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Blood Lipid Profiles as Biomarkers for Disease Activity In Patients with Severe Ulcerative Colitis: A Cross-Sectional Observational Study

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A B S T R A C T

Background: Ulcerative colitis (UC) is a chronic inflammatory condition characterized by periods of activity and remission. Identifying accessible biomarkers that correlate with disease activity could aid in disease monitoring. This study evaluated the potential of lipid profile components as biomarkers for disease activity in patients with severe UC.

Methods: A retrospective analysis was conducted on 220 patients diagnosed with severe UC. Fasting lipid profiles—including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TGs)—were compared between patients in active and remission phases. Correlation analyses were performed between lipid values and C-reactive protein (CRP) levels as well as Mayo scores. Logistic regression and ROC curve analysis assessed the predictive performance of lipid parameters for active disease.

Results: No statistically significant differences were found in TC, LDL-C, HDL-C, or TG levels between patients with active UC and those in remission (all p-values > 0.39). Correlations between lipid markers and CRP or Mayo scores were weak and nonsignificant (HDL-C and CRP: r = 0.01, p = 0.87; TGs and CRP: r = –0.05, p = 0.63). None of the lipid components were independent predictors of disease activity in multivariate analysis. The combined lipid model yielded a modest discriminative performance (AUC = 0.58).

Conclusion: Serum lipid profiles did not significantly differ between active and remission states of severe UC and showed limited utility as standalone biomarkers of disease activity. Further prospective studies are warranted to explore alternative noninvasive markers for monitoring UC severity.

Keywords: ulcerative colitis, lipid profile, disease activity, HDL cholesterol, biomarkers.

INTRODUCTION

Ulcerative colitis (UC) is a chronic inflammatory bowel disease characterized by relapsing episodes of mucosal inflammation in the colon. Accurate assessment of disease activity is essential for guiding treatment and predicting outcomes. While clinical scoring systems and endoscopic findings remain standard tools, there is increasing interest in identifying noninvasive biomarkers that reflect underlying inflammatory activity and can be easily monitored in routine practice.

Lipid metabolism has emerged as a potential area of relevance in inflammatory diseases, including UC. Inflammatory cytokines are known to influence hepatic lipid synthesis, lipoprotein metabolism, and lipid transport, suggesting a mechanistic basis for altered lipid profiles during active inflammation. Recent studies have explored the utility of conventional lipid panels in this context. For example, Zhu et al. (2025) reported that patients with severe UC exhibited lower HDL-C and elevated triglyceride levels compared to those in remission, proposing their potential use as adjunctive biomarkers of disease activity [1]. Similarly, Liu et al. (2022) found that dyslipidemia was significantly associated with severe disease and worse prognosis in a Chinese UC cohort [4].

Expanding on lipid-related biomarkers, Marinelli et al. (2022) highlighted elevated serum PCSK9 levels in active UC patients, suggesting it may serve as a more specific surrogate marker of mucosal inflammation [2]. Metabolomic and lipidomic profiling have also revealed distinctive lipid signatures in UC. Tews et al. (2024) demonstrated unique lipidomic profiles in both UC and Crohn's disease compared to healthy individuals, implicating disrupted lipid pathways in disease pathogenesis [5]. Moreover, Koutroumpakis et al. (2016), in a large cohort study, reported long-term associations between abnormal lipid profiles and disease severity in IBD patients, reinforcing the relevance of metabolic changes over time [3].

It is also important to consider how treatment modalities influence lipid parameters. For instance, Sands et al. (2021) observed that tofacitinib therapy in UC patients led to shifts in lipid profiles, raising questions about both cardiovascular risk and the interpretive value of lipid levels as disease markers under pharmacologic modulation [6].

Despite these insights, the clinical utility of routine lipid panels as biomarkers for UC activity remains uncertain. Existing studies vary in population characteristics and methodologies, and their findings are not always consistent. Furthermore, many investigations rely on advanced profiling techniques not typically available in routine clinical practice.

