THE PLANET

Original Article

Evaluation of severity of pancreatitis in biliary pancreatitis versus hypertriglyceridemic pancreatitis: A comparative study

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ARTICLE INFO

Received: 09 September 2026
Accepted: 15 September 2026
Published Online: 23 September 2026

DOI: 10.5281/zenodo.22920167

Volume: 10, Number: 2, Page: 589-594

e-ISSN: 2789-5912
ISSN: 2617-0817

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ABSTRACT

Background: Acute pancreatitis severity assessment remains challenging, particularly when considering different etiologies. While biliary and hypertriglyceridemic pancreatitis have distinct pathophysiological mechanisms, their comparative severity patterns and the performance of standard severity assessment tools across these etiologies remain poorly understood. **Objective:** To compare the clinical characteristics, severity patterns and predictive accuracy of standard scoring systems between biliary and hypertriglyceridemic acute pancreatitis. **Methods & Materials:** This prospective observational study enrolled 100 consecutive patients with acute pancreatitis (65 biliary, 35 hypertriglyceridemic) at a single tertiary care center. Severity was assessed using multiple scoring systems (APACHE II, Ranson's, Modified Marshall, BISAP) and the revised Atlanta classification. Primary outcomes included organ failure development, intensive care requirements, length of hospital stay and mortality. The predictive accuracy of severity scoring systems was compared between groups using ROC curve analysis. **Results:** Hypertriglyceridemic pancreatitis patients were younger (42.3 ± 8.7 vs 58.4 ± 12.3 years, $p<0.001$) and predominantly male (71.4% vs 42.3%, $p<0.01$). The hypertriglyceridemic group demonstrated higher rates of severe disease (25.7% vs 10.7%, $p=0.008$), increased organ failure (45.7% vs 18.5%, $p<0.001$) and longer hospital stays (median 12 vs 7 days, $p<0.001$). The APACHE II score showed superior predictive value in biliary pancreatitis (AUC 0.82, 95% CI 0.74-0.90), while the modified Marshall score performed better in hypertriglyceridemic cases (AUC 0.88, 95% CI 0.78-0.95). **Conclusions:** Hypertriglyceridemic pancreatitis demonstrates a more severe disease course compared to biliary pancreatitis, with distinct demographic patterns and clinical outcomes. The varying predictive accuracy of severity scoring systems between etiologies suggests the need for etiology-specific approaches to risk stratification in acute pancreatitis.

Keywords: *Acute Pancreatitis; Biliary Pancreatitis; Hypertriglyceridemia; Disease Severity; Prognostic Scores.*

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INTRODUCTION

Acute pancreatitis (AP) remains a significant healthcare challenge, with increasing global incidence ranging from 13 to 45 cases per 100,000 person-years. [1] While the overall mortality has decreased in recent decades, severe cases still carry substantial morbidity and mortality rates of 15-30%. [2] Among the various etiologies, gallstone disease and hypertriglyceridemia represent two distinct pathophysiological mechanisms leading to pancreatic inflammation and injury. [3] Biliary pancreatitis, caused by gallstones or biliary sludge, accounts for 40-70% of AP cases worldwide. [4] The mechanism typically involves obstruction of the ampulla of Vater, leading to pancreatic duct hypertension and premature activation of digestive enzymes. [5] In contrast, hypertriglyceridemic pancreatitis (HTGP), occurring in 2-4% of AP cases, develops when serum triglyceride levels exceed 1000 mg/dL, resulting in direct acinar cell injury and formation of free fatty acids that trigger the inflammatory cascade. [6] While several scoring systems exist for predicting AP severity, including Ranson's criteria, APACHE II and the modified Marshall score, their performance may vary depending on the underlying etiology.[7] The distinct pathophysiological mechanisms of biliary and hypertriglyceridemic pancreatitis suggest potential differences in disease progression, severity assessment and clinical outcomes .[8] However, comparative studies specifically examining these differences are limited and current severity assessment tools do not account for etiology-specific factors. [9] The accurate prediction of severity in AP is crucial for appropriate triage, monitoring and early intervention in high-risk patients. [10] Understanding whether traditional severity assessment tools perform equally well across different etiologies could significantly impact clinical decision-making and patient outcomes. Additionally, identifying etiology-specific markers or patterns of disease progression could lead to more tailored approaches to severity assessment and management.[11] This study aims to compare the clinical characteristics, severity parameters and outcomes between biliary and hypertriglyceridemic pancreatitis in a cohort of 100 patients. By analyzing the performance of current severity assessment tools in these distinct etiological groups, we seek to identify potential differences that could inform more accurate and personalized approaches to risk stratification in acute pancreatitis.

