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Role of Inflammatory Marker Hs CRP Serum Ferritin and Lipoprotein a in Type 2diabetes Mellitus

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KEYWORDS

Type 2 diabetes mellitus; High-sensitivity C-reactive protein; Serum ferritin; Lipoprotein(a); Dyslipidaemia; Inflammation; Cardiovascular risk

ABSTRACT:

Background: Type 2 diabetes mellitus (T2DM) is increasingly recognised as a chronic inflammatory disorder associated with dyslipidaemia and heightened cardiovascular risk. Inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP), serum ferritin, and lipoprotein(a) [Lp(a)] have been implicated in the pathophysiology of T2DM, but their interrelationship with lipid parameters remains incompletely understood.

Objectives: To evaluate the levels of hs-CRP, serum ferritin, and lipoprotein(a) in patients with type 2 diabetes mellitus and to assess their association with lipid profile parameters in comparison with healthy controls.

Materials and Methods: This case–control study included patients with T2DM (cases) and age- and sex-matched healthy individuals (controls). Anthropometric measurements, blood pressure, and biochemical parameters, including HbA1c, hs-CRP, serum ferritin, lipoprotein(a), and lipid profile, were assessed. Statistical analysis was performed using the Mann–Whitney U test for group comparisons and correlation analysis to evaluate associations between inflammatory markers and lipid parameters.

Results: Diabetic patients had significantly higher body weight, systolic and diastolic blood pressure, and HbA1c levels compared to controls ($p < 0.05$). Levels of hs-CRP, serum ferritin, and lipoprotein(a) were markedly elevated in cases ($p < 0.001$). Diabetic subjects also demonstrated significant dyslipidaemia with increased VLDL, LDL, triglycerides, and total cholesterol ($p < 0.001$). Correlation analysis showed a significant positive association between hs-CRP and LDL cholesterol, while serum ferritin exhibited a negative correlation with triglycerides. No significant correlation was observed among the inflammatory markers themselves.

Conclusion: Type 2 diabetes mellitus is characterized by elevated inflammatory markers and significant lipid abnormalities, reflecting an increased cardiovascular risk. Hs-CRP, serum ferritin, and lipoprotein(a) may serve as useful adjunctive biomarkers for assessing inflammation and atherogenic risk in patients with T2DM.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterised by chronic hyperglycaemia resulting from insulin resistance and relative insulin deficiency. The global burden of T2DM has increased dramatically over the past few decades, particularly in developing countries like India, where rapid urbanisation, sedentary lifestyles, and dietary transitions have contributed to rising prevalence rates [1]. Beyond hyperglycaemia, T2DM is increasingly recognised as a state of chronic low-grade inflammation, which plays a pivotal role in the development of insulin resistance and associated vascular complications [2].

Inflammation contributes significantly to endothelial dysfunction, atherosclerosis, and cardiovascular morbidity in patients with T2DM. High-sensitivity C-reactive protein (hs-CRP), an acute-phase reactant synthesized by the liver under the influence of pro-inflammatory cytokines such as interleukin-6, is a well-established marker of systemic inflammation. Elevated hs-CRP levels have been consistently associated with insulin resistance, poor glycaemic control, and increased cardiovascular risk in diabetic individuals [3,4].

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Serum ferritin, traditionally considered a marker of iron stores, has emerged as an important inflammatory biomarker. Ferritin levels tend to be elevated in chronic inflammatory states, including T2DM, and excess iron has been implicated in oxidative stress–mediated pancreatic β-cell dysfunction and impaired insulin action [5]. Several studies have demonstrated a positive association between elevated ferritin levels, insulin resistance, and metabolic syndrome components, suggesting its role in the pathophysiology of T2DM [6].

Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein particle structurally similar to low-density lipoprotein (LDL) but with an additional apolipoprotein(a) moiety. Elevated Lp(a) levels are considered an independent risk factor for atherosclerotic cardiovascular disease due to their pro-atherogenic, pro-thrombotic, and pro-inflammatory properties [7]. In patients with T2DM, altered lipid metabolism and chronic inflammation may further amplify the atherogenic potential of Lp(a), increasing cardiovascular risk [8].

