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# THE RELATIONSHIP BETWEEN DYSLIPIDEMIA AND PANCREATIC ENZYME LEVELS IN PATIENTS WITH ACUTE PANCREATITIS TREATED AT THAI BINH GENERAL HOSPITAL

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## ABSTRACT

**Background:** Dyslipidemia, particularly hypertriglyceridemia, has become an increasingly important cause of acute pancreatitis and may influence the interpretation of pancreatic enzyme measurements in clinical practice. In patients with marked lipid abnormalities, serum amylase and lipase levels do not always accurately reflect the extent of pancreatic injury, which may complicate diagnosis and early assessment. This study aimed to analyse the relationship between dyslipidemia and pancreatic enzyme concentrations in patients with acute pancreatitis treated at Thai Binh General Hospital.

**Materials and Methods:** A hospital-based retrospective cross-sectional analytical study was conducted using medical records and routine laboratory data of adult patients diagnosed with acute pancreatitis. Demographic, clinical, and biochemical variables were collected, including serum triglyceride, total cholesterol, amylase, and lipase levels. Pancreatic enzyme concentrations were compared across triglyceride categories. Correlation analyses were performed using Pearson's and Spearman's coefficients, including subgroup analyses by age, sex, body mass index, and etiology. Multivariable linear regression was used to identify independent factors associated with pancreatic enzyme levels.

**Results:** Serum amylase declined significantly with increasing triglyceride (TG) levels, decreasing from 435.6 in the normal TG group to 203.2, 158.3, 89.4, and 124.1 across the mild, moderate, high, and very high TG groups, respectively ($p < 0.001$). Serum lipase also differed across TG categories, although the pattern was less consistent ($p = 0.044$). In overall analyses, TG and total cholesterol were inversely correlated with amylase, whereas no significant correlations were observed with lipase. The inverse TG–amylase association remained evident in several subgroups, particularly among patients aged 40–59 years, both sexes, those with BMI 18.5–24.9 kg/m$^2$, and in alcohol-, gallstone-, and hypertriglyceridemia-related acute pancreatitis. However, after multivariable adjustment, TG and total cholesterol were no longer independently associated with amylase or lipase.

**Conclusion:** Dyslipidemia, especially elevated triglyceride levels, was associated with lower serum amylase concentrations in acute pancreatitis, suggesting that hyperlipidemia may affect the biochemical presentation of the disease. These findings support a cautious interpretation of pancreatic enzyme levels, particularly amylase, in patients with marked lipid abnormalities.

***Keywords:*** Acute pancreatitis; dyslipidemia; hypertriglyceridemia; amylase; lipase; triglycerides.

## 1. INTRODUCTION

Acute pancreatitis (AP) is one of the most common gastrointestinal emergencies, with clinical manifestations ranging from mild self-limited disease to severe forms associated with organ failure and death. Early diagnosis, etiological identification, and severity assessment are essential for appropriate management and improved outcomes. In Vietnam, in addition to the traditional causes such as gallstones and alcohol use, dyslipidemia, particularly hypertriglyceridemia, has increasingly been recognized as an important cause of AP [1,2].

In current clinical practice, serum amylase and lipase remain important biochemical markers for the diagnosis of AP. However, in hypertriglyceridemia-associated acute pancreatitis, pancreatic enzyme levels do not always rise proportionally to the degree of pancreatic injury. Recent international evidence has shown that a considerable proportion of patients with hypertriglyceridemic acute pancreatitis may present with normal or only mildly

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elevated serum amylase levels, which may lead to underdiagnosis or delayed diagnosis [3,4]. This phenomenon is mainly explained by the presence of triglyceride-rich lipoprotein particles in lipemic serum, which can interfere with laboratory assays and produce falsely low enzyme values [3,4].

Dyslipidemia is not only related to the etiology of AP but may also be associated with disease severity and clinical outcomes. Previous studies have reported positive associations between triglyceride and LDL-C concentrations and severity scores, such as the Ranson and APACHE II scores [5]. In addition, recent reviews have emphasised the pathogenic role of triglyceride-rich lipoproteins, the importance of risk stratification, and the need for recurrence prevention in patients with hypertriglyceridemia-associated AP [3]. Dyslipidemia-related pancreatitis has also been considered an understudied condition, especially in patients with coexisting metabolic risk factors such as diabetes mellitus and alcohol use [6].

