ANALYSIS OF LIPID PROFILE IN PATIENTS WITH ACUTE PANCREATITIS AND CORRELATING IT WITH SOFA SCORE: A CROS-SECTIONAL STUDY.

ABSTRACT:

Background: Acute Pancreatitis (AP) is an inflammatory condition of the pancreas that can trigger a systemic inflammatory response and result in multiorgan dysfunction. Early evaluation of disease severity is crucial for optimizing management and improving clinical outcomes. Disturbances in lipid metabolism have increasingly been implicated in the severity of AP and the development of organ dysfunction.

Aim: To assess the variations in lipid profile, and to correlate lipid profile with SOFA score for organ dysfunction in patients with AP.

Methods: This isa cross-sectional study included total of 50 patients newly diagnosed with AP. The SOFA score was recorded. The lipid profile parameters were assayed in serum samples using clinical chemistry analyzer.

Results: The mean age of the participants was 48.2 ± 15.2 years, with male predominance (58%). The mean SOFA score was 2.7 ± 4.0. The mean triglyceride, cholesterol, LDL, VLDL, and HDL levels were 193.9 ± 79.9 mg/dL, 186.3 ± 59.8 mg/dL, 115.2 ± 43.3 mg/dL, 38.9 ± 17.3 mg/dL, and 37.5 ± 8.6 mg/dL, respectively. Significant positive correlations were observed between SOFA score and triglycerides (r=0.577, p=0.001), cholesterol (r=0.410, p=0.003), LDL (r=0.297, p=0.036), and VLDL (r=0.557, p=0.001). Whereas, HDL showed a significant negative correlation with SOFA score (r=-0.396, p=0.004).

Conclusion: Lipid profile abnormalities were significantly associated with organ dysfunction in patients with acute pancreatitis. Lower HDL levels were associated with increased severity. Routine lipid profile assessment may serve as a useful and cost-effective tool in the early prognostic evaluation of AP patients.

Keywords: Acute pancreatitis, Lipid profile, SOFA score, Organ dysfunction, Triglycerides

INTRODUCTION

Pancreatitis is a multifaceted condition with diverse causes, clinical presentations, and outcomes.[1] Early and efficient assessment is particularly valuable in resource-limited settings like India, where any delay in appropriate treatment can result in severe or life-threatening complications.[2] Acute pancreatitis (AP) is defined as a sudden inflammatory process of the pancreas. As per the Atlanta classification, a diagnosis of AP is established when any two of the below criteria are present: (i) Characteristic abdominal pain, (ii) Serum amylase or lipase levels exceeding three times the upper normal limit, and (iii) Imaging findings on CT or MRI consistent with pancreatitis.[3] Severity of AP classified as mild, moderately severe, or severe. Mild AP involves neither organ failure nor complications, while moderately severe AP includes transient organ dysfunction (lasting <48 hours) affecting one or multiple organs.[4]

The causes of AP are varied and include alcohol consumption, gallstones, severe hypertriglyceridemia, and genetic abnormalities involving the *PRSS1*, *SPINK1*, and *CFTR* genes.[5] Globally, AP incidence is approximately 34 per 100,000 persons annually.[6] In countries such as England, Denmark, and the United States, the incidence ranges from 4.8 to 24.2 per

100,000 people.[7] In India, the prevalence of pancreatitis is reported as 7.9 per 100,000, with rates of 8.6 and 8.0 per 100,000 among men and women, respectively.[5]

Several scoring systems are used to estimate prognosis in AP, including the Ranson score,[8] the APACHE II and III systems,[9] and the SOFA score.[10] These tools more accurately capture the evolving clinical status of critically ill individuals.[11] Among them, SOFA has been noted for its better performance and ease of use.[12] However, more recently, serum triglyceride (TG) levels have emerged as an important marker for assessing AP severity and aiding treatment decisions.[13] TG testing is straightforward, inexpensive, and yields rapid results. Triglycerides, central to lipid metabolism, appear to influence inflammatory pathways involved in AP. Elevated serum TG levels may exacerbate pancreatic injury through lipotoxic and inflammatory mechanisms. Although triglycerides themselves are not directly toxic, their breakdown by pancreatic lipase releases free fatty acids that cause lipotoxicity. The extent of pancreatic damage is primarily related to the inflammatory response and lipotoxic injury.[14,15]

Furthermore, metabolic syndrome has also been linked to both the development and severity of AP.[16] Severe hypertriglyceridemia (sHTG), defined as TG levels >11.3 mmol/L, is recognized as an important cause of AP.[17] With this context, current study was designed with the main purpose to assess the variations in lipid profile, and to correlate lipid profile with SOFA score for organ dysfunction in patients with AP.