Dyslipidaemia is a common metabolic abnormality in T2DM, typically characterized by elevated triglycerides, increased LDL, and reduced HDL levels. Inflammatory markers such as hs-CRP and ferritin have been shown to interact with lipid parameters, thereby accelerating atherosclerosis and cardiovascular complications in diabetic patients [9]. However, the interrelationships between inflammatory markers and lipid profile components remain inconsistent across different populations.

Despite growing evidence linking inflammation, iron metabolism, and lipid abnormalities with T2DM, data exploring the combined role of hs-CRP, serum ferritin, and lipoprotein(a) in Indian diabetic populations remain limited. Understanding the association of these inflammatory markers with lipid parameters may help in early risk stratification and prevention of cardiovascular complications in T2DM.

Therefore, the present study was undertaken to evaluate the role of inflammatory markers—hs-CRP, serum ferritin, and lipoprotein(a)—in patients with type 2 diabetes mellitus and to assess their relationship with lipid profile parameters in comparison with healthy controls.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based case–control study conducted in the Department of Biochemistry in collaboration with the Department of Medicine at a tertiary care teaching hospital. The study was carried out over a defined study period after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment.

Study Population

The study included two groups:

- Cases: Patients diagnosed with type 2 diabetes mellitus based on the American Diabetes Association (ADA) diagnostic criteria.
- Controls: Apparently healthy, age- and sex-matched individuals without a history of diabetes or other chronic systemic illness.

A total of 180 participants were enrolled, comprising 90 cases and 90 controls.

Inclusion Criteria

- Adults aged between 30 and 60 years
- Diagnosed cases of type 2 diabetes mellitus (for the case group)
- Apparently healthy individuals with normal fasting blood glucose and HbA1c (for the control group)

Exclusion Criteria

- Type 1 diabetes mellitus
- Acute or chronic inflammatory diseases
- Hepatic, renal, or thyroid disorders
- Cardiovascular disease, malignancy, or acute infections
- Pregnancy and lactation
- Patients on lipid-lowering drugs, iron therapy, or anti-inflammatory medications

Clinical and Anthropometric Assessment

Detailed clinical history was obtained from all participants. Anthropometric measurements including

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height and weight were recorded using standard techniques, and body weight was expressed in kilograms. Blood pressure was measured in the sitting position using a standard sphygmomanometer after adequate rest, and the average of two readings was taken for analysis.

Sample Collection

After an overnight fast of 10–12 hours, venous blood samples were collected under aseptic precautions. Samples were allowed to clot and centrifuged to separate serum, which was used for biochemical analysis. All parameters were analyzed on the same day or stored appropriately until analysis.

Biochemical Analysis

- HbA1c was measured using a standardized immunoturbidimetric method.
- Lipid profile, including total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, and VLDL, was estimated using enzymatic colorimetric methods.
- High-sensitivity C-reactive protein (hs-CRP) was measured by immunoturbidimetric assay.
- Serum ferritin was estimated using a chemiluminescence immunoassay method.
- Lipoprotein(a) levels were measured using an immunoturbidimetric method.

All biochemical estimations were performed on an automated analyzer following the manufacturer's instructions, with appropriate quality control measures in place.

Statistical Analysis

Data were entered and analyzed using standard statistical software. Continuous variables were expressed as mean ± standard deviation (SD) and median values where appropriate. Categorical variables were expressed as frequencies and percentages. The Chi-square test was used to compare categorical variables. The Mann–Whitney U test was applied for comparison of continuous variables between cases and controls. Correlation analysis between inflammatory markers and lipid parameters was performed using Pearson's correlation coefficient. A p value of < 0.05 was considered statistically significant.