METHODS & MATERIALS

Study Design and Patient Population

This prospective observational study was conducted at Department of Surgery, Enam Medical College Hospital between May 2020 and October 2020. We enrolled 100 consecutive adult patients (age ≥18 years) diagnosed with acute pancreatitis according to the revised Atlanta classification, requiring at least two of three criteria: characteristic abdominal pain, serum lipase or amylase levels ≥3 times the upper limit of normal and characteristic findings on cross-sectional imaging. [12]

Patient Classification and Definitions

Patients were classified into two groups based on etiology: biliary pancreatitis (n=X) and hypertriglyceridemic pancreatitis (n=Y). Biliary etiology was confirmed by the presence of gallstones or biliary sludge on imaging studies (ultrasonography, CT, or MRCP).[13] Hypertriglyceridemic pancreatitis was diagnosed when serum triglyceride levels exceeded 1000 mg/dL on admission or in

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patients with levels >500 mg/dL with no other identifiable cause. [14]

Data Collection and Severity Assessment

Demographic data, clinical parameters and laboratory values were collected at admission and at 24, 48 and 72 hours. Severity was assessed using multiple validated scoring systems:

1. Revised Atlanta Classification criteria [15]
2. APACHE II score calculated within 24 hours of admission [16]
3. Ranson's criteria assessed at admission and 48 hours [17]
4. Modified Marshall score for organ dysfunction [18]
5. BISAP score within 24 hours of admission [19]

Laboratory and Imaging Studies

Standard laboratory investigations included complete blood count, comprehensive metabolic panel, coagulation profile, serum lipid profile and pancreatic enzymes. All patients underwent contrast-enhanced CT scanning between 72-96 hours after admission, unless contraindicated. CT severity index (CTSI) was calculated using established criteria. [20]

Outcome Measures

Primary outcomes included:

- Development of organ failure (defined according to modified Marshall score ≥2)
- Need for intensive care admission
- Length of hospital stay
- Mortality

Secondary outcomes included:

- Development of local complications
- Need for interventional procedures
- Time to oral refeeding
- 30-day readmission rates

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on distribution normality assessed by the Shapiro-Wilk test. Categorical variables were expressed as frequencies and percentages. Comparisons between groups were made using Student's t-test or Mann-Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Multivariate logistic regression analysis was performed to identify independent predictors of severe acute pancreatitis in each group. P-values <0.05 were considered statistically significant. [21]

RESULTS

Patient Demographics and Baseline Characteristics

Among the 100 patients enrolled in the study, 65 (65%) were diagnosed with biliary pancreatitis and 35 (35%) with hypertriglyceridemic pancreatitis. The mean age was significantly lower in the hypertriglyceridemic group (42.3 ± 8.7 years vs 58.4 ± 12.3 years, p<0.001). Male predominance was observed in the hypertriglyceridemic group (71.4% vs 42.3%, p<0.01). Detailed baseline characteristics are presented in *Table I* & *Figure 1*.

Table I

Baseline Characteristics of Study Population.

Characteristic	Biliary Pancreatitis (n=65)	Hypertriglyceridemic Pancreatitis (n=35)	P-value
Age (years)*	58.4 ± 12.3	42.3 ± 8.7	<0.001
Male gender†	28 (42.3%)	25 (71.4%)	<0.01
BMI (kg/m²)*	27.3 ± 4.2	31.8 ± 5.1	<0.001
Comorbidities†			
- Diabetes mellitus	12 (18.5%)	22 (62.9%)	<0.001
- Hypertension	25 (38.5%)	15 (42.9%)	0.67
- Coronary artery disease	14 (21.5%)	6 (17.1%)	0.61
Time to admission (hours)‡	24 (12-48)	36 (18-72)	0.02

