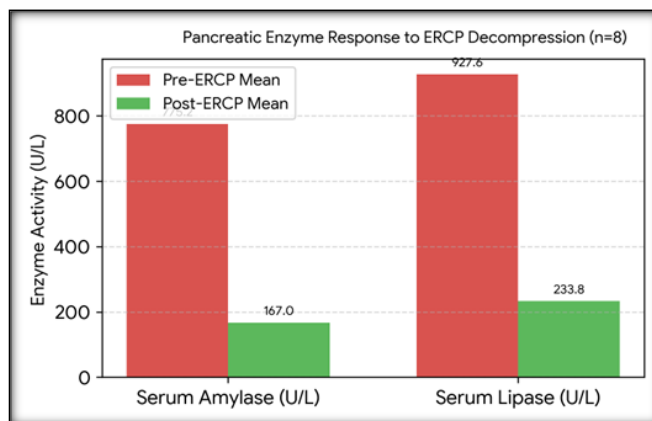


Figure 4: Pre- vs. Post-ERCP Pancreatic Enzyme Response

Clinical Outcomes and Mortality: Overall recovery without intensive care was achieved in 67.9% of gallstone patients and 81.8% of non-gallstone patients. ICU admission was required in 25.0% ($n = 7$) of gallstone cases vs. 18.2% ($n = 4$) of non-gallstone cases. Two deaths occurred in the gallstone group (7.1% mortality), both attributable to severe necrotizing pancreatitis with persistent multi-organ failure, whereas zero mortality was observed in the non-gallstone group [Table 9, Figure 5].

Table 5: Comparison of MRCP Findings, ERCP Outcomes, and Serum Biomarker Levels Before and After ERCP

Investigation / Intervention		Category / Outcome	Frequency (%)
MRCP Results (n=28)		Choledocholithiasis / CBD Stones	10 (35.7%)
		No CBD Stone Detected	18 (64.3%)
ERCP Results (n=10)		Successful Cannulation & Extraction	8 (80.0%)
		Unsuccessful Cannulation	2 (20.0%)
Biomarker	Pre-Procedure Mean	Post-Procedure Mean	p-value (Paired t-test)
Serum Amylase (IU/L)	775.25	167.00	< 0.05
Serum Lipase (IU/L)	927.63	233.75	< 0.05

Table 6: Comparison of Clinical Outcomes Between Gallstone and Non-Gallstone Pancreatitis

Clinical Outcome	Gallstone Pancreatitis (n=28)	Non-Gallstone Pancreatitis (n=22)
Recovered (Ward Care)	19 (67.9%)	18 (81.8%)
ICU Admission Required	7 (25.0%)	4 (18.2%)
Mortality	2 (7.1%)	0 (0.0%)

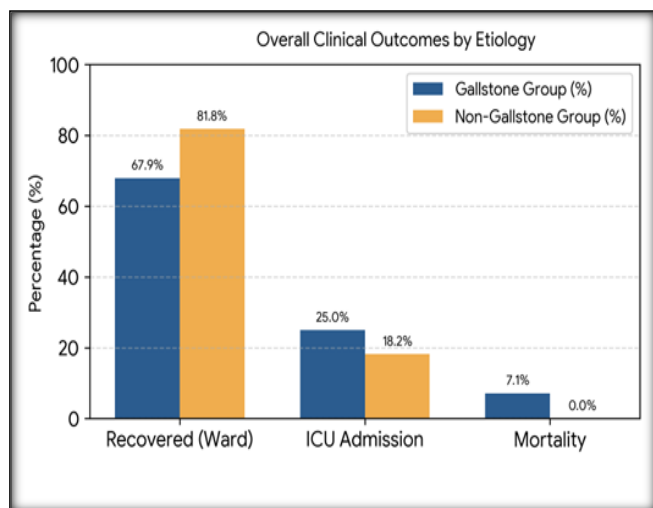


Figure 5: Overall Clinical Outcomes by Etiology Group

DISCUSSION

The first aim of this prospective observational study was to assess the clinical profile, diagnostic significance and prognosis of biochemical markers in differentiating gallstone acute pancreatitis from non-gallstone causes. Of the 50 patients, gallstone pancreatitis (GWAP) accounted for 56.0% and non-gallstone pancreatitis (NWAP) for 44.0%. This distribution is very similar to the world epidemiology data, in which gallstones account for most of the acute pancreatitis in the world.^[23-26]

Demographic analysis showed acute pancreatitis affects more in the middle age group as 56.0% of the group was in 41 to 60 age group. Importantly, our study revealed a highly significant, statistically significant association between being female and the etiology of gallstones ($p < 0.001$) – 92.3% of female patients had a diagnosis of biliary pancreatitis. By contrast, 83.3% of male patients developed non-gallstone pancreatitis and chronic alcohol consumption was a major factor in the development of non-gallstone pancreatitis (59.1% of cases). Results obtained here are

in line with the classical observational literature which shows that female sex hormones and an altered bile acid dynamics are associated with risk of gallstones, while male gender is associated with increased risk of alcohol-induced pancreatic acinar injury.^[27-30]