RESULTS AND OBSERVATIONS;

Table 1 shows the gender-wise distribution of participants in the case and control groups. Among the cases, males constituted 62.2% (n = 56) and females 37.8% (n = 34). Similarly, in the control group, males accounted for 60% (n = 54) while females constituted 40% (n = 36).

The comparison of gender distribution between cases and controls was performed using the Chi-square test. The p value obtained was 0.76, which is greater than 0.05, indicating that there was no statistically significant difference in gender distribution between the two groups.

Table 1: Gender wise distribution in case a control group.

		Cases		Controls		
		Frequency	Percent (%)	Frequency	Percent (%)	p value
Sex	Male	56	62.2	54	60	0.76
	Female	34	37.8	36	40	
Chi square test						

Table 2 compares age, height, and weight between the case and control groups. The mean age of cases was 42.07 ± 6.35 years, while controls had a mean age of 43.27 ± 8.15 years, with no statistically significant difference between the groups (p = 0.323), indicating age comparability. Similarly, mean height was comparable between cases (163.45 ± 7.89 cm) and

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controls (163.06 ± 7.49 cm), and the difference was not statistically significant (p = 0.546). However, the mean body weight was significantly higher among cases (68.05 ± 10.54 kg) compared to controls (61.21 ± 9.85 kg), with this difference being statistically significant (p < 0.001). The analysis was performed using the Mann–Whitney U test.

Table 2: Comparison of Age, Height, and Weight between Cases and Controls.

Group	Cases			Controls			p value
	Mean	S.D.	Median	Mean	S.D.	Median	
Age	42.07	6.35	42	43.27	8.15	43	0.323
Hight	163.45	7.89	165	163.06	7.49	163	0.546
Weight	68.05	10.54	68	61.21	9.85	63	<0.001
p value is calculated using Mann whitney U test.							

Table 3 compares systolic blood pressure, diastolic blood pressure, and HbA1c levels between the case and control groups. The mean systolic blood pressure was significantly higher in cases (135.89 ± 13.26 mmHg) compared to controls (123.28 ± 8.70 mmHg), and this difference was statistically significant (p < 0.001). Similarly, the mean diastolic blood pressure was higher among cases (82.38 ± 9.76 mmHg) than controls (78.48 ± 5.51 mmHg), with the difference reaching statistical significance (p = 0.004). Glycaemic status, assessed by HbA1c levels, was markedly elevated in the case group (8.91 ± 1.57 mmol) compared to the control group (4.67 ± 0.44 mmol), and this difference was highly significant (p < 0.001).

Table 3: Comparison of Systolic Blood Pressure, Diastolic Blood Pressure, and HbA1c Levels between Cases and Controls.

Group	Cases			Controls			p value
	Mean	S.D.	Median	Mean	S.D.	Median	
Systolic	135.89	13.26	132	123.28	8.7	122	<0.001
Diastolic	82.38	9.76	82	78.48	5.51	80	0.004
HbA1c (mmol	8.91	1.57	9	4.67	0.44	5	<0.001
p value is calculated using Mann whitney U test.							

Table 4 compares Lipoprotein(a), high-sensitivity C-reactive protein (hs-CRP), and serum ferritin levels between the case and control groups. The mean Lipoprotein(a) level was significantly higher in cases (21.33 ± 5.71) compared to controls (74.14 ± 21.88), and this difference was statistically significant (p < 0.001). Similarly, cases demonstrated markedly elevated hs-CRP levels (9.34 ± 2.13 mg) in comparison to controls (1.25 ± 0.54 mg), indicating a significantly higher inflammatory status among cases (p < 0.001). Serum ferritin levels were also substantially increased in the case group (309.37 ± 32.72 ng) compared to the control group (67.93 ± 14.68 ng), with the difference being highly statistically significant (p < 0.001). The Mann–Whitney U test was used for analysis, suggesting that cases had significantly higher inflammatory and iron-storage markers than controls, reflecting increased systemic inflammation in the case group.