In Vietnam, several recent studies have provided initial evidence on the clinical and laboratory characteristics of AP associated with lipid abnormalities. Vo Duy Thong et al. reported that very severe hypertriglyceridemia was associated with more severe disease and worse clinical outcomes [1]. Tran Ngoc Tuan et al. documented a considerable proportion of lipid disorders among patients with AP treated at provincial hospitals [2]. Vo Thi Doan Thuc and Pham Thi Ngoc Thao demonstrated significant changes in triglyceride concentrations and other biochemical parameters after plasma exchange in severe hypertriglyceridemia-induced AP [7]. Notably, a study conducted in Thai Binh by Nguyen Thi Minh Thai showed an association between amylase, lipase, and complications in patients with AP [8].

At Thai Binh General Hospital, the number of AP patients with concomitant dyslipidemia appears to be increasing, but evidence on the quantitative relationship between lipid abnormalities and pancreatic enzyme levels remains limited. Clarifying this relationship may help improve laboratory interpretation, reduce the risk of missed diagnosis in patients with marked hyperlipidemia, and support early assessment of disease severity. Therefore, this study was conducted to analyse the relationship between dyslipidemia and pancreatic enzyme concentrations in patients with acute pancreatitis treated at Thai Binh General Hospital.

## 2. MATERIALS AND METHODS

### 2.1. Study design and setting

This was a hospital-based retrospective cross-sectional analytical study conducted at Thai Binh General Hospital, Thai Binh, Vietnam. The study reviewed medical records and routine laboratory data of patients admitted with acute pancreatitis during the study period from [month/year] to [month/year]. The main objective was to evaluate the relationship between dyslipidemia, particularly serum triglyceride and total cholesterol levels, and pancreatic enzyme concentrations in patients with acute pancreatitis.

### 2.2. Study subjects

The study population included adult patients aged 18 years or older who were diagnosed with acute pancreatitis and treated at Thai Binh General Hospital during the study period. Acute pancreatitis was diagnosed when at least two of the following criteria were present: characteristic abdominal pain, serum amylase and/or lipase levels more than three times the upper limit of normal, and imaging findings consistent with acute pancreatitis [3].

Patients were excluded if their medical records were incomplete; if serum triglyceride, total cholesterol, amylase, or lipase results were unavailable; if they had chronic pancreatitis, pancreatic malignancy, or a history of pancreatic surgery; or if they had been transferred from another hospital after receiving substantial prior treatment. In patients with more than one admission during the study period, only the first eligible episode was included in the analysis.

For subgroup analysis, patients were further classified according to age group (<40, 40–59, and ≥60 years), sex, BMI category (<18.5, 18.5–24.9, and ≥25 kg/m$^2$), and etiology of acute pancreatitis (alcohol-related, gallstone-related, hypertriglyceridemia-related, or other causes).

### 2.3. Sample size and sampling method

No formal sample size calculation was performed because this study used available hospital-based secondary data. All consecutive patients who met the inclusion criteria and none of the exclusion criteria during the study period were included. Therefore, a consecutive sampling method was applied. The final analytic sample consisted of [n] patients.

### 2.4. Measurements and data collection

Data were extracted retrospectively from hospital medical records using a standardised data collection form. The collected variables included demographic characteristics (age and sex), anthropometric data (height, weight, and BMI), clinical characteristics, and laboratory findings.

Clinical variables included alcohol use, gallstone-related disease, dyslipidemia/hyperlipidemia status, and etiology of acute pancreatitis. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m$^2$) and categorised into three groups: <18.5, 18.5–24.9, and ≥25 kg/m$^2$.

Laboratory variables included serum triglyceride (TG), total cholesterol (TC), amylase, and lipase levels. For analysis, serum triglyceride and total cholesterol were evaluated as continuous variables. In addition, triglyceride levels were categorised into predefined groups from the study dataset to compare pancreatic enzyme levels across TG strata.

The primary outcomes were serum amylase and serum lipase concentrations. The main exposure variables were serum triglyceride and total cholesterol levels. Other covariates included age, sex, BMI, alcohol-related etiology, gallstone-related etiology, and documented dyslipidemia/hyperlipidemia.

### 2.5. Statistical analysis

Data were entered and analysed using [SPSS/Stata/R] version [x.x]. Categorical variables were presented as frequencies and percentages. Continuous variables were summarised as mean ± standard deviation if normally distributed, or median and interquartile range if non-normally distributed.

Comparisons of pancreatic enzyme levels across triglyceride categories were performed using one-way analysis of variance (ANOVA) for normally distributed variables or the Kruskal–Wallis test for non-normally distributed variables, as appropriate.