One of the most important clinical problems in the treatment of acute pancreatitis is the early differentiation of etiology. Pancreatic enzymes (amylase and lipase) are not sensitive and specific for the diagnosis of pancreatic inflammation. In our study, there were no statistically significant differences in the levels of serum amylase and lipase between gallstone group and non-gallstone group ($p = 0.5673$, $p = 0.210$, respectively). Moreover, when we compared C-reactive protein (CRP), a known inflammatory marker, to reflect the intensity of systemic inflammation, there was no difference between groups (139.61 vs. 134.96 mg/L, $p = 0.6438$), indicating that the intensity of systemic inflammation is determined by the degree of necrosis of the acini, not the initial cause.^[31,32]

Liver transaminases and cholestatic markers gave very significant differential diagnosis, quite the opposite. Admission ALT was markedly higher in gallstone pancreatitis compared to non-gallstone cases (164.99 ± 44.11 U/L vs. 122.15 ± 41.54 U/L, $p = 0.0010$). This result is a strong confirmation of the current clinical guidelines that have indicated that a rise in ALT > 150 U/L in the first 24-48 hours is a valuable indicator of biliary pancreatitis. The shortening of a gall stone through the duct causes acute biliary hypertension and micro-necrosis of hepatic cells which causes immediate release of ALT and AST in circulation. In addition, all of the three tests (AST, ALP, and total bilirubin) were significantly elevated in the gallstone group ($P < 0.05$), suggesting acute mechanical obstruction of the common bile duct.^[33]

Our interventional subset is an excellent example of the therapeutic effect of early etiologic diagnosis. MRCP identified choledocholithiasis in 35.7% of gallstone patients leading to therapeutic ERCP of the biliary tract in targeted patients. 80% of these patients achieved endoscopic decompression and had a quick, highly significant decrease in serum pancreatic enzymes and stabilization. Lastly, overall clinical outcomes revealed that those with gallstone pancreatitis were just more likely to have been admitted to the ICU (25.0% vs. 18.2%) and were more likely to die (7.1% vs. 0%). The present higher morbidity in biliary cases is to highlight the need for early diagnosis, early clearance of ductal obstruction and same admission cholecystectomy.^[34]

CONCLUSION

Early biochemical profiling is a crucial and very useful tool to determine the etiology of AP on admission.

Diagnosis and extent of inflammation can be confirmed with serum markers of amylase, lipase and CRP, hepatobiliary markers, such as Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP) and Total Bilirubin, are also significantly elevated, but reliably differentiate gallstone from non-gallstone causes. Incorporation of the initial bedside ultrasonography and early

ALT and total bilirubin measurements can facilitate fast etiologic classification and timely therapeutic intervention like ERCP and SACH, which will ultimately improve clinical outcomes.

Acknowledgements: The authors express their sincere gratitude to the patients who participated in this study, as well as the medical, surgical, and laboratory staff of Government Mohan Kumaramangalam Medical College & Hospital (GMKMCH), Salem, Tamil Nadu, for their dedicated clinical support and assistance in data collection.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Banks, P. A., Bollen, T. L., Dervenis, C., et al. (2013). Classification of acute pancreatitis--2012: revision of the Atlanta classification and consensus. *Gut*, 62(1), 102-111.
2. Forsmark, C. E., Vege, S. S., & Wilcox, C. M. (2016). Acute Pancreatitis. *N Engl J Med*, 375(20), 1972-1981.
3. Sakorafas, G. H., Tsiotou, A. G., & Peros, G. (2007). Mechanisms of pain in chronic pancreatitis. *J Clin Gastroenterol*, 41(7), 689-699.
4. Yadav, D., & Lowenfels, A. B. (2013). The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*, 144(6), 1252-1261.
5. Tenner, S., Baillie, J., DeWitt, J., et al. (2013). ACG guideline: management of acute pancreatitis. *Am J Gastroenterol*, 108(9), 1400-1416.
6. Gurusamy, K. S., Davidson, C., Gluud, C., & Davidson, B. R. (2013). Early vs delayed laparoscopic cholecystectomy. *Cochrane Database Syst Rev*, (6), CD005440.
7. Tse, F., Yuan, Y., Moayyedi, P., & Leontiadis, G. I. (2012). Guidewire-assisted cannulation for post-ERCP pancreatitis prevention. *Cochrane Database Syst Rev*, 12, CD009662.
8. Xiao, A. Y., Tan, M. L., Wu, L. M., et al. (2016). Global incidence and mortality of pancreatic diseases. *Lancet Gastroenterol Hepatol*, 1(1), 45-55.
9. Blamey, S. L., Imrie, C. W., O'Neill, J., et al. (1984). Prognostic factors in acute pancreatitis. *Gut*, 25(12), 1340-1346.
10. Dumnicka, P., Maduzia, D., Ceranowicz, P., et al. (2017). Inflammation, coagulation and endothelial injury in acute pancreatitis. *Int J Mol Sci*, 18(2), 354.
11. Mofidi, R., Duff, M. D., Wigmore, S. J., et al. (2006). Early SIRS, severity of organ dysfunction and death in AP. *Br J Surg*, 93(6), 738-744.
12. Tenner, S., Dubner, H., & Steinberg, W. (1994). Predicting gallstone pancreatitis with laboratory tests: a meta-analysis. *Am J Gastroenterol*, 89(10), 1863-1866.
13. Shaheen, N. J., Hansen, R. A., Morgan, D. R., et al. (2006). The burden of gastrointestinal and liver diseases. *Am J Gastroenterol*, 101(9), 2128-2138.
14. Zhao, K., Adam, S. Z., Keswani, R. N., et al. (2015). Acute Pancreatitis: Revised Atlanta Classification and imaging. *AJR Am J Roentgenol*, 205(1), W32-W41.
15. Zhou, B., Zhang, J., Li, G., et al. (2025). The global, regional, and national burden of pancreatitis 1990 to 2021. *J Gastroenterol Hepatol*, 40(5), 1297-1306.
16. Iannuzzi, J. P., King, J. A., Leong, J. H., et al. (2022). Global incidence of acute pancreatitis is increasing over time. *Gastroenterology*, 162(1), 122-134.
17. Li, T., Qin, C., Zhao, B. et al. (2024). Global and regional burden of