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Table 4: Comparison of Lipoprotein(a), High-Sensitivity C-Reactive Protein, and Serum Ferritin Levels between Cases and Controls.

Group	Cases			Controls			p value
	Mean	S.D.	Median	Mean	S.D.	Median	
Lipoprotein A	21.33	5.71	22	74.14	21.88	76	<0.001
hsCRP (mg)	9.34	2.13	9	1.25	0.54	1	<0.001
Ferretin (ng)	309.37	32.72	321	67.93	14.68	69	<0.001
p value is calculated using Mann whitney U test.							

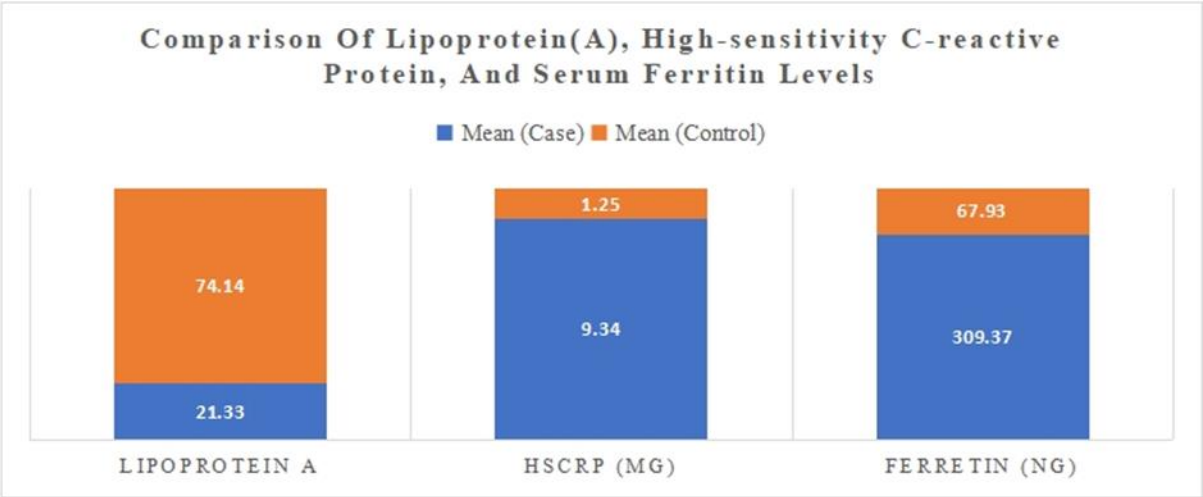

Table 5 compares the lipid profile parameters between the case and control groups. The mean levels of very low-density lipoprotein (VLDL) were significantly higher in cases (52.56 ± 7.65) compared to controls (22.29 ± 7.97), with the difference being statistically significant ($p < 0.001$). Similarly, low-density lipoprotein (LDL) levels were markedly elevated among cases (147.04 ± 7.68) in comparison to controls (121.09 ± 8.78), showing a highly significant difference ($p < 0.001$). Triglyceride levels were also significantly higher in the case group (162.47 ± 7.55) than in the control group (136.71 ± 8.89) ($p < 0.001$). Total cholesterol levels followed a similar pattern, with cases demonstrating higher mean values (199.19 ± 6.11) compared to controls (179.19 ± 11.69), and this difference was statistically significant ($p < 0.001$). Additionally, high-density lipoprotein (HDL) levels differed significantly between the two groups, with cases showing higher mean values (82.19 ± 9.80) compared to controls (49.30 ± 9.02) ($p < 0.001$). The analysis was performed using the Mann–Whitney U test, indicating a significant alteration in lipid profile parameters among cases when compared to controls, suggestive of an associated dyslipidaemic pattern in the case group.

Table 5: Comparison of Lipid Profile Parameters between Cases and Controls.