The relationships between lipid parameters and pancreatic enzyme levels were assessed using Pearson's correlation coefficient for parametric data and Spearman's rank correlation coefficient for nonparametric data. Stratified correlation analyses were also conducted according to age group, sex, BMI group, and etiology of acute pancreatitis.

To identify independent factors associated with pancreatic enzyme levels, multivariable linear regression models were constructed with serum amylase and serum lipase as dependent variables. Independent variables included TG, TC, age, sex, BMI, alcohol-related etiology, gallstone-related etiology, and dyslipidemia/hyperlipidemia status. Regression coefficients (β), 95% confidence intervals (95% CI), and p-values were reported. A two-sided p-value of less than 0.05 was considered statistically significant.

### 2.6. Ethical consideration

The study protocol was reviewed and approved by the Ethics Committee of Thai Binh University of Medicine and Pharmacy under Approval No. 10/GCN-HDDD-YDTB, dated February 10, 2026. As this was a retrospective study based on existing medical records and routine laboratory data, the Ethics Committee waived the requirement for written informed consent. All patient information was kept strictly confidential throughout the study. Personal identifiers were removed before data analysis, and the collected data were used solely for research purposes.

## 3. RESULTS

**Table 1. Pancreatic enzyme levels according to triglyceride categories**

| Triglyceride group | Amylase | p-value* | Lipase | p-value* |
|---|---|---|---|---|
| | Mean±SD | | Mean±SD | |
| Normal | 435.6 ± 1024.5 | < 0.001 | 802.9 ± 2119.5 | 0.04 |
| Mild elevation | 203.2 ± 1695.6 | | 444.0 ± 3061.5 | |
| Moderate elevation | 158.3 ± 647.0 | | 520.5 ± 1494.8 | |
| High elevation | 89.4 ± 108.7 | | 225.7 ± 378.9 | |
| Very high elevation | 124.1 ± 257.2 | | 405.3 ± 739.0 | |

*Mean ± SD: Mean ± standard deviation; *: Overall p-value across triglyceride groups. A p-value <0.05 was considered statistically significant.*

Pancreatic enzyme levels differed significantly across triglyceride categories. Amylase decreased progressively with increasing triglyceride levels, whereas lipase also varied across groups but without a clear linear trend.

**Table 2. Pearson correlations between lipid parameters and pancreatic enzyme levels**

| Lipid parameter | Amylase, $r_s$ | p-value | Lipase, $r_s$ | p-value |
|---|---|---|---|---|
| TG (mmol/L) | -0.19 | 0.02 | -0.121 | 0.15 |
| TC (mmol/L) | -0.21 | 0.01 | -0.151 | 0.07 |

*TG: Triglyceride; TC: Total cholesterol; r: Pearson correlation coefficient; $r_s$: Spearman rank correlation coefficient. A p-value <0.05 was considered statistically significant*

TG and TC showed weak but significant inverse correlations with amylase, whereas no significant Pearson correlation was observed with lipase.

**Table 3. Spearman correlations between lipid parameters and pancreatic enzyme levels**

| Lipid parameter | Amylase, $r_s$ | p-value | Lipase, $r_s$ | p-value |
|---|---|---|---|---|
| TG (mmol/L) | -0.38 | <0.001 | -0.16 | 0.06 |
| TC (mmol/L) | -0.23 | 0.006 | -0.07 | 0.37 |

*TG: Triglyceride; TC: Total cholesterol; r: Pearson correlation coefficient; $r_s$: Spearman rank correlation coefficient. A p-value <0.05 was considered statistically significant.*

Spearman analysis confirmed a stronger inverse association between triglycerides and amylase, while correlations with lipase remained non-significant.

**Table 4. Correlations between triglycerides and pancreatic enzymes by age group**

| Age group (years) | TG – Amylase, r | p-value | TG – Lipase, r | p-value |
|---|---|---|---|---|
| <40 | -0.32 | 0.12 | 0.02 | 0.91 |
| 40–59 | -0.37 | <0.001 | -0.13 | 0.22 |
| ≥60 | -0.346 | 0.091 | -0.173 | 0.407 |

*r: Pearson correlation coefficient; A p-value <0.05 was considered statistically significant.*

A significant inverse correlation between triglycerides and amylase was observed only in patients aged 40–59 years. No significant age-stratified correlation was found for lipase.