METHODOLOGY

Ethical approval

The approval of study conduction was obtained from Institutional Ethical Committee of J. J. M. Medical College (JJMMC), Davangere, Karnatakawith Ref No. JJMMC/IEC-Syc-215-2024;

Dated:23.02.2024. A written informed consent was obtained from all the patients enrolled in to the study.

Study design and patients

This was a cross-sectional study involving50 patients diagnosed with AP at Department of Emergency Medicine, Bapuji Hospital, J. J. M. Medical College, Davangere. The sample size was calculated using the formula for estimating a proportion:

$$N = \frac{Z^2 pq}{d^2}$$

where Z is the standard normal deviate corresponding to a 95% confidence interval (1.96), p is the reportedprevalence ofAP was 30 per 100 000 population overall(0.03%),[18] $q = 100 - p$ (99.97), and d is the allowable error (5%). After substituting these values, and after considering a 10% attrition/non-response rate, the final sample size was rounded up to 50.

Inclusion criteria

Patients:

1. Age: >18 years
2. Newly diagnosed with AP
3. Willingness to participant in the study

Exclusion criteria

Patients with:

1. Pregnancy
2. Any complicated diseases, malignant tumor

Data collection and Assessment Parameters

Information regarding demographic data like age, gender, comorbidity, habits (alcoholism),presenting complaints, and SOFA score (Box I)were collected using the structured proforma.

Box I. SOFA score

Sequential [Sepsis-Related] Organ Failure Assessment Score					
	Score				
System	0	1	2	3	4
Respiratory					
PaO_2/FiO_2, mmHg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) and mechanically ventilated	<100 (13.3) and mechanically ventilated
Coagulation					
Platelets, x $10^3/\mu L$	≥150	<150	<100	<50	<20
Liver					
Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0 (204)
Cardiovascular	MAP ≥70 mmHg	MAP <70 mmHg	Dopamine <5 or dobutamine (any dose)	Dopamine 5.1–15 or epinephrine ≤0.1 or norepinephrine ≤0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Central nervous system					
Glasgow coma scale score	15	13–14	10–12	6–9	<6
Renal					
Creatinine, mg/dL (μmol/L) or urine output,	<1.2 (110)	1.2–1.9 (110–170)	2.0–3.4 (171–299)	3.5–4.9 (300–440) or <500 mL/day	>5.0 (440) <200 mL/day

Lipid profile assay

The 2 ml of blood sample was drawn from superficial veins located in the antecubital fossa. The serum was separated by centrifugation at 5000 rpm for 5 mins. The lipid profile parameters in the serum sample *viz.* triglycerides (mg/dL), cholesterol (mg/dL), low-density lipoprotein (LDL),

very low-density lipoprotein (VLDL), and high-density lipoprotein (HDL) were analyzed using ARCHITECT c16000 clinical chemistry analyzer (Abbott Diagnostics, USA).

STATISTICAL ANALYSIS

Data were entered in Microsoft Excel 2021 and statistical analysis was done using IBM Statistical Software for Social Sciences (SPSS) version 23. Categorical variables were represented in the form of frequencies and percentages. Continuous variables were presented as descriptive statistics such as mean and standard deviation (SD). Inferential statistics like Pearson's correlation was applied to correlate lipid profile with SOFA Score. $p \leq 0.05$ was considered statistically significant.

RESULTS

The mean age was 48.2 ± 15.2 years, with the largest proportion aged >60 years (26.0%), followed by 41–50 years (24.0%). Males constituted the majority of the cohort (58.0%), while females accounted for 42.0%. A history of alcohol abuse was present in 54.0% of patients. The most common presenting complaint was abdominal pain with vomiting (54.0%), followed by the triad of fever, abdominal pain, and vomiting (30.0%). Less frequent presentations included abdominal distension and fever in combination with abdominal pain and vomiting, each accounting for ≤6.0% of cases (Table 1).