Group	Cases			Controls			p value
	Mean	S.D.	Median	Mean	S.D.	Median	
VLDL	52.56	7.65	52	22.29	7.97	22	<0.001

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LDL	**147.04**	**7.68**	**147**	**121.09**	**8.78**	**123**	**<0.001**
Trigiceride	**162.47**	**7.55**	**162**	**136.71**	**8.89**	**138**	**<0.001**
Cholesterol	**199.19**	**6.11**	**199**	**179.19**	**11.69**	**179**	**<0.001**
HDL	**82.19**	**9.8**	**82**	**49.3**	**9.02**	**50**	**<0.001**
p value is calculated using Mann whitney U test.							

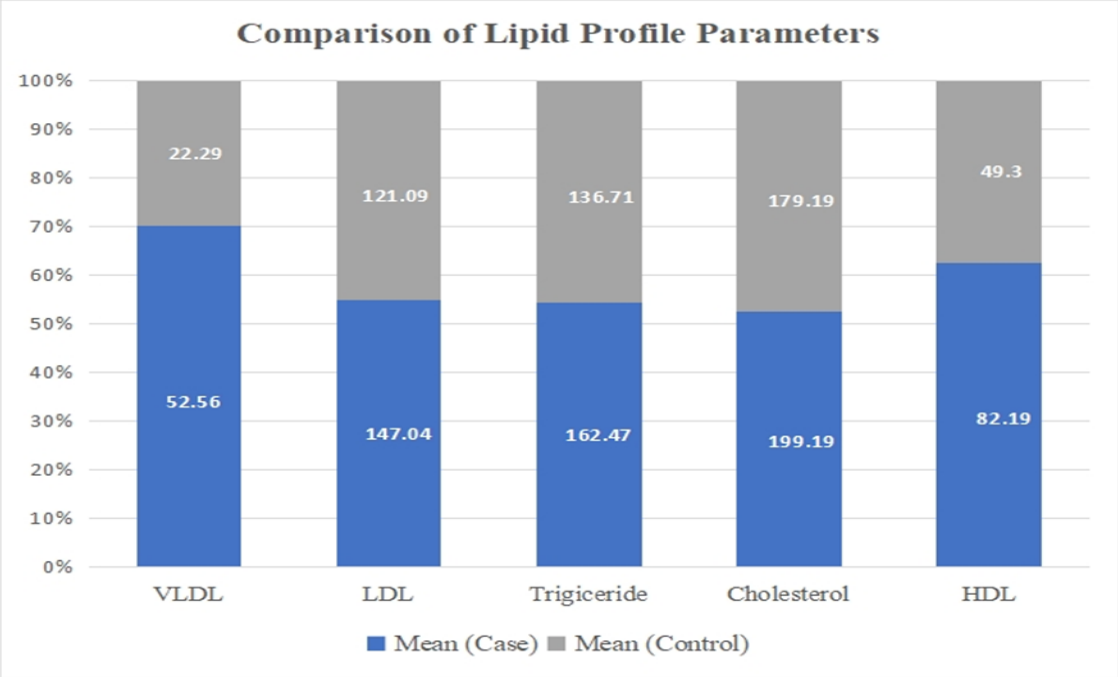

Table 6 presents the correlation analysis between Lipoprotein(a), high-sensitivity C-reactive protein (hs-CRP), and serum ferritin levels among cases. Lipoprotein(a) showed a weak positive correlation with hs-CRP ($r = 0.063$) and a weak negative correlation with serum ferritin ($r = -0.021$); however, both associations were not statistically significant ($p = 0.555$ and $p = 0.847$, respectively). Similarly, hs-CRP demonstrated a very weak negative correlation with serum ferritin ($r = -0.012$), which was also not statistically significant ($p = 0.908$). Overall, the findings indicate that there was no significant correlation between Lipoprotein(a), hs-CRP, and serum ferritin levels in the case group, suggesting that these biomarkers varied independently of each other in the studied population.