Given this uncertainty, the present study aimed to evaluate whether conventional lipid parameters—total cholesterol (TC), LDL-C, HDL-C, and triglycerides (TGs)—differ between active and remission phases of severe UC, and whether they correlate with established markers of inflammation and clinical disease activity. Through this analysis, we sought to determine the potential role of lipid profiles as accessible, noninvasive indicators of disease activity in UC.

OBJECTIVES

The primary objective of this study was to evaluate the association between blood lipid profiles and disease activity in patients with severe ulcerative colitis (UC).
Specific objectives included:

1. To compare lipid parameters (total cholesterol, LDL-C, HDL-C, and triglycerides) between patients in active disease and those in clinical remission.
2. To assess correlations between lipid profiles and inflammatory markers, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR).
3. To identify lipid-based predictors of active disease using multivariable logistic regression analysis.
4. To evaluate the diagnostic performance of lipid biomarkers in discriminating active UC from remission using ROC curve analysis.

METHODS

Study Design and Setting

This cross-sectional observational study was conducted at SLN Medical College and Hospital (Koraput, India) over a four-month period (March–June 2025). Adult patients diagnosed with severe ulcerative colitis (UC), based on clinical, endoscopic, and histological criteria, were consecutively recruited from the gastroenterology outpatient and inpatient services.

Participants

Inclusion criteria were:

- Age ≥18 years
- Confirmed diagnosis of ulcerative colitis
- Classified as either in clinical remission or having active disease based on clinical assessment and physician global assessment

Exclusion criteria included:

- History of Crohn's disease or indeterminate colitis
- Use of lipid-lowering agents
- Active systemic infections or chronic liver/kidney disease
- Incomplete laboratory data

Data Collection

Demographic, clinical, and biochemical data were collected from medical records and patient interviews. Laboratory investigations included:

- Lipid profile: total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides
- Inflammatory markers: C-reactive protein (CRP), erythrocyte sedimentation rate (ESR)
- Other variables: hemoglobin, albumin, total leukocyte count, BMI, and disease duration

Disease activity status was categorized as **active** or **in remission** based on the Truelove and Witts criteria and physician global assessment at the time of data collection.

Statistical Analysis

Descriptive statistics were presented as mean ± standard deviation (SD) for continuous variables and counts (percentages) for categorical variables.

Group comparisons of lipid parameters between active and remission groups were performed using independent samples t-tests. Pearson correlation coefficients were used to assess relationships between lipid profiles and inflammatory markers (CRP, ESR).

Multivariable logistic regression analysis was conducted to identify independent lipid-related predictors of active disease. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI).

A Receiver Operating Characteristic (ROC) curve was plotted to assess the diagnostic performance of key lipid biomarkers, and the Area Under the Curve (AUC) was calculated.

All statistical analyses were performed using Python v3.11, Stats models, and Scikit-learn, with SPSS v26 used for verification. A p-value <0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 220 patients diagnosed with severe ulcerative colitis (UC) were enrolled in the study over a four-month period at SLN Medical College and Hospital, Koraput. The mean age of participants was 41.5 ± 11.4 years, and the cohort showed a slight female predominance (53.6%). The mean body mass index (BMI) was 23.9 ± 3.4 kg/m², and the average disease duration was 4.5 ± 4.9 years. The average Mayo score among participants was 7.5 ± 1.9, indicating moderate to severe disease activity. Mean C-reactive protein (CRP) was elevated at 10.9 ± 6.7 mg/L, consistent with systemic inflammation.

Demographic profiles revealed that 60.0% of patients were never-smokers, and 55.0% resided in rural areas. In terms of socioeconomic background, the majority belonged to the middle (50.0%) or low (40.0%) strata. Disease extent was most commonly left-sided colitis (50.0%), followed by pancolitis (30.0%) and proctitis (20.0%). At the time of assessment, 65.0% of patients had active disease, while 35.0% were in clinical remission. Use of corticosteroids was noted in 55.0% of cases, and 35.0% of patients were on biologic therapy. Extraintestinal manifestations (EIMs) were present in 30.0% of participants, and 40.0% had a history of prior hospitalization.