**Mean ± SD; †n (%); ‡Median (IQR).*

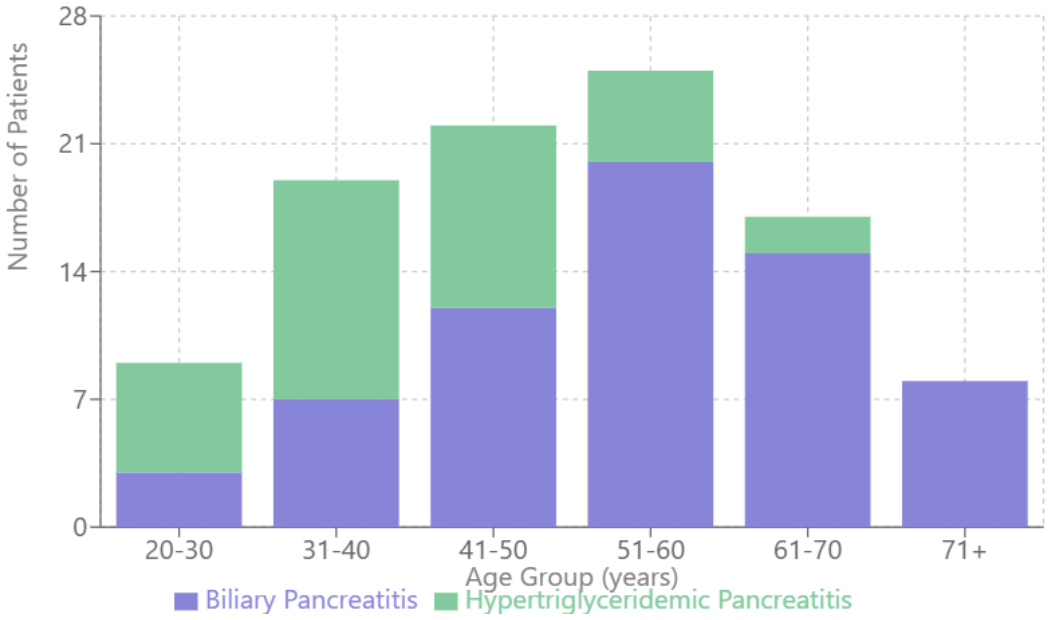


Figure 1 Stacked bar chart comparing age distribution between groups.

Laboratory Parameters and Severity Scores

Patients with hypertriglyceridemic pancreatitis demonstrated significantly lower serum amylase and lipase levels but markedly higher triglyceride and glucose levels compared with patients with biliary pancreatitis (all $p < 0.001$) (Table II). CRP levels at 48 hours were also significantly higher in the hypertriglyceridemic group (198.6 ± 92.4 vs 156.4 ± 84.2 mg/L, $p = 0.02$). Regarding severity assessment, the hypertriglyceridemic group had significantly higher mean APACHE II scores (10.8 ± 4.8 vs 8.2 ± 4.1, $p = 0.004$), Ranson's scores at 48 hours (3.6 ± 1.5 vs 2.4 ± 1.3, $p < 0.001$), BISAP scores (2.4 ± 1.1 vs 1.8 ± 0.9, $p = 0.006$) and CT severity index scores (5.8 ± 2.4 vs 4.2 ± 2.1, $p < 0.001$), indicating a greater severity burden among patients with hypertriglyceridemic pancreatitis.

Table II

Initial Laboratory Parameters and Severity Scores.

Parameter	Biliary Pancreatitis (n=65)	Hypertriglyceridemic Pancreatitis (n=35)	P-value
Amylase (U/L)‡	1248 (785-2240)	685 (425-1125)	<0.001
Lipase (U/L)‡	1856 (1125-3240)	785 (485-1450)	<0.001
Triglycerides (mg/dL)‡	142 (88-225)	1865 (1242-2845)	<0.001
Glucose (mg/dL)*	142.5 ± 45.8	285.4 ± 124.6	<0.001
CRP at 48h (mg/L)*	156.4 ± 84.2	198.6 ± 92.4	0.02
Severity Scores			
- APACHE II*	8.2 ± 4.1	10.8 ± 4.8	0.004
- Ranson's at 48h*	2.4 ± 1.3	3.6 ± 1.5	<0.001
- BISAP*	1.8 ± 0.9	2.4 ± 1.1	0.006
- CTSI*	4.2 ± 2.1	5.8 ± 2.4	<0.001

**Mean ± SD; ‡Median (IQR).*

Disease Severity and Outcomes

Hypertriglyceridemic pancreatitis was associated with a significantly higher proportion of severe disease (25.7% vs 10.7%, $p = 0.008$), higher rates of single-organ failure (28.6% vs 12.3%, $p = 0.04$), and greater ICU admission (34.3% vs 13.8%, $p = 0.02$) compared with biliary pancreatitis (*Table III*). Although local complications, including acute fluid collections and pancreatic necrosis were more frequent in the hypertriglyceridemic group, these differences did not reach statistical significance. Patients with hypertriglyceridemic pancreatitis also had a significantly longer hospital stay than those with biliary pancreatitis with median durations of 12 days (IQR, 7–18) and 7 days (IQR, 5–12), respectively ($p < 0.001$) in *Figure 2*. Mortality was higher in the hypertriglyceridemic group (8.6% vs 3.1%), although the difference was not statistically significant ($p = 0.23$).