Table 6: Correlation Analysis of Lipoprotein(a), hs-CRP, and Serum Ferritin in Cases

Cases		Lipoprotein A	HsCRP (mg	Ferretin ng
Lipoprotein A	**Correlation cof.**		**0.063**	**-0.021**
	p value		**0.555**	**0.847**
HsCRP (mg)	**Correlation cof.**	**0.063**		**-0.012**
	p value	**0.555**		**0.908**

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	Correlation cof.	**-0.021**	**-0.012**	
Ferretin (ng)	**p value**	**0.847**	**0.908**	

Table 7 presents the correlation analysis between Lipoprotein(a), high-sensitivity C-reactive protein (hs-CRP), and serum ferritin levels among the control group. The correlation coefficients indicate the strength and direction of the linear relationship between the variables, while the associated p-values show whether these correlations are statistically significant. In the controls, Lipoprotein (a) did not show any significant correlation with either hs-CRP ($r = -0.027$, $p = 0.803$) or serum ferritin ($r = 0.016$, $p = 0.878$). hs-CRP exhibited a significant negative correlation with serum ferritin ($r = -0.343$, $p = 0.001$), suggesting that higher hs-CRP levels were associated with lower ferritin levels in this group. Overall, except for the hs-CRP and ferritin relationship, the other correlations were weak and not statistically significant, indicating minimal association among these biomarkers in the control population.

Table 7: Correlation between Lipoprotein(a), High-Sensitivity C-Reactive Protein, and Serum Ferritin Levels among Controls.

Control		**Lipoprotein A**	**HsCRP (mg**	**Ferretin ng**
Lipoprotein A	**Correlation cof**		**-0.027**	**0.016**
	p value		**0.803**	**0.878**
HsCRP (mg	**Correlation cof**	**-0.027**		**-0.343**
	p value	**0.803**		**0.001**
Ferretin ng	**Correlation cof**	**0.016**	**-0.343**	
	p value	**0.878**	**0.001**	

Table 8 shows the correlation between inflammatory markers (Lipoprotein(a), high-sensitivity C-reactive protein [hs-CRP], and serum ferritin) and lipid profile parameters (VLDL, LDL, triglycerides, cholesterol, and HDL) in the cases. Lipoprotein(a) did not show any significant correlation with any of the lipid parameters, indicating minimal association in this group. Hs-CRP exhibited a significant positive correlation with LDL ($r = 0.256$, $p = 0.015$), suggesting that higher levels of systemic inflammation were associated with elevated LDL levels. Serum ferritin showed a significant negative correlation with triglycerides ($r = -0.225$, $p = 0.033$), indicating that higher ferritin levels were linked to lower triglyceride levels in cases.

Table 8: Correlation between Inflammatory Markers and Lipid Profile Parameters among Cases.

Cases		**VLDL**	**LDL**	**Trigiceride**	**Cholesterol**	**HDL**
Lipoprotein A	**Correlation Coefficient**	**0.018**	**0.084**	**0.042**	**-0.029**	**0.022**
	p-value	**0.866**	**0.429**	**0.693**	**0.785**	**0.84**
HsCRP (mg	**L**	**0.003**	**.256***	**0.175**	**-0.002**	**-0.019**
	p-value	**0.98**	**0.015**	**0.099**	**0.988**	**0.857**