Table 1. Baseline Characteristics of Study Participants (N = 220)

Variable	Value
Continuous Variables	
Age, years (mean ± SD)	41.5 ± 11.4
BMI, kg/m² (mean ± SD)	23.9 ± 3.4
Disease Duration, years	4.5 ± 4.9
Mayo Score (mean ± SD)	7.5 ± 1.9
CRP, mg/L (mean ± SD)	10.9 ± 6.7
Categorical Variables	
Sex	Male: 102 (46.4%), Female: 118 (53.6%)
Smoking Status	Never: 132 (60.0%), Former: 55 (25.0%), Current: 33 (15.0%)
Residence	Urban: 99 (45.0%), Rural: 121 (55.0%)
Socioeconomic Status	Low: 88 (40.0%), Middle: 110 (50.0%), High: 22 (10.0%)

Variable	Value
Extent of Disease	Proctitis: 44 (20.0%), Left-sided: 110 (50.0%), Pancolitis: 66 (30.0%)
Disease Activity	Active: 143 (65.0%), Remission: 77 (35.0%)
Corticosteroids Use	Yes: 121 (55.0%), No: 99 (45.0%)
Biologic Therapy	Yes: 77 (35.0%), No: 143 (65.0%)
Extraintestinal Manifestations (EIMs)	Yes: 66 (30.0%), No: 154 (70.0%)
Prior Hospitalization	Yes: 88 (40.0%), No: 132 (60.0%)

Comparison of Lipid Profiles by Disease Activity

We assessed differences in blood lipid levels between patients with active ulcerative colitis and those in clinical remission. As summarized in Table 2, there were no statistically significant differences in total cholesterol, LDL, HDL, or triglyceride levels between the two groups.

Mean total cholesterol levels were nearly identical in both groups (171.7 ± 35.3 mg/dL in active disease vs. 171.6 ± 33.8 mg/dL in remission; p = 0.9855). LDL levels were slightly higher in the active group (100.8 ± 24.7 mg/dL) compared to those in remission (97.7 ± 26.1 mg/dL), but this difference was not statistically significant (p = 0.3982; Cohen's d = 0.12). Similarly, HDL levels (44.5 ± 9.7 vs. 44.5 ± 10.8 mg/dL; p = 0.9919) and triglyceride levels (150.4 ± 52.4 vs. 148.3 ± 55.1 mg/dL; p = 0.7783) showed no meaningful variation between the groups

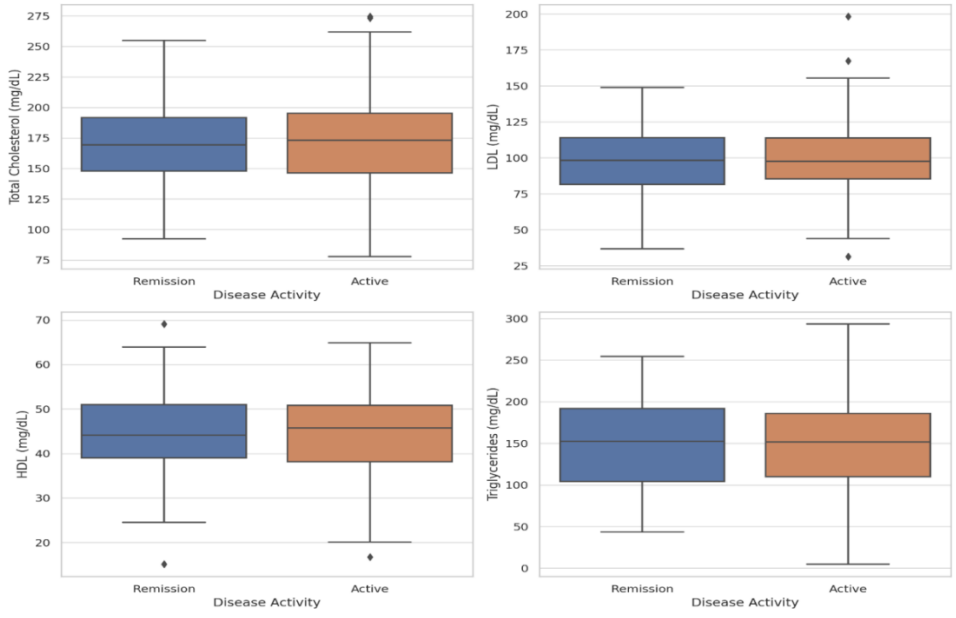
Table 2. Comparison of Lipid Profiles Between Active Disease and Remission Groups