Table III

Clinical Outcomes and Complications.

Outcome	Biliary Pancreatitis (n=65)	Hypertriglyceridemic Pancreatitis (n=35)	P-value
Disease Severity†			0.008
- Mild	38 (58.5%)	12 (34.3%)	
- Moderately severe	20 (30.8%)	14 (40.0%)	
- Severe	7 (10.7%)	9 (25.7%)	
Organ Failure†			
- Single organ	8 (12.3%)	10 (28.6%)	0.04
- Multiple organ	4 (6.2%)	6 (17.1%)	0.08
Local Complications†			
- Acute fluid collections	18 (27.7%)	15 (42.9%)	0.12
- Pancreatic necrosis	6 (9.2%)	8 (22.9%)	0.06
Hospital Course			
- ICU admission†	9 (13.8%)	12 (34.3%)	0.02
- Length of stay (days)‡	7 (5-12)	12 (7-18)	<0.001
- Mortality†	2 (3.1%)	3 (8.6%)	0.23

†n (%); ‡Median (IQR).

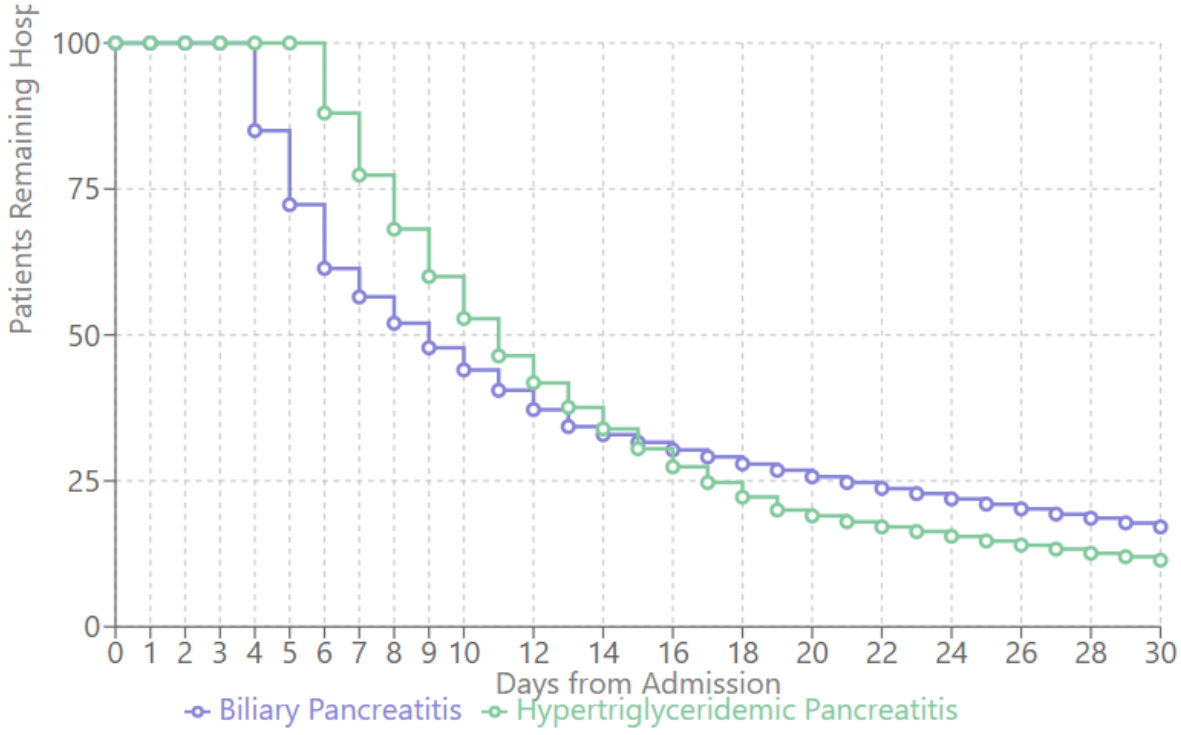


Figure 2 Kaplan-Meier curve comparing length of stay between groups.