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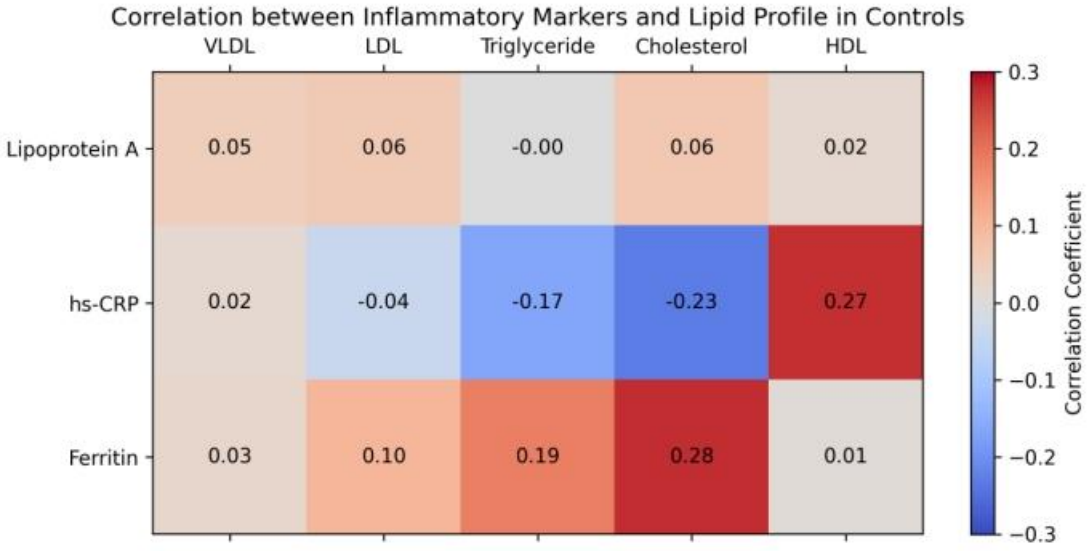
Ferretin ng	**dl**	**-0.09**	**-0.095**	**-.225***	**-0.103**	**-0.022**
	p-value	**0.401**	**0.374**	**0.033**	**0.333**	**0.838**

Table 9 presents the correlation between inflammatory markers (Lipoprotein(a), high-sensitivity C-reactive protein [hs-CRP], and serum ferritin) and lipid profile parameters (VLDL, LDL, triglycerides, cholesterol, and HDL) among the control group. Lipoprotein(a) did not show any significant correlation with any of the lipid parameters, indicating no meaningful association in controls. Hs-CRP demonstrated a significant negative correlation with cholesterol (r = -0.232, p = 0.028) and a significant positive correlation with HDL (r = 0.272, p = 0.009), suggesting that higher systemic inflammation may be associated with lower cholesterol but higher HDL levels in this group. Serum ferritin showed a significant positive correlation with cholesterol (r = 0.275, p = 0.009), indicating that higher ferritin levels were associated with higher cholesterol. All other correlations were weak and statistically non-significant, suggesting limited interaction between these inflammatory markers and lipid parameters in the control population.

Table 9: Correlation between Inflammatory Markers and Lipid Profile Parameters among Controls.

Control		**VLDL**	**LDL**	**Trigiceride**	**Cholesterol**	**HDL**
Lipoprotein A	**Correlation Coefficient**	**0.05**	**0.059**	**-0.001**	**0.064**	**0.019**
	p-value	**0.639**	**0.58**	**0.991**	**0.547**	**0.857**
HsCRP (mg	**L**	**0.018**	**-0.042**	**-0.165**	**-0.232**	**0.272**
	p-value	**0.868**	**0.695**	**0.121**	**0.028**	**0.009**
Ferretin ng	**Dl**	**0.026**	**0.102**	**0.187**	**0.275**	**0.009**
	p-value	**0.805**	**0.338**	**0.077**	**0.009**	**0.932**



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DISCUSSION

Type 2 diabetes mellitus (T2DM) is increasingly recognized as a chronic inflammatory and metabolic disorder rather than a condition limited to hyperglycaemia alone. The present study evaluated the role of inflammatory markers—high-sensitivity C-reactive protein (hs-CRP), serum ferritin, and lipoprotein(a)—and their association with lipid profile parameters in patients with T2DM compared to healthy controls. The findings provide important insights into the inflammatory milieu and dyslipidaemia associated with T2DM.

In the present study, cases and controls were comparable with respect to age, sex, and height, eliminating major demographic confounders. However, body weight was significantly higher among diabetic patients, consistent with the well-established association between increased adiposity and insulin resistance. Excess adipose tissue contributes to chronic low-grade inflammation through the secretion of pro-inflammatory cytokines, thereby exacerbating metabolic derangements in T2DM [1].