Lipid Marker	Active Disease (Mean ± SD)	Remission (Mean ± SD)	t-value	p-value	Cohen's d
Total Cholesterol (mg/dL)	171.7 ± 35.3	171.6 ± 33.8	0.02	0.9855	0.00
LDL (mg/dL)	100.8 ± 24.7	97.7 ± 26.1	0.85	0.3982	0.12
HDL (mg/dL)	44.5 ± 9.7	44.5 ± 10.8	-0.01	0.9919	0.00
Triglycerides (mg/dL)	150.4 ± 52.4	148.3 ± 55.1	0.28	0.7783	0.04

Note: Independent samples t-test was used to compare lipid parameters between patients with active disease and those in remission. Cohen's d values indicate effect size.

Figure 1. Comparison of Lipid Profiles by Disease Activity in Severe Ulcerative Colitis

Boxplots illustrating distributions of total cholesterol, LDL, HDL, and triglyceride levels among patients with active disease versus those in remission. No significant differences were observed between groups for any lipid parameter (p > 0.05 for all comparisons).



Correlation of Lipid Profiles with Inflammatory Markers

To evaluate the relationship between lipid metabolism and inflammatory burden, Pearson correlation analysis was conducted between serum lipid markers and inflammatory indices (CRP and Mayo score). As shown in Table 3, no statistically significant correlations were observed between any lipid parameter and either CRP or Mayo score.

Total cholesterol showed a weak, negative correlation with CRP ($r = –0.03$, $p = 0.6846$) and virtually no correlation with Mayo score ($r = –0.00$, $p = 0.9952$). LDL levels also showed negligible associations with CRP ($r = –0.00$, $p = 0.9778$) and Mayo score ($r = –0.02$, $p = 0.7562$). Similarly, HDL and triglyceride levels did not demonstrate meaningful correlations with inflammatory burden (all $p > 0.5$).

These findings suggest that in patients with severe ulcerative colitis, circulating lipid levels are largely independent of traditional inflammatory markers.

Table 3. Correlation of Lipid Profiles with Inflammatory Markers

Lipid Marker	Inflammatory Marker	Pearson r	p-value
Total Cholesterol	CRP	–0.03	0.6846
Total Cholesterol	Mayo Score	–0.00	0.9952
LDL	CRP	–0.00	0.9778
LDL	Mayo Score	–0.02	0.7562
HDL	CRP	0.01	0.8712
HDL	Mayo Score	–0.01	0.8804
Triglycerides	CRP	–0.05	0.5036
Triglycerides	Mayo Score	0.00	0.9864

Note: Pearson correlation coefficients (r) and corresponding p-values are reported. None of the lipid parameters showed significant associations with inflammatory burden ($p > 0.05$).

Multivariable Regression Analysis of Lipid Predictors of Inflammation

To explore the potential of lipid markers as predictors of inflammatory burden and disease severity in severe ulcerative colitis, two multivariable linear regression models were constructed. One model assessed predictors of C-reactive protein (CRP) levels, and the other evaluated associations with Mayo clinical score. Both models adjusted for age, sex, and all four major lipid parameters (total cholesterol, LDL, HDL, and triglycerides).