Table 1. Demographic and baseline characteristics

Variables	N (%)
Age (Years)	
21 – 30	10 (20.0)

31 – 40	5 (10.0)
41 – 50	12 (24.0)
51 – 60	10 (20.0)
>60	13 (26.0)
Mean ± SD	48.2 ± 15.2
Gender	
Female	21 (42.0)
Male	29 (58.0)
Alcohol abuse	
Yes	27 (54.0)
No	23 (46.0)
Presenting complaints	
Abdominal pain + Vomiting	27 (54.0)
Fever + Abdominal pain + Vomiting	15 (30.0)
Fever + Abdominal pain + Vomiting + Abdominal distension	3 (6.0)
Abdominal pain	3 (6.0)
Abdominal distension + Abdominal pain + Vomiting	1 (2.0)
Fever + Abdominal pain	1 (2.0)

Values wereN (%) unless otherwise stated

The distribution of co-morbidities revealed that 22.0% of the patients had no associated comorbid conditions. Among those with co-morbidities, the most common condition was the combination of hypertension and Type 2 diabetes mellitus, accounting for 16.0%, followed by Type 2 diabetes mellitus alone at 14.0% and hypertension alone at 12.0% (Figure 1).

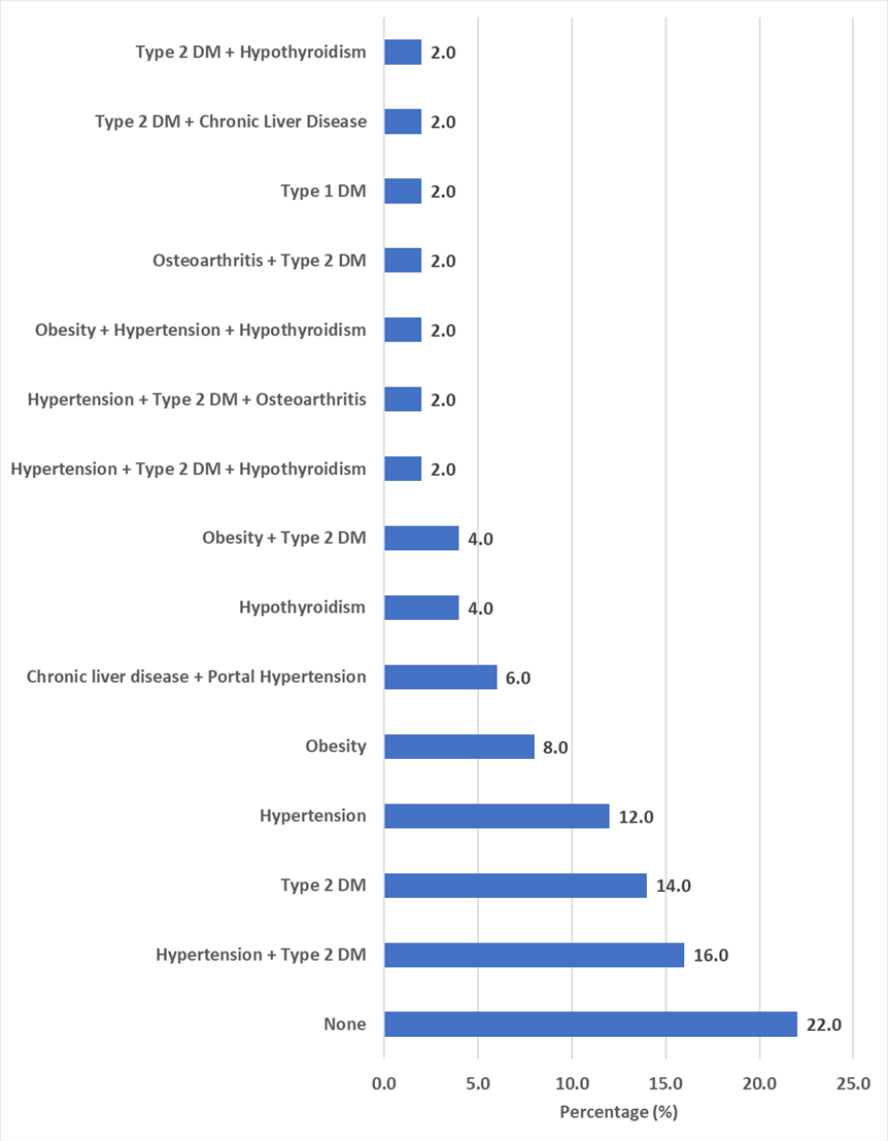

Figure 1. Distribution of patients based on comorbidities

The mean (±SD) triglyceride, cholesterol, LDL, VLDL, and HDL levels was 193.9 (±79.9) mg/dL, 186.3 (±59.8) mg/dL, 115.2 (±43.3) mg/dL, 38.9 (±17.3) mg/dL, and 37.5 (±8.6) mg/dL respectively. The findings demonstrated variability in lipid parameters among the study participants. The mean (±SD) SOFA score was 2.7 (±4.0), with values ranging from 0 to 18, indicating substantial heterogeneity in the severity of organ dysfunction among study participants (Table 2).