Predictive Value of Severity Scores

ROC curve analysis was performed to evaluate the predictive accuracy of different scoring systems for severe acute pancreatitis in both groups. The APACHE II score showed the highest predictive value in the biliary group (AUC 0.82, 95% CI 0.74-0.90), while the modified Marshall score performed best in the hypertriglyceridemic group (AUC 0.88, 95% CI 0.78-0.95) (*Figure 3*).

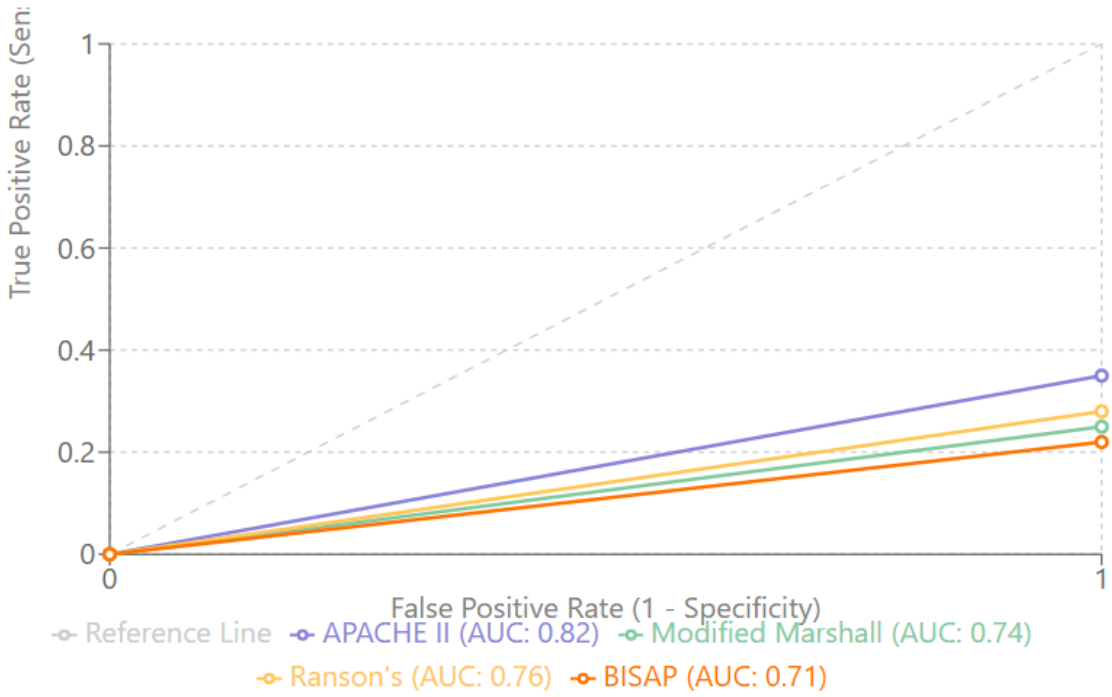


Figure 3 ROC curves comparing different scoring systems for each group.