As shown in Table 4A, none of the lipid markers demonstrated statistically significant associations with CRP levels. Triglycerides showed a negative trend ($\beta = –0.0033$, $p = 0.1746$), though this did not reach significance. Total cholesterol ($\beta = 0.0005$, $p = 0.8955$), LDL ($\beta = –0.0016$, $p = 0.7587$), and HDL ($\beta = 0.0021$, $p = 0.8681$) also showed no meaningful predictive value. The model's intercept was statistically significant ($p < 0.001$), suggesting elevated baseline CRP values, but none of the independent variables contributed significantly.

In the model predicting Mayo score (Table 4B), similar results were found: all lipid variables had nonsignificant coefficients (all $p > 0.1$), and neither age nor sex emerged as significant contributors. These findings reinforce the earlier correlation analysis, indicating a weak or absent relationship between serum lipid levels and disease severity indices in this cohort.

Table 4A. Multivariable Linear Regression Predicting CRP Levels

Variable	Coefficient	Std. Error	t	p-value	95% CI Lower	95% CI Upper
Constant	11.1176	4.4208	2.5148	0.0126	2.4034	19.8318
Total Cholesterol	-0.0063	0.0133	-0.4729	0.6368	-0.0325	0.0199
LDL	-0.0006	0.0184	-0.0305	0.9757	-0.0368	0.0356
HDL	0.0051	0.0458	0.1105	0.9121	-0.0853	0.0954
Triglycerides	0.0071	0.0086	0.8165	0.4151	-0.01	0.0241
Age	-0.0155	0.0404	-0.3823	0.7026	-0.0951	0.0642
Sex Code	0.6056	0.9172	0.6602	0.5098	-1.2024	2.4136

Table 4B. Multivariable Linear Regression Predicting Mayo Score

Variable	Coefficient	Std. Error	t	p-value	95% CI Lower	95% CI Upper
Constant	7.8735	1.2356	6.3724	0.0	5.438	10.309
Total Cholesterol	0.0005	0.0037	0.1315	0.8955	-0.0068	0.0078
LDL	-0.0016	0.0051	-0.3075	0.7587	-0.0117	0.0085
HDL	0.0021	0.0128	0.1662	0.8681	-0.0231	0.0274
Triglycerides	-0.0033	0.0024	-1.3622	0.1746	-0.0081	0.0015
Age	0.0058	0.0113	0.5176	0.6053	-0.0164	0.0281
Sex Code	-0.2248	0.2564	-0.877	0.3815	-0.7301	0.2805