Table 2. Lipid profile and SOFA score

Variables	Values	Range
Triglycerides, mg/dL	193.9 ± 79.9	90 – 389
Cholesterol, mg/dL	186.3 ± 59.8	96 – 300
LDL, mg/dL	115.2 ± 43.3	38 – 209
VLDL, mg/dL	38.9 ± 17.3	17 – 88
HDL, mg/dL	37.5 ± 8.6	20 – 55
SOFA score	2.7 ± 4.0	0 – 18

Values were mean ± SD unless otherwise stated
LDL, Low-density lipoprotein; VLDL, Very low-density lipoprotein; HDL, High-density lipoprotein; SOFA, Sequential organ failure assessment

Pearson's correlation analysis between SOFA score and lipid profile parameters demonstrated a statistically significant positive correlation between SOFA score and triglyceride levels ($r = 0.577$, $p = 0.001$), total cholesterol levels ($r = 0.410$, $p = 0.003$), LDL levels ($r = 0.297$, $p = 0.036$), and VLDL levels ($r = 0.557$, $p = 0.001$). In contrast, HDL levels showed a statistically significant negative correlation with SOFA score ($r = -0.396$, $p = 0.004$). These findings indicated that higher SOFA scores were associated with elevated triglycerides, cholesterol, LDL, and VLDL levels, whereas HDL levels tended to decrease with increasing SOFA scores (Table 3 and Figure 2).

Table 3. Correlation between SOFA score and lipid profile

SOFA score vs.	r - value	p - value
Triglycerides	0.577	0.001
Cholesterol	0.410	0.003
LDL	0.294	0.036
VLDL	0.557	0.001
HDL	-0.396	0.004

Values were mean ± SD unless otherwise stated
LDL, Low-density lipoprotein; VLDL, Very low-density lipoprotein; HDL, High-density lipoprotein; SOFA, Sequential organ failure assessment