**Table 5. Correlations between triglycerides and pancreatic enzymes by sex**

| Sex | TG – Amylase, r | p-value | TG – Lipase, r | p-value |
|---|---|---|---|---|
| Male | -0.37 | < 0.001 | -0.12 | 0.20 |
| Female | -0.52 | 0.013 | -0.34 | 0.13 |

*r: Pearson correlation coefficient; A p-value <0.05 was considered statistically significant.*

The inverse correlation between triglycerides and amylase was significant in both sexes and appeared stronger in women. No significant correlation was found between triglycerides and lipase.

**Table 6. Correlations between triglycerides and pancreatic enzymes by BMI group**

| BMI group (kg/m$^2$) | TG – Amylase, r | p-value | TG – Lipase, r | p-value |
|---|---|---|---|---|
| <18.5 | -0.21 | 0.41 | -0.01 | 0.98 |
| 18.5–24.9 | -0.36 | < 0.001 | -0.12 | 0.25 |
| ≥25.0 | -0.29 | 0.15 | -0.15 | 0.46 |

*r: Pearson correlation coefficient; A p-value <0.05 was considered statistically significant.*

The inverse TG–amylase correlation was significant only in the normal-BMI group. No BMI-stratified association was observed for lipase.

**Table 7. Correlations between triglycerides and pancreatic enzymes by etiology**

| Etiology | TG – Amylase, r | p-value | TG – Lipase, r | p-value |
|---|---|---|---|---|
| Other causes | -0.43 | 0.16 | -0.28 | 0.38 |
| Alcohol-related | -0.38 | 0.001 | -0.19 | 0.11 |
| Gallstone-related | -0.73 | 0.002 | -0.51 | 0.05 |
| Hypertriglyceridemia-related | -0.43 | 0.005 | -0.16 | 0.32 |

*r: Pearson correlation coefficient; A p-value <0.05 was considered statistically significant.*

Triglycerides were inversely correlated with amylase in alcohol-, gallstone-, and hypertriglyceridemia-related acute pancreatitis, with the strongest association in the gallstone group. No significant association with lipase was identified across etiological subgroups.

**Table 8. Multivariable linear regression analysis for factors associated with pancreatic enzyme levels**

| Independent variable | β (95% CI) for amylase | p-value | β (95% CI) for lipase | p-value |
|---|---|---|---|---|
| TG | -8.97 (-21.84 - 3.90) | 0.170 | -16.39 (-53.74 - 20.96) | 0.38 |
| TC | -6.82 (-49.54 - 35.89) | 0.752 | -19.82 (-143.79 - 104.15) | 0.75 |
| Age | 3.21 (-5.85 - 12.26) | 0.485 | -4.16 (-30.43 - 22.12) | 0.76 |
| Sex | -497.91 (-782.46 - -213.36) | 0.001 | -1251.62 (-2077.54 - -425.70) | 0.003 |
| BMI | -40.18 (-79.43 - -0.93) | 0.045 | -149.39 (-263.32 - -35.46) | 0.01 |
| Alcohol-related etiology | -612.37 (-983.10 - -241.63) | 0.001 | -2096.44 (-3172.50 - -1020.38) | <0.001 |
| Gallstone-related etiology | -187.15 (-656.21 - 281.92) | 0.431 | -733.29 (-2094.77 - 628.19) | 0.29 |
| Dyslipidemia | -104.80 (-501.38 - 291.78) | 0.602 | -633.39 (-1784.48 - 517.71) | 0.28 |

*β: Regression coefficient; CI: Confidence interval; TG: Triglyceride; TC: Total cholesterol; BMI: body mass index. A p-value <0.05 was considered statistically significant.*

In multivariable regression, TG and TC were not independently associated with either amylase or lipase. Sex, BMI, and alcohol-related etiology remained independently associated with both pancreatic enzymes.

## 4. DISCUSSION

The present study demonstrated that pancreatic enzyme levels, particularly serum amylase, were inversely associated with lipid abnormalities in patients with acute pancreatitis. Amylase levels decreased significantly across increasing triglyceride categories, and both triglyceride and total cholesterol showed significant inverse correlations with amylase in the overall analysis. In contrast, the associations between lipid parameters and lipase were weaker and did not reach statistical significance in most analyses. After multivariable adjustment, triglyceride and total cholesterol were no longer independently associated with either amylase or

lipase, whereas sex, BMI, and alcohol-related etiology remained significant correlates. These findings suggest that dyslipidemia may influence the interpretation of pancreatic enzyme results, especially amylase, but that this relationship is also shaped by other clinical and metabolic factors.