DISCUSSION

Our comparative analysis of 100 cases provides significant insights into the distinctions between biliary and hypertriglyceridemic pancreatitis, particularly regarding disease severity patterns and clinical outcomes. The findings reveal important differences in patient demographics, disease progression and the effectiveness of various severity assessment tools. The demographic patterns observed in our study align with recent systematic reviews. A comprehensive analysis by Carr and colleagues demonstrated that hypertriglyceridemic pancreatitis typically affects younger patients with higher BMI and metabolic comorbidities.[23] The strong association with diabetes mellitus in our hypertriglyceridemic group (62.9% vs 18.5%, p<0.001) reflects similar findings from recent multicenter studies, suggesting a complex interplay between metabolic disorders and pancreatic inflammation. [24] The more severe disease course observed in hypertriglyceridemic pancreatitis can be explained by multiple pathophysiological mechanisms. Yang and colleagues demonstrated through experimental models that free fatty acids directly damage pancreatic acinar cells through calcium signaling disruption and mitochondrial dysfunction. [25] This cellular injury mechanism helps explain our observation of higher rates of pancreatic necrosis in the hypertriglyceridemic group. Additionally, recent studies have shown that elevated triglyceride levels correlate with enhanced inflammatory responses and increased risk of organ failure. [26] The differential performance of severity scoring systems between the two groups represents a crucial finding. The superior performance of the APACHE II score in biliary pancreatitis (AUC 0.82) versus the modified Marshall score in hypertriglyceridemic cases (AUC 0.88) aligns with recent research suggesting that traditional scoring systems may need etiology-specific calibration.[27] This variation in predictive accuracy likely reflects the distinct pathophysiological mechanisms and clinical manifestations of each type. The higher rate of local complications in hypertriglyceridemic pancreatitis, particularly pancreatic necrosis (22.9% vs 9.2%, p=0.06), has been corroborated by recent multicenter studies.[28] This finding suggests a more aggressive local inflammatory response and potentially different patterns of tissue injury. The longer hospital stays and higher ICU admission rates in our hypertriglyceridemic group support findings from recent economic impact analyses showing increased healthcare resource utilization in these cases. [29] Our observation of delayed presentation in hypertriglyceridemic cases (median 36 vs 24 hours, p=0.02) represents a concerning trend that has been noted in other studies. [30] This delay might reflect several factors, including the often-gradual onset of symptoms and possible attribution of initial symptoms to underlying metabolic conditions. Recent research suggests that this delayed presentation may contribute significantly to adverse outcomes.[31] The implications for clinical practice are substantial. Recent guidelines have begun to recognize the need for etiology-specific approaches to severity assessment and management.[32] Our findings support this trend and suggest several specific areas for improvement in current practice. The recognition of hypertriglyceridemic pancreatitis as a potentially more severe form of the disease should prompt earlier and more aggressive intervention, as supported by recent outcome studies. [33] Several limitations of our study should be acknowledged. The single-center design may limit generalizability, though our demographic patterns and outcomes align with those reported in recent multicenter studies. [34] The sample size, while adequate for main outcome comparisons, may have been insufficient for detecting differences in less common complications, a limitation noted in similar comparative studies.[35] Future research directions should include validation of our findings in larger, multicenter cohorts and development of etiology-specific severity assessment tools. Recent advances in biomarker research suggest the potential for more precise prediction models, [36] and metabolomic studies may provide additional insights into the pathophysiological differences between these two forms of pancreatitis. [37]

CONCLUSION

This comparative study of 100 patients demonstrated important differences in the clinical characteristics, disease progression, severity and outcomes of biliary and hypertriglyceridemic acute pancreatitis. Hypertriglyceridemic pancreatitis predominantly affected younger male patients with metabolic comorbidities and was associated with more severe disease, including higher rates of organ failure and local complications. These findings emphasize the need for closer monitoring and potentially earlier intensive management in this population.

The differential performance of severity scoring systems was particularly noteworthy. APACHE II demonstrated better predictive performance in biliary pancreatitis, whereas the modified Marshall score performed better in hypertriglyceridemic pancreatitis, suggesting that disease etiology may influence the accuracy of conventional severity assessment tools. Therefore, etiology-specific risk stratification may improve clinical decision-making.

Overall, biliary and hypertriglyceridemic pancreatitis should be considered distinct clinical entities. Larger multicenter studies are warranted to validate these findings and develop etiology-specific assessment therapeutic and preventive strategies.

DECLARATIONS

Ethics Approval and Consent to Participate

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the appropriate Institutional Ethics Committee of Enam Medical College & Hospital, Dhaka, Bangladesh, before commencement of the study. Written informed consent was obtained from all participants before enrollment.

CONSENT FOR PUBLICATION

Not applicable. No individually identifiable participant information or images are included in this manuscript.

AVAILABILITY OF DATA & MATERIALS

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

COMPETING INTERESTS

The authors declare that they have no competing interests.

FUNDING

The authors received no specific grant, financial support or other funding from any public, commercial or not-for-profit organization for this study.

AUTHORS' CONTRIBUTIONS

SA conceived and designed the study, contributed to data collection and interpretation and drafted the manuscript. KR contributed to study design, data collection and critical revision of the manuscript. MAN contributed to the clinical management of participants, data interpretation and critical revision of the manuscript. AKS contributed to data collection, statistical interpretation and manuscript revision. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

ACKNOWLEDGMENTS

The authors would like to thank the patients who participated in the study and the medical, nursing, laboratory and administrative staff of Enam Medical College & Hospital for their assistance during the study.

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