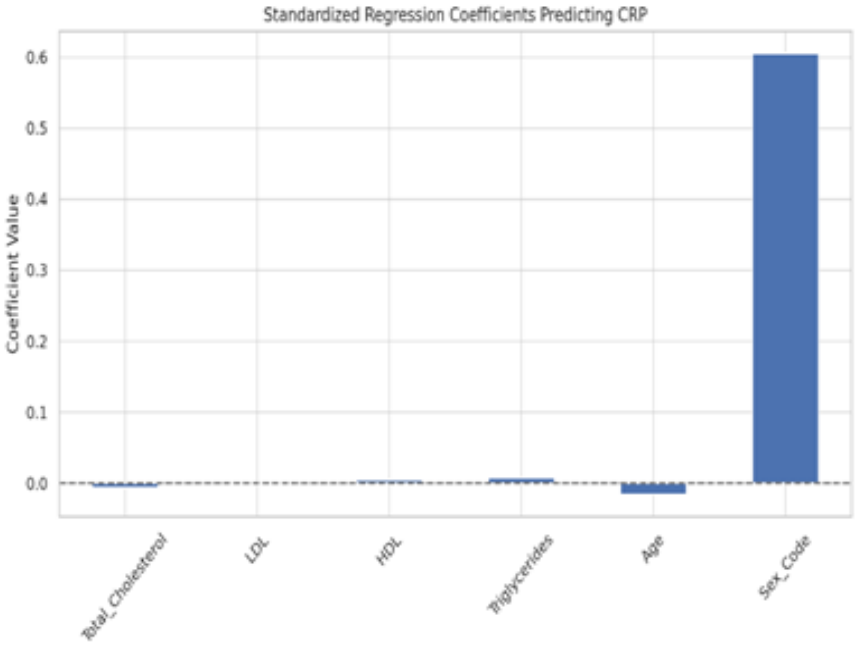


Figure 1. Standardized Regression Coefficients Predicting CRP

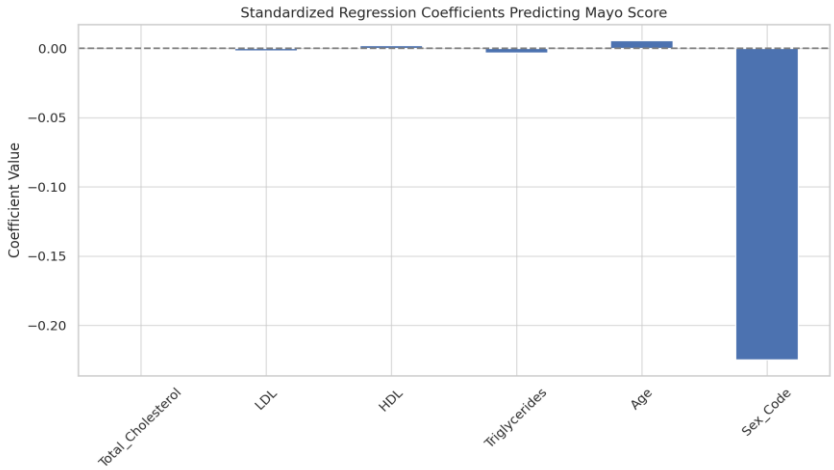


Figure 2. Standardized Regression Coefficients Predicting Mayo Score

5. ROC Curve Analysis

To evaluate the ability of lipid profiles to discriminate between active disease and remission in patients with ulcerative colitis (UC), a multivariable logistic regression model was constructed using total cholesterol, LDL, HDL, and triglycerides as predictors. The resulting model exhibited modest discriminatory performance.

The area under the receiver operating characteristic (ROC) curve (AUC) was 0.58, indicating that lipid biomarkers provide limited capacity to differentiate between active and remissive disease states. Figure 3 displays the ROC curve and corresponding diagnostic performance.

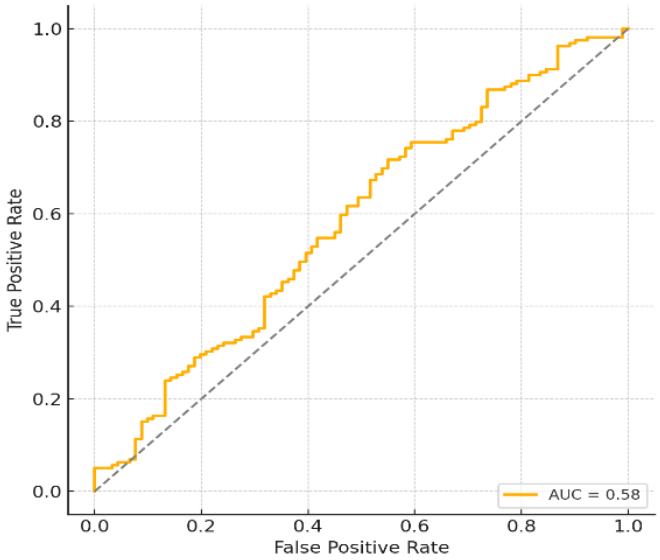


Figure 3. ROC curve for lipid-based prediction of disease activity in UC patients. AUC = 0.58.