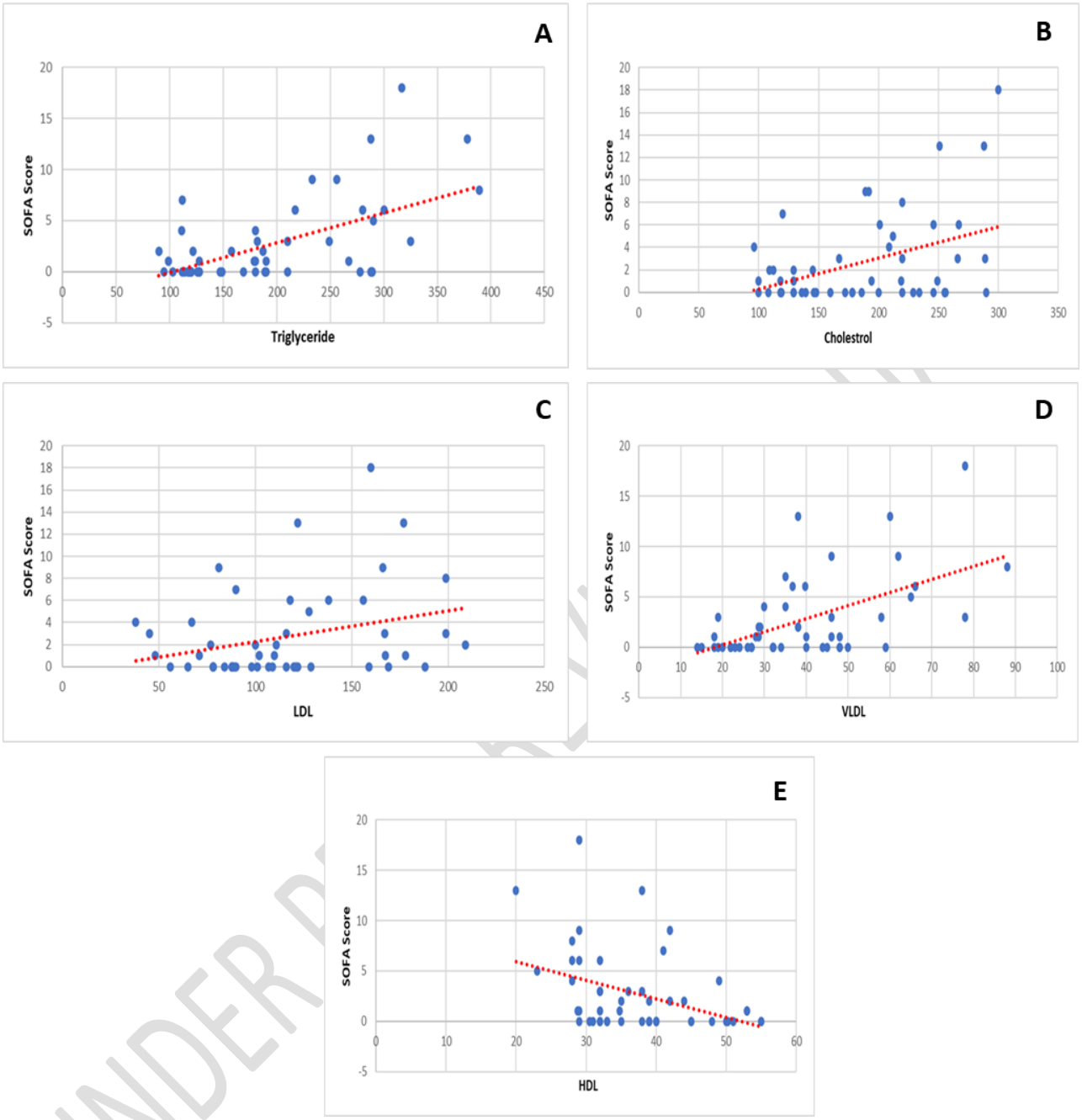

Figure 2. Correlation between SOFA score with triglycerides (A), cholesterol (B), LDL (C), VLDL (D), and HDL (E)

DISCUSSION

Acute Pancreatitis is an inflammatory disorder of the pancreas associated with considerable morbidity and mortality, particularly in cases complicated by organ dysfunction. Prompt

recognition of disease severity is crucial for early intervention and improved clinical outcomes.[19] Disturbances in lipid metabolism, especially hypertriglyceridemia and other lipid profile abnormalities, have increasingly been implicated in the progression and severity of AP as well as the systemic inflammatory response. The SOFA score is commonly utilized to assess organ dysfunction in critically ill individuals.[20]Therefore, the present cross-sectional study was undertaken to evaluate lipid profile alterations in AP patients and correlate these parameters with SOFA scores for assessing organ dysfunction.

In the present study, the mean age of participants was 48.2 years, and the largest proportion of patients belonged to the age group above 60 years (26%). These findings suggest that AP affects a wide age range but occurs more frequently among middle-aged and elderly populations. Similar findings were described by Koziel et al., who reported that AP predominantly affects middle-aged adults, with disease severity increasing among older individuals.[21]A predominance of male participants was observed in the current study, with males accounting for 58% of the study population. This observation aligns with report by Rajesh et al., who documented a higher incidence of AP among males, largely attributed to increased alcohol intake and metabolic risk factors.[22]