- pancreatitis: trends and projections to 2050. *BMC Gastroenterol*, 24, 398.
18. Zilio, M. B., Eyff, T. F., Azeredo-Da-Silva, A. L. F., et al. (2019). Meta-analysis of the aetiology of acute pancreatitis. *HPB*, 21(3), 259-267.
 19. Nesvaderani, M., Eslick, G. D., Vagg, D., et al. (2015). Epidemiology, aetiology and outcomes of acute pancreatitis. *Int J Surg*, 23, 68-74.
 20. Fan, Z., Zhang, Y., Li, J., et al. (2025). Global burden of hypertriglyceridemia-induced acute pancreatitis. *Sci China Life Sci*, 68(10), 3010-3020.
 21. Akca, S., Ocal, S., Koc, S.G. et al. (2025). Epidemiology of acute pancreatitis: age and gender influence. *BMC Gastroenterol*, 25, 627.
 22. Samanta, J., Dhaka, N., Gupta, P., et al. (2019). Outcome between alcohol and gallstone pancreatitis. *JGH Open*, 3(4), 338-343.
 23. Patel, M. L., Shyam, R., Atam, V., et al. (2022). Clinical profile, etiology, and outcome of acute pancreatitis. *Ann Afr Med*, 21(2), 118-123.
 24. da Costa, D. W., Bouwense, S. A., Schepers, N. J., et al. (2015). Same-admission vs interval cholecystectomy (PONCHO). *Lancet*, 386(10000), 1261-1268.
 25. Roberts, S. E., Akbari, A., Thorne, K., et al. (2013). Acute pancreatitis: impact of social deprivation and alcohol. *Gut*, 62(7), 1022-1030.
 26. Peery, A. F., Crockett, S. D., Murphy, C. C., et al. (2019). Burden and cost of gastrointestinal diseases. *Gastroenterology*, 156(1), 254-272.
 27. Garg, P. K., & Singh, V. P. (2019). Organ failure and infection in acute pancreatitis. *Gastroenterol Clin North Am*, 48(1), 35-54.
 28. Sadr-Azodi, O., Andrén-Sandberg, A., & Orsini, N. (2012). Cigarette smoking, alcohol and risk of AP. *Gut*, 61(10), 1443-1447.
 29. Yadav, D., & Whitcomb, D. C. (2010). The role of alcohol and smoking in pancreatitis. *Nat Rev Gastroenterol Hepatol*, 7(3), 131-145.
 30. Krishna, S. G., Kamboj, A. K., Hart, P. A., et al. (2017). Epidemiology and outcomes of acute pancreatitis in US. *Dig Dis Sci*, 62(5), 1241-1249.
 31. Gomez, D., Addison, A., De Rosa, A., et al. (2012). ALT levels as predictor of biliary etiology in AP. *HPB Surg*, 2012, 510317.
 32. Working Party of the BSG (2005). UK guidelines for the management of acute pancreatitis. *Gut*, 54(suppl 3), iii1-iii9.
 33. Khanna, A. K., Meher, S., Prakash, S., et al. (2013). Comparison of Ranson, APACHE II, BISAP, MCTSI in AP. *Gastroenterol Res Pract*, 2013, 367581.
 34. van Santvoort, H. C., Besselink, M. G., Bollen, T. L., et al. (2011). Compression and drainage in acute pancreatitis. *N Engl J Med*, 362(16), 1491-1502.