DISCUSSION

This cross-sectional study aimed to evaluate the relationship between blood lipid profiles and disease activity in patients with ulcerative colitis (UC). Contrary to initial hypotheses and findings from prior literature, our results did not demonstrate significant differences in lipid markers—specifically total cholesterol (TC), low-density lipoprotein (LDL-C), high-density lipoprotein (HDL-C), or triglycerides (TGs)—between patients in active disease and those in remission. Additionally, no statistically significant correlations were observed between lipid components and either C-reactive protein (CRP) levels or Mayo scores, commonly used indicators of disease activity.

These findings differ from several earlier reports that suggested a link between systemic inflammation and altered lipid metabolism in inflammatory bowel disease (IBD). For example, studies such as those by Daniluk et al. [7] and Urbano et al. [8] have highlighted significant metabolic shifts, particularly involving HDL-C and TGs, in patients with active disease. However, our results suggest that such alterations may not be consistently observable across all UC populations, especially when assessed using conventional fasting lipid profiles.

Furthermore, our multivariable regression analysis did not identify any lipid parameter as an independent predictor of disease activity after adjusting for age, sex, and body mass index (BMI). These findings are in contrast to studies such as Chen et al. [14], which have suggested a potential role for HDL and TG levels as predictors of IBD activity. It is possible that differences in disease phenotype, population characteristics, or analytical methods contribute to these discrepancies.
The overall discriminative performance of the lipid profile as a diagnostic tool was modest, with the combined model yielding an area under the curve (AUC) of 0.58. This level of predictive ability is below the threshold generally considered clinically meaningful and aligns with the findings of Guan et al. [16], who demonstrated that individual lipid markers have limited utility as standalone diagnostic tools. Their results, and ours, support the idea that while lipid changes may accompany systemic inflammation, they are unlikely to serve as reliable biomarkers on their own.

Despite the lack of significant findings, the study contributes to the growing body of literature exploring the metabolic dimensions of UC. Lipidomics and advanced profiling approaches, as discussed by Tefas et al. [9] and Bazarganipour et al. [15], have shown promise in identifying lipid species such as sphingomyelins and ceramides that may modulate mucosal immunity. While our study did not employ these high-resolution techniques, our results reinforce the notion that standard lipid panels may be insufficiently sensitive to detect the subtle metabolic signatures of disease activity in UC.
In summary, this study did not find evidence to support significant differences or predictive value in fasting lipid profiles between active and remission phases of UC. While lipid metabolism remains an area of interest in UC pathophysiology, conventional lipid parameters appear to have limited utility as standalone biomarkers. Future research using more refined lipidomic methods and longitudinal designs may better clarify the role of lipid metabolism in disease activity and therapeutic response.

Limitations

This study has several limitations that warrant consideration. First, its cross-sectional design restricts causal inference between lipid alterations and disease activity in ulcerative colitis (UC). Longitudinal data would better clarify temporal relationships. Second, while the sample size was adequate for detecting moderate associations, it may have limited power to assess more nuanced subgroup effects or rare lipid abnormalities. Third, although adjustments were made for confounders such as age, sex, and BMI, unmeasured factors—including dietary intake, physical activity, and medication history—could influence lipid levels. Finally, our study was conducted in a single-centre tertiary care setting, which may affect the generalizability of findings to broader UC populations with differing disease severities or healthcare access.

CONCLUSION

In this study of patients with severe ulcerative colitis, conventional lipid profile components—including total cholesterol, LDL-C, HDL-C, and triglycerides—did not show significant differences between active and remission phases. Moreover, these parameters demonstrated no significant correlation with inflammatory markers such as C-reactive protein or with clinical disease activity scores. The overall predictive performance of lipid markers for distinguishing disease activity was modest, with limited standalone diagnostic utility.

While previous research has highlighted potential links between lipid metabolism and inflammatory bowel disease, our findings suggest that standard fasting lipid profiles may not serve as reliable biomarkers for disease monitoring in clinical practice. Further studies employing lipidomic analyses or integrating lipid data with other inflammatory and clinical indices may provide deeper insights into the metabolic landscape of ulcerative colitis and help identify more precise, multi-marker models for disease assessment.

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