The presence of hypertension and type 2 diabetes mellitus was established as being the most common comorbid conditions among the population under consideration. Metabolic disorders such as obesity, diabetes mellitus, and dyslipidemia have been found to be causative factors in the development of pancreatic inflammation. This finding is consistent with the findings of Mikolasevis et al., which highlighted the significant role played by metabolic syndrome in the development and severity of AP.[23]Alcohol consumption was reported in 46% of participants. Alcohol remains one of the major etiological contributors to AP. Similar findings

were reported by Apte et al., where alcohol use and gallstone disease were identified as the leading causes of AP globally.[24]

Abdominal pain accompanied by vomiting was the most frequent presenting symptom, occurring in 54% of participants. Fever associated with abdominal pain and vomiting was noted in 30% of subjects. Existing literature similarly describes severe epigastric pain radiating to the back along with nausea and vomiting as the hallmark clinical features of AP.[25]

The mean triglyceride level was 193.9 mg/dL, while the mean cholesterol, LDL, VLDL, and HDL levels were 186.3 mg/dL, 115.2 mg/dL, 38.9 mg/dL, and 37.5 mg/dL, respectively. Considerable variability in lipid profile parameters was observed among the participants. Literature reports evidenced that hypertriglyceridemia is increasingly recognized as both a causative factor and a consequence of AP. Elevated triglyceride levels contribute to pancreatic injury through mechanisms such as free fatty acid toxicity, endothelial dysfunction, and inflammatory cytokine release.[26]The findings of the current study are comparable to those of Yang et al. wherein authors reported a strong association between elevated triglycerides, inflammatory burden, and disease severity.[27]

The reduced HDL levels detected in our study are clinically relevant because HDL possesses anti-inflammatory and antioxidant properties. Lower HDL levels may reflect heightened systemic inflammatory activity and impaired lipid metabolism during acute illness.[28]Similar observations were documented by Zhang et al., who demonstrated that reduced HDL levels were associated with increased pancreatic necrosis and systemic complications.[29]The elevated VLDL levels identified in the present study may result from altered hepatic lipid metabolism and increased transport of triglycerides during inflammatory states. These findings

were in accordance with Khan et al., who observed significantly elevated VLDL concentrations in severe AP compared with mild disease.[30]

The other important finding from the current research is the positive association of the SOFA score with triglycerides ($r = 0.577$, $p = 0.001$), cholesterol ($r = 0.410$, $p = 0.003$), LDL ($r = 0.297$, $p = 0.036$), and VLDL ($r = 0.557$, $p = 0.001$). HDL, on the other hand, showed a significant negative correlation with the SOFA score ($r = -0.396$, $p = 0.004$). These results imply that there were important alterations in the lipid profile that were strongly associated with organ dysfunction.[31]The SOFA score is a validated system extensively used for assessing multiorgan dysfunction in critically ill patients. The current findings support those of Wu et al., who indicated that triglycerides and LDL were positively correlated with organ dysfunction score and length of stay.[32]

The inverse relationship between HDL and SOFA score observed in this study is particularly noteworthy. HDL plays an essential role in neutralizing endotoxins, limiting oxidative stress, and regulating inflammatory pathways. Therefore, lower HDL levels may predispose patients to exaggerated systemic inflammation and subsequent organ failure.[33]This was further supported by Thacker et al., where reduced HDL levels were strongly associated with inflammatory cytokine activity and severity of critical illness.[34]The positive association between VLDL and SOFA score suggests that VLDL may serve as a marker of systemic metabolic dysregulation in AP. Elevated VLDL levels likely reflect increased hepatic triglyceride production and impaired lipid clearance secondary to inflammation.[31]

However, certain limitations should be taken into account. The sample size was rather small, with only 50 patients enrolled, which may limit the possibility for external validity. The study design, being a single center cross-sectional research, does not guarantee the results'

applicability to the entire AP population. The lipid profile parameters were measured at one time point; longitudinal measurements of lipids were not performed. Other factors influencing lipid levels, such as nutrition, previous use of lipid-lowering therapy, and preexisting dyslipidemia, were not considered.

CONCLUSION

In conclusion, lipid profile abnormalities were significantly associated with organ dysfunction in acute pancreatitis. Lower HDL levels were associated with increased severity. Thus, dyslipidemia play an essential role in the pathogenesis and course of acute pancreatitis. Routine lipid profile assessment may serve as a useful and cost-effective tool in the early prognostic evaluation of acute pancreatitis patients.