Blood pressure parameters were significantly elevated in diabetic patients, indicating a higher prevalence of hypertension in this group. This observation aligns with previous studies reporting coexistence of hypertension and diabetes as part of the metabolic syndrome, increasing cardiovascular risk [2]. Additionally, HbA1c levels were markedly higher in cases, confirming poor glycaemic control and validating the study population as true diabetic subjects.

A key finding of the present study was the significantly elevated hs-CRP levels among diabetic patients compared to controls. This supports the concept that T2DM is a state of chronic systemic inflammation. Elevated hs-CRP reflects activation of the innate immune system and has been strongly linked with insulin resistance, endothelial dysfunction, and atherosclerosis [3,4]. The observed elevation of hs-CRP in cases reinforces its role as an inflammatory biomarker and a potential predictor of cardiovascular complications in T2DM.

Serum ferritin levels were also significantly higher in the diabetic group. Ferritin, apart from being an indicator of iron stores, acts as an acute-phase reactant and rises in inflammatory conditions. Elevated ferritin may contribute to oxidative stress through iron-mediated free radical generation, leading to pancreatic β-cell damage and worsening insulin resistance [5]. Several studies have demonstrated a positive association between increased ferritin levels and T2DM, supporting the findings of the present study [6,7].

Lipoprotein(a) levels differed significantly between cases and controls, highlighting its potential role in diabetic dyslipidaemia and cardiovascular risk. Lipoprotein(a) possesses pro-atherogenic and pro-thrombotic properties and has been identified as an independent cardiovascular risk factor [8]. Alterations in lipid metabolism and inflammatory status in T2DM may influence Lp(a) levels, thereby increasing susceptibility to atherosclerotic complications.

The lipid profile analysis revealed significantly higher levels of VLDL, LDL, triglycerides, total cholesterol, and HDL in diabetic patients. These findings indicate a pronounced dyslipidaemic pattern in T2DM. Dyslipidaemia in diabetes is primarily driven by insulin resistance, leading to increased hepatic lipoprotein production and impaired lipid clearance [9]. Such lipid abnormalities significantly contribute to accelerated atherosclerosis in diabetic individuals.

Correlation analysis among inflammatory markers in the case group revealed no significant association between hs-CRP, serum ferritin, and lipoprotein(a), suggesting that these biomarkers may act through independent pathological pathways in T2DM. Similar observations have been reported by previous studies, indicating that inflammatory and iron-related markers may reflect distinct aspects of metabolic dysfunction [10].

When evaluating the relationship between inflammatory markers and lipid parameters in cases, hs-CRP showed a significant positive correlation with LDL cholesterol, emphasizing the interplay between inflammation and atherogenic lipids. This finding supports the hypothesis that systemic inflammation may exacerbate lipid abnormalities, thereby promoting cardiovascular risk in T2DM [11]. Interestingly, serum ferritin demonstrated a significant negative correlation with triglycerides, a finding that may reflect complex regulatory mechanisms of iron metabolism and lipid homeostasis, warranting further investigation.

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In the control group, hs-CRP exhibited a significant negative correlation with ferritin and cholesterol, while ferritin showed a positive association with cholesterol. These findings suggest that even in non-diabetic individuals, inflammatory and iron-related markers may influence lipid metabolism, although the associations appear weaker than those observed in diabetic patients.

Overall, the present study highlights the significant elevation of inflammatory markers and lipid abnormalities in T2DM patients. The lack of strong correlations among inflammatory markers suggests multifactorial mechanisms contributing to metabolic and cardiovascular risk. These findings underscore the importance of incorporating inflammatory and iron-related biomarkers into the comprehensive assessment of patients with T2DM.

CONCLUSION

Type 2 diabetes mellitus is associated with significantly elevated inflammatory markers and adverse lipid profile alterations. Increased levels of hs