Our findings are clinically relevant because the diagnosis of acute pancreatitis is still based on the revised Atlanta framework, which requires at least two of three criteria: typical abdominal pain, pancreatic enzyme elevation greater than three times the upper limit of normal, or compatible imaging findings [3]. Therefore, in patients with marked dyslipidemia, particularly hypertriglyceridemia, reliance on serum amylase alone may lead to under-recognition of the disease. This interpretation is consistent with recent evidence showing that hypertriglyceridemic acute pancreatitis may present with normal or only mildly elevated serum amylase, partly because lipemic serum interferes with laboratory measurement and may yield falsely low results [4,5]. These mechanisms also help explain why, in our study, amylase showed a clearer inverse relationship with triglycerides than lipase did.

The present results are also broadly consistent with previous Vietnamese and international studies. The local study from Thai Binh reported that lipase was more closely associated with disease severity and complications than amylase, suggesting that lipase may better reflect clinically meaningful pancreatic injury in routine practice [6]. In our dataset, lipase did not show a strong inverse correlation with triglycerides, which may indicate that it is less affected by lipid-related analytical interference than amylase. Similarly, studies in Vietnam have shown that severe hypertriglyceridemia is associated with more severe disease and poorer outcomes in acute pancreatitis [1]. Taken together, these observations suggest that enzyme values in hyperlipidemic patients should be interpreted alongside the lipid profile and overall clinical context, rather than as isolated diagnostic markers.

Another notable finding of our study was that the inverse triglyceride–amylase relationship remained significant in several subgroup analyses, including patients aged 40–59 years, both sexes, the BMI 18.5–24.9 kg/m$^2$ group, and alcohol-, gallstone-, and hypertriglyceridemia-related acute pancreatitis. The strongest correlation was observed in the gallstone-related subgroup. However, these subgroup findings should be interpreted with caution, as smaller sample sizes and between-group heterogeneity may influence stratified analyses. More importantly, the disappearance of the triglyceride and cholesterol effects in the multivariable models suggests that the crude associations were at least partly confounded by other determinants, particularly alcohol-related etiology, BMI, and sex. This pattern is biologically plausible because dyslipidemia-associated pancreatitis is increasingly recognised as a multifactorial condition influenced by metabolic stress, obesity, alcohol use, diabetes, and other secondary or genetic contributors [5,7].

From a practical perspective, our findings support a more cautious diagnostic approach for patients with acute pancreatitis and marked hyperlipidemia. In patients with typical abdominal pain and high clinical suspicion, a relatively low amylase concentration should not be used to exclude acute pancreatitis, especially when triglyceride levels are elevated. In such cases, lipase measurement, repeat testing after appropriate sample handling, or imaging evaluation may be more informative [3-5]. This is particularly important in provincial and general hospital settings, where acute pancreatitis is common and where dyslipidemia appears to be an increasingly relevant etiological and prognostic factor[1,2].

This study has several limitations. First, it was a single-centre study, which may limit generalizability. Second, the retrospective use of routine laboratory data did not allow direct evaluation of pre-analytical factors, such as dilution protocols for lipemic serum, which may affect enzyme measurement. Third, although significant correlations were observed in several subgroups, these analyses were exploratory and may have been underpowered. Finally, because triglyceride and cholesterol were not independently associated with enzyme concentrations after multivariable adjustment, the present findings should be interpreted primarily as evidence of clinically relevant association rather than causation.

Despite these limitations, the study provides useful local evidence from Thai Binh and adds to the growing literature indicating that dyslipidemia, especially hypertriglyceridemia, can complicate the biochemical interpretation of acute pancreatitis. Future multicenter studies with standardised laboratory processing and integrated severity outcomes are needed to clarify whether lipid-related enzyme suppression has direct prognostic implications and to determine the most reliable diagnostic strategy in this patient population.

## 5. CONCLUSION

This study found that dyslipidemia, particularly elevated triglyceride levels, was inversely associated with serum amylase in patients with acute pancreatitis, whereas the relationship with lipase was weaker and less consistent. Amylase levels tended to decline across increasing triglyceride categories, suggesting that hyperlipidemia may influence the biochemical presentation of acute pancreatitis. However, after adjustment for potential confounding factors, triglycerides and total cholesterol were not independently associated with pancreatic enzyme levels. These findings suggest that acute pancreatitis should not be excluded solely on the basis of a relatively low or non-elevated amylase level in patients with marked hypertriglyceridemia. A more comprehensive assessment, including serum lipase, lipid profile, clinical manifestations, and imaging findings, may help improve diagnostic accuracy in clinical practice. Further large-scale, multicenter prospective studies are warranted to clarify the diagnostic and prognostic value of pancreatic enzymes in acute pancreatitis associated with dyslipidemia.

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