REFERENCES

1. Ellis I, Lerch MM, Whitcomb DC; Consensus Committees of the European Registry of Hereditary Pancreatic Diseases, Midwest Multi-Center Pancreatic Study Group, International Association of Pancreatology. Genetic testing for hereditary pancreatitis: guidelines for indications, counselling, consent and privacy issues. Pancreatology. 2001;1(5):405-15.
2. Toppo S, Besra RC, Mardi N, Bodra P, Mundu M, Kujur A, Tudu MB, Bharti B, Baskey H. To evaluate the effectiveness and usefulness of qSOFA scoring in predicting mortality in acute pancreatitis - A teaching institute study. J Family Med Prim Care. 2025;14(8):3233-3238.
3. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Gregory G. Tsiotos, and Santhi Swaroop Vege. Classification of Acute Pancreatitis-2012: Revision of

the Atlanta Classification and Definitions by International Consensus. Gut. 2013;62(1):102-11.

4. Vege SS, Gardner TB, Chari ST. Low mortality, and high morbidity in severe acute pancreatitis without organ failure: a case for revising the Atlanta classification to include moderately severe acute pancreatitis. Am J Gastroenterol. 2009; 104:710-5.
5. Nawahirsha S, Kumar BS, Naik BK, Parthasarathy EA. Clinical profile and outcomes of patients with acute pancreatitis and correlation with severity index from a tertiary care centre in South India-retrospective analysis. Int J Adv Med 2021; 8:814-8.
6. Tahir MB, Tahir AB, Meer F, Hussain I, Rahman MS, Saeed MR, Rehman UU, Ali RS, Asif AE, Ahmed A, Khan TM. Association Between Serum Triglyceride Levels and the Severity of Acute Pancreatitis: A Retrospective Study. Cureus. 2025;17(1):e78053.
7. Sehgal R, Singh IP, Mittal J. Clinical Profile and Outcome of Patients with Severe Acute Pancreatitis. AsianJ.Med.Res.2020;9(3):8-11.
8. Ranson JH, Rifkind KM, Roses DF, Fink SD, Eng K, Spencer FC. Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet. 1974;139(1):69-81.
9. Williams M, Simms HH. Prognostic usefulness of scoring systems in critically ill patients with severe acute pancreatitis. Crit Care Med. 1999; 27:901–7.
10. Russell PS, Mittal A, Brown L, McArthur C, Phillips AJR, Petrov M, Windsor JA. Admission, management and outcomes of acute pancreatitis in intensive care. ANZ J Surg. 2017;87(12):E266-E270.

11. de Grooth HJ, Geenen IL, Girbes AR, Vincent JL, Parienti JJ, Oudemans-van Straaten HM. SOFA and mortality endpoints in randomized controlled trials: a systematic review and meta-regression analysis. Crit Care. 2017;21(1):38.
12. Juneja D, Gopal PB, Ravula M. Scoring systems in acute pancreatitis: which one to use in intensive care units? J Crit Care 2010; 25:358.e9–15.
13. Haq IU, Daud M, Khan MA, Ullah F, Attullah A, Rehman MU, Hussain K, Habib M. Impact of Serum Triglyceride Levels on Severity and Outcomes in Acute Biliary Pancreatitis: A Retrospective Cohort Study. Cureus. 2024;16(8):e65928.
14. Zhang Q, Xu Q, Zhu Q, Zhu L, Pan C, Chen J, Liu J, Deng D, Chen W, Chen C. Association between serum triglyceride level and severity of acute biliary pancreatitis. Pancreatology. 2024;24(3):343-349.
15. Qiu M, Zhou X, Zippi M, Goyal H, Basharat Z, Jagielski M, Hong W. Comprehensive review on the pathogenesis of hypertriglyceridaemia-associated acute pancreatitis. Ann Med. 2023;55(2):2265939.
16. Niknam R, Moradi J, Jahanshahi KA, Mahmoudi L, Ejtehadi F. Association between metabolic syndrome and its components with severity of acute pancreatitis. Diabetes Metab Syndr Obes. 2020; 13:1289–96.
17. Zhu Y, Pan X, Zeng H, He W, Xia L, Liu P, Zhu Y, Chen Y, Lv N. A study on the etiology, severity, and mortality of 3260 patients with acute pancreatitis according to the revised Atlanta classification in Jiangxi, China over an 8 year period. Pancreas. 2017; 46:504–9.

