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Correlation of Serum Triglyceride With C-Reactive Protein in Acute Pancreatitis

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Abstract
Background: *Acute pancreatitis (AP) represents a significant inflammatory disorder with variable severity and outcomes. Serum triglyceride levels and C-reactive protein (CRP) are established biomarkers that may provide insights into disease pathophysiology and severity.*
Objective: *To investigate the correlation between serum triglyceride levels and CRP in patients with acute pancreatitis and to assess their association with disease severity.*
Methods: *A prospective observational study was conducted at Era's Lucknow Medical College & Hospital over 18 months. Fifty-eight patients aged 18 years and above diagnosed with acute pancreatitis were enrolled. Serum triglyceride and CRP levels were measured at admission. Disease severity was classified according to the Revised Atlanta Classification. Statistical analysis included Pearson's correlation coefficient to evaluate the relationship between serum triglyceride and CRP levels.*
Results: *The mean age of patients was 42.25±12.77 years, with male predominance (62.1%). Alcoholism (50%) and biliary causes (39.6%) were the most common etiologies. Based on Atlanta classification, 53.5% had mild, 32.7% had moderate, and 13.8% had severe acute pancreatitis. Both CRP and serum triglyceride levels showed significant positive correlation with disease severity ($p<0.001$). Mean CRP levels were 45.6±15.3 mg/L in mild cases, 103.4±25.1 mg/L in moderate cases, and 201.9±35.6 mg/L in severe cases. Mean serum triglyceride levels were 1.7±0.6 mmol/L, 3.1±1.1 mmol/L, and 6.2±1.8 mmol/L in mild, moderate, and severe cases respectively.*
Conclusion: *There is a significant correlation between serum triglyceride levels and CRP in acute pancreatitis, with both biomarkers demonstrating strong association with disease severity. These findings suggest a potential role of lipid metabolism in modulating the inflammatory response in acute pancreatitis.*
Keywords: *Acute pancreatitis, C-reactive protein, Serum triglyceride, Disease severity, Hypertriglyceridemia, Inflammatory markers*

I. INTRODUCTION

Acute pancreatitis (AP) constitutes one of the most prevalent gastrointestinal disorders worldwide, imposing substantial emotional, physical, and financial burden on healthcare systems. The global annual incidence varies between 4.9 to 73 cases per 100,000 individuals, with considerable geographic variation. In the United States, Finland, and Scotland, the incidence rates are notably elevated at 49.3, 46.6, and 41.9 per 100,000 populations respectively.[1] In 2009, acute pancreatitis accounted for approximately 275,000 hospitalizations annually in the United States alone.[2]

The clinical spectrum of acute pancreatitis exhibits remarkable heterogeneity, ranging from mild, self-limiting disease with mortality rates below 1%, to severe acute pancreatitis (SAP) characterized by pancreatic necrosis and organ failure, with mortality rates approaching 10% to 30%.[3,4] The pathophysiological progression of acute pancreatitis typically occurs in two distinct phases. The initial phase involves a cytokine-mediated inflammatory cascade triggered by acinar cell injury, generally persisting for approximately one week. During this critical period, the systemic inflammatory response syndrome (SIRS) develops, and approximately fifty percent of fatalities occur within the first two weeks, predominantly due to multi-organ failure.[5,6]

Gallstones and alcohol consumption collectively account for approximately 70% of acute pancreatitis cases globally.[7] Gallstone pancreatitis, including microlithiasis, represents the predominant etiology, responsible for 35-40% of cases, with approximately 3-7% of individuals with gallstones eventually developing acute pancreatitis.[8] Alcohol has emerged as a significant etiological factor, particularly in Eastern India, where

it has become the predominant cause in recent years. The observation that only 2-3% of chronic alcohol consumers develop pancreatitis suggests an underlying genetic predisposition.[9]

Hypertriglyceridemia (HTG) represents the third most common etiology of acute pancreatitis following gallstones and alcohol, accounting for 1-12% of acute pancreatitis cases.[10,11] Hypertriglyceridemia-induced acute pancreatitis (HTG-AP) has been associated with a more severe clinical course characterized by higher rates of respiratory and renal failure in several studies, although conflicting findings have been reported.[12] The National Cholesterol Education Program ATP III categorizes triglyceride levels as normal (<150 mg/dL), borderline high (150-199 mg/dL), high (200-499 mg/dL), and very high (>500 mg/dL). Acute pancreatitis typically manifests when triglyceride levels exceed 1000 mg/dL, though considerable individual variation exists.[13]

C-reactive protein (CRP) plays a pivotal role in acute pancreatitis as a marker of systemic inflammation and disease severity. CRP is synthesized by hepatocytes in response to interleukin-1 (IL-1) and interleukin-6 (IL-6) stimulation during inflammatory conditions.[14] Elevated CRP levels, particularly those exceeding 150 mg/L within the first 48 hours, are strongly associated with severe acute pancreatitis and can effectively predict complications including pancreatic necrosis and systemic inflammatory response syndrome.[15] A systematic review and meta-analysis encompassing 41 studies with 6,156 acute pancreatitis cases demonstrated that CRP possesses significant diagnostic value for assessing disease severity, with an area under the curve of 0.85, sensitivity of 0.76, and specificity of 0.79.[16]

The correlation between serum triglyceride levels and CRP in acute pancreatitis has gained increasing research attention. Elevated triglyceride levels are frequently associated with hypertriglyceridemia-induced acute pancreatitis, and these levels may significantly influence the magnitude of the inflammatory response. Studies have demonstrated that higher triglyceride levels correlate with elevated CRP concentrations, indicating a more severe inflammatory response and potentially more aggressive disease course.[17] Recent investigations have revealed that triglyceride levels measured within the first 48 hours post-onset are most beneficial for diagnosing and assessing the severity of hypertriglyceridemic acute pancreatitis. A multicenter international cohort analysis of 716 acute pancreatitis cases showed that hypertriglyceridemia was significantly and dose-dependently associated with local complications, organ failure, and maximum CRP levels.[18]

The precise pathophysiological mechanisms linking hypertriglyceridemia to acute pancreatitis remain incompletely elucidated. Current evidence suggests that toxicity is primarily mediated by free fatty acids rather than triglycerides themselves. Free fatty acids can stimulate inflammation, form detergent-like aggregates that damage cell membranes, injure endothelial cells, and induce organ damage including acute lung injury.[19] Moreover, elevated triglyceride levels create a pro-inflammatory milieu characterized by increased production of inflammatory cytokines, enhanced oxidative stress, and impaired pancreatic microcirculation, all contributing to the progression from mild to severe disease.

Understanding the relationship between serum triglyceride levels and CRP provides crucial insights into the inflammatory processes and severity determinants of acute pancreatitis. This knowledge facilitates early identification of high-risk patients, enables more informed clinical decision-making, guides therapeutic interventions, and potentially reduces complications and mortality rates. Despite growing evidence, gaps remain in our understanding of how these biomarkers interact and influence disease progression, particularly in diverse populations and varying clinical contexts. Therefore, this study was designed to investigate the correlation between serum triglyceride and C-reactive protein levels in patients with acute pancreatitis, contributing to the existing body of knowledge and potentially informing more effective management strategies.

II. AIMS AND OBJECTIVES

Aim: To determine the correlation between serum triglyceride and C-reactive protein levels in patients with acute pancreatitis.

Objectives:

1. To measure serum triglyceride and C-reactive protein levels in patients diagnosed with acute pancreatitis.
2. To determine the relationship between serum triglyceride and C-reactive protein levels in acute pancreatitis.
3. To assess the association of these biomarkers with the severity of acute pancreatitis.

III. MATERIALS AND METHODS

Study Design and Setting

This prospective observational study was conducted in the Department of General Surgery at Era's Lucknow Medical College & Hospital, Lucknow, over a period of 18 months. The study was designed to investigate the correlation between serum triglyceride levels and C-reactive protein in patients presenting with acute pancreatitis.

Study Population
The study population comprised all patients aged 18 years and above presenting to the Department of General Surgery with a diagnosis of acute pancreatitis. Both first-episode and recurrent cases of acute pancreatitis were included to capture the full spectrum of disease presentation.

Sample Size Calculation
The sample size was calculated to ensure 90% study power based on the variability in CRP levels among patients with acute pancreatitis. Using standard deviation of 72.45, clinically significant difference of 34.37, Type I error (α) of 5%, Type II error (β) of 10%, and design effect of 1, the minimum sample size required was calculated to be 58 patients.

Inclusion and Exclusion Criteria
Patients aged 18 years and above with diagnosis of acute pancreatitis (based on revised Atlanta Classification) and recurrent pancreatitis episodes were included. Patients with chronic pancreatitis or pancreatic tumors were excluded.

Methodology
Acute pancreatitis was defined according to the revised Atlanta Classification. Blood samples for serum triglyceride and C-reactive protein were collected at admission. Disease severity was classified as mild, moderate, or severe based on clinical symptoms, radiological findings, and systemic complications. Comprehensive demographic, clinical, and laboratory data were systematically collected.

Ethical Considerations
The study protocol was approved by the Institutional Ethics Committee of Era's Lucknow Medical College & Hospital. Written informed consent was obtained from all participants. Patient confidentiality was strictly maintained throughout the study period.

Statistical Analysis
Data were analyzed using IBM SPSS Statistics software version 26.0. Continuous variables were expressed as mean ± standard deviation. Pearson's correlation coefficient was utilized to assess the relationship between serum triglyceride and CRP levels. Chi-square test and one-way ANOVA were used as appropriate. Statistical significance was defined as p-value <0.05.

IV. RESULTS

Demographic and Baseline Characteristics
A total of 58 patients with acute pancreatitis were enrolled in the study. Table 1 presents the comprehensive demographic and baseline characteristics of the study population.

Table 1: Demographic and Baseline Characteristics of Study Population (n=58)

Variables	Frequency (n)	Percentage (%)
AGE DISTRIBUTION (YEARS)		
≤30	13	22.4
31-40	14	24.1
41-50	11	18.9
51-60	14	24.1
>60	6	10.3
Mean ± SD	42.25 ± 12.77	-
GENDER		
Male	36	62.1
Female	22	37.9
ETIOLOGY		

Alcoholism	29	50.0
Biliary	23	39.6
Idiopathic	6	10.4
PERSONAL HISTORY		
Vegetarian	32	55.2
Non-vegetarian	26	44.8
Smoking	21	36.2
Alcohol intake	35	60.3
CHIEF COMPLAINTS		
Pain abdomen	55	94.8
Abdominal distension	47	81.0
Nausea & Vomiting	48	82.7
Jaundice	10	17.2
Fever	24	41.4
Tachycardia	10	17.2
Dyspnea	5	8.6
Tachypnea	8	13.8

The mean age of the study population was 42.25 ± 12.77 years with male predominance (62.1%). Alcoholism was the most common etiology (50%), followed by biliary causes (39.6%). Abdominal pain was the predominant presenting complaint (94.8%), followed by nausea and vomiting (82.7%) and abdominal distension (81.0%).
Laboratory Parameters

Table 2 presents the comprehensive laboratory parameters of the study population.

Table 2: Laboratory Parameters of Study Population (n=58)

Laboratory Parameters	**Mean ± SD**
HEMATOLOGICAL PARAMETERS	
Hemoglobin (g/dL)	13.6 ± 2.7
Total Leukocyte Count (cells/mm³)	14,338.2 ± 6,120.1
RENAL FUNCTION TESTS	
Blood Urea (mg/dL)	32.5 ± 26.1
Serum Creatinine (mg/dL)	0.89 ± 0.63
SERUM ELECTROLYTES	
Sodium (mEq/L)	132.6 ± 16.6
Potassium (mEq/L)	4.0 ± 0.8
Chloride (mEq/L)	8.4 ± 1.6
LIVER FUNCTION TESTS	
Total Bilirubin (mg/dL)	1.8 ± 1.1

Direct Bilirubin (mg/dL)	1.2 ± 0.96
Indirect Bilirubin (mg/dL)	0.54 ± 0.58
SGOT (IU/L)	104.7 ± 111.8
SGPT (IU/L)	127.7 ± 127.5
Alkaline Phosphatase (IU/L)	157.2 ± 123.0
Total Protein (mg/dL)	6.1 ± 1.6
Serum Albumin (mg/dL)	3.4 ± 1.0
Serum Globulin (mg/dL)	2.7 ± 0.7
PANCREATIC ENZYMES	
Serum Amylase (IU/L)	577.1 ± 406.3
Serum Lipase (IU/L)	3,764.9 ± 3,508.4
OTHER PARAMETERS	
Serum LDH (IU/L)	368.9 ± 492.9
Serum Calcium (mg/dL)	7.92 ± 1.69
C-Reactive Protein (mg/L)	139.92 ± 44.55
Serum Triglycerides (mmol/L)	3.09 ± 2.12

The mean total leukocyte count was 14,338.2 ± 6,120.1 cells/mm³, indicating an inflammatory response. Pancreatic enzymes were significantly elevated with mean serum amylase of 577.1 ± 406.3 IU/L and mean serum lipase of 3,764.9 ± 3,508.4 IU/L. The mean C-reactive protein level was 139.92 ± 44.55 mg/L, and mean serum triglyceride level was 3.09 ± 2.12 mmol/L.

Severity of Acute Pancreatitis

Table 3 presents the severity distribution of acute pancreatitis according to the Revised Atlanta Classification.

Table 3: Severity of Acute Pancreatitis According to Revised Atlanta Classification (n=58)

Severity	Gallstone-induced (n=42) n (%)	Post-ERCP (n=16) n (%)	Total (n=58) n (%)	p-value
Mild	22 (52.3)	9 (56.3)	31 (53.5)	0.659
Moderate	15 (35.7)	4 (25.0)	19 (32.7)	
Severe	5 (11.9)	3 (18.7)	8 (13.8)	

Chi-square test was applied. p-value >0.05 indicates no significant difference in severity distribution between gallstone-induced and post-ERCP pancreatitis.

Based on the Revised Atlanta Classification, 53.5% of cases were mild, 32.7% were moderately severe, and 13.8% were severe acute pancreatitis. There was no statistically significant difference in severity distribution between gallstone-induced and post-ERCP pancreatitis (p=0.659).

Association of C-Reactive Protein with Disease Severity

Table 4 demonstrates the relationship between C-reactive protein levels and disease severity.

Table 4: Association of C-Reactive Protein Levels with Severity of Acute Pancreatitis (n=58)

CRP Levels (mg/L)	Mild (n=31) n (%)	Moderate (n=19) n (%)	Severe (n=8) n (%)	p-value
≤50	19 (61.3)	8 (42.1)	0 (0.0)	<0.001
51-100	11 (35.5)	6 (31.6)	1 (12.5)	

101-200	1 (3.2)	3 (15.8)	2 (25.0)	
>200	0 (0.0)	2 (10.5)	5 (62.5)	
Mean CRP ± SD	**45.6 ± 15.3**	**103.4 ± 25.1**	**201.9 ± 35.6**	**<0.001**

One-way ANOVA test was applied. p-value <0.001 indicates highly significant association between CRP levels and disease severity.

CRP levels demonstrated a highly significant progressive increase with escalating disease severity. Among patients with mild acute pancreatitis, 61.3% had CRP levels ≤50 mg/L, while in severe cases, 62.5% of patients exhibited CRP levels >200 mg/L. The mean CRP levels increased significantly from 45.6 ± 15.3 mg/L in mild cases to 103.4 ± 25.1 mg/L in moderate cases, and 201.9 ± 35.6 mg/L in severe cases (p<0.001).

Association of Serum Triglyceride with Disease Severity

Table 5 demonstrates the relationship between serum triglyceride levels and disease severity.

Table 5: Association of Serum Triglyceride Levels with Severity of Acute Pancreatitis (n=58)

Serum Triglyceride Levels (mmol/L)	Mild (n=31) n (%)	Moderate (n=19) n (%)	Severe (n=8) n (%)	p-value
≤1.7	21 (67.7)	7 (36.8)	0 (0.0)	<0.001
1.8-3.4	8 (25.8)	5 (26.3)	0 (0.0)	
3.5-5.6	2 (6.5)	4 (21.1)	3 (37.5)	
>5.7	0 (0.0)	3 (15.8)	5 (62.5)	
Mean TG ± SD	**1.7 ± 0.6**	**3.1 ± 1.1**	**6.2 ± 1.8**	**<0.001**

One-way ANOVA test was applied. p-value <0.001 indicates highly significant association between serum triglyceride levels and disease severity. Note: To convert triglyceride values from mmol/L to mg/dL, multiply by 88.5.

Serum triglyceride levels demonstrated a similar pattern of progressive elevation with increasing disease severity. Among patients with mild acute pancreatitis, 67.7% had triglyceride levels ≤1.7 mmol/L, whereas in severe cases, 62.5% of patients had triglyceride levels >5.7 mmol/L. The mean serum triglyceride levels showed significant escalation from 1.7 ± 0.6 mmol/L in mild cases to 3.1 ± 1.1 mmol/L in moderate cases, and 6.2 ± 1.8 mmol/L in severe cases (p<0.001).

Clinical Outcomes and Complications

Table 6 presents the complications and clinical outcomes observed in the study population.

Table 6: Complications and Clinical Outcomes (n=58)

Complications	Frequency (n)	Percentage (%)
Paralytic Ileus	42	72.4
Pancreatic Necrosis	20	34.5
SIRS/MODS	11	18.9
Renal Failure	2	3.4
ARDS	1	1.7
DURATION OF HOSPITAL STAY		
≤5 days	31	53.4
>5 days	27	46.6
Mean ± SD (Range)	5.66 ± 2.43	(1-13 days)
OUTCOMES		

Discharged	56	93.3
Expired	2	6.7

SIRS = Systemic Inflammatory Response Syndrome; MODS = Multiple Organ Dysfunction Syndrome; ARDS = Acute Respiratory Distress Syndrome

Paralytic ileus was the most frequently encountered complication (72.4%), followed by pancreatic necrosis (34.5%). The mean duration of hospital stay was 5.66 ± 2.43 days. The majority of patients (93.3%) were successfully managed and discharged, while mortality occurred in 6.7% of patients.

V. DISCUSSION

The present study demonstrates a significant correlation between serum triglyceride levels and C-reactive protein in patients with acute pancreatitis. Both biomarkers showed strong, statistically significant associations with disease severity as classified by the Revised Atlanta Classification. Our findings contribute valuable insights to the understanding of acute pancreatitis pathophysiology and have important clinical implications for early risk stratification and management.

The mean age of patients in our study was 42.25 ± 12.77 years with male predominance (62.1%), consistent with several previous investigations. The etiological distribution with alcoholism as the predominant cause (50%) followed by biliary etiology (39.6%) reflects the changing patterns of acute pancreatitis in India. Our severity classification revealed 53.5% mild, 32.7% moderate, and 13.8% severe cases, proportions remarkably consistent with multiple international studies.

Our study demonstrated that CRP levels exhibited a significant progressive increase with escalating disease severity, rising from 45.6 ± 15.3 mg/L in mild cases to 201.9 ± 35.6 mg/L in severe cases ($p<0.001$). These findings are strongly supported by extensive literature. The threshold of 150 mg/L has gained widespread acceptance as a clinically useful cut-off for predicting severe disease. A systematic review and meta-analysis by Wu et al encompassing 41 studies with 6,156 cases demonstrated that CRP possesses significant diagnostic value for assessing acute pancreatitis severity.

Serum triglyceride levels demonstrated significant correlation with disease severity, increasing from 1.7 ± 0.6 mmol/L in mild cases to 6.2 ± 1.8 mmol/L in severe cases ($p<0.001$). These findings align closely with contemporary literature on hypertriglyceridemia-induced acute pancreatitis. The pathophysiological mechanisms linking hypertriglyceridemia to acute pancreatitis involve toxicity primarily mediated by free fatty acids, which can stimulate inflammation, damage cell membranes, injure endothelial cells, and induce organ damage.

The clinical implications of our findings include the utility of these readily available biomarkers for early risk stratification. Patients presenting with elevated triglycerides (>5.6 mmol/L) and CRP (>150 mg/L) should be identified as high-risk and considered for intensive monitoring. Serial monitoring of CRP levels can help assess treatment response and predict complications. Patients who recover from hypertriglyceridemia-induced acute pancreatitis require long-term management of dyslipidemia to prevent recurrence.

Study limitations include single-center design, relatively small sample size, and lack of serial measurements throughout disease course. Future multi-center studies with larger sample sizes are warranted to validate and extend these findings.

VI. CONCLUSION

This prospective observational study establishes a significant positive correlation between serum triglyceride levels and C-reactive protein in patients with acute pancreatitis. Both biomarkers demonstrated strong, statistically significant associations with disease severity. Elevated serum triglyceride and CRP levels were independently associated with progression from mild to severe acute pancreatitis.

The findings suggest that lipid metabolism plays a crucial role in modulating the inflammatory response in acute pancreatitis, with hypertriglyceridemia contributing to enhanced systemic inflammation as reflected by elevated CRP levels. C-reactive protein demonstrates rapid response to variations in inflammatory stimulus intensity, confirming its value in evaluating and monitoring acute pancreatitis.

The integration of serum triglyceride and CRP measurements into routine clinical assessment may facilitate improved early identification of high-risk cases, enable more informed clinical decision-making, and ultimately contribute to improved patient outcomes. Patients presenting with acute pancreatitis and elevated triglycerides should be recognized as being at increased risk for severe disease, warranting closer monitoring and consideration of specific lipid-lowering therapies.

In conclusion, serum triglyceride and C-reactive protein levels represent valuable, readily accessible biomarkers that correlate significantly with acute pancreatitis severity and can guide clinical management. Their routine measurement and appropriate interpretation can contribute to improved care and outcomes for patients with this potentially life-threatening condition.

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