18. Roberts SE, Akbari A, Thorne K, Atkinson M, Evans PA. The incidence of acute pancreatitis: impact of social deprivation, alcohol consumption, seasonal and demographic factors. Aliment Pharmacol Ther. 2013;38(5):539-48.
19. Wang CF, Tariq A, Chandra S. Acute pancreatitis. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2026.
20. Mazibuko J, Elebo N, Williams AA, Omoshoro-Jones J, Devar JW, Smith M, Cacciatore S, Fru PN. Metabolites and lipoproteins may predict the severity of early acute pancreatitis in a South African cohort. Biomedicines. 2024;12(11):2431.
21. Koziel D, Gluszek-Osuch M, Suliga E, Zak M, Gluszek S. Elderly persons with acute pancreatitis - specifics of the clinical course of the disease. Clin Interv Aging. 2018;14:33-41.
22. Rajesh S, Raju PV. A clinical study on epidemiology and etiological factors of acute pancreatitis-hospital based study in South India. Int Surg J. 2021;8(10):3060-3063.
23. Mikolasevic I, Milic S, Orlic L, Poropat G, Jakopcic I, Franjic N, Klanac A, Kristo N, Stimac D. Metabolic syndrome and acute pancreatitis. Eur J Intern Med. 2016;32:79-83.
24. Apte MV, Pirola RC, Wilson JS. Mechanisms of alcoholic pancreatitis. J Gastroenterol Hepatol. 2010;25(12):1816-1826.
25. National Health Service. Acute pancreatitis. NHS website. Available from: https://www.nhs.uk/conditions/acute-pancreatitis/ Accessed 2026 May 12.
26. Gao JW, Hao QY, Lin Y, Li ZH, Huang ZG, Bai ZQ, Zhang HF, Wu YB, Xiong ZC, You S, Wang JF, Zhang SL, Liu PM. Variability in lipid profiles during young adulthood and the risk of coronary artery calcium incidence in midlife: insights from the CARDIA study. Circ Cardiovasc Imaging. 2024;17(9):e016842.

27. Yang AL, McNabb-Baltar J. Hypertriglyceridemia and acute pancreatitis. Pancreatology. 2020;20(5):795-800.
28. Barter PJ, Nicholls S, Rye KA, Anantharamaiah GM, Navab M, Fogelman AM. Antiinflammatory properties of HDL. Circ Res. 2004;95(8):764-772.
29. Zhang Y, Guo F, Li S, Wang F, Meng Z, Zhao J, Liu Z, Wang B, Fan P, Wang C, Wu H. Decreased high density lipoprotein cholesterol is an independent predictor for persistent organ failure, pancreatic necrosis and mortality in acute pancreatitis. Sci Rep. 2017;7(1):8064.
30. Khan J, Nordback I, Sand J. Serum lipid levels are associated with the severity of acute pancreatitis. Digestion. 2013;87(4):223-8.
31. Fang C, Ding Y, Wang X, Teng X. Metabolic disturbances in acute pancreatitis: mechanisms and therapeutic implications. Front Endocrinol. 2025;16:1579457.
32. Wu Q, Fu M, Zheng K, Bo H, Yang H, Zhong X, Liang G, Xu Y, Hao B, Hu Z, Zhang Z, Jin X, Kang Y. Elevated triglycerides level in hospital stay as a risk factor of mortality in patients with severe acute pancreatitis. PLoS One. 2018;13(11):e0207875.
33. Feingold KR, Grunfeld C. Effect of inflammation on HDL structure and function. Curr Opin Lipidol. 2016;27(5):521-530.
34. Thacker SG, Zarzour A, Chen Y, Alcicek MS, Freeman LA, Sviridov DO, Demosky SJ Jr, Remaley AT. High-density lipoprotein reduces inflammation from cholesterol crystals by inhibiting inflammasome activation. Immunology. 2016;149(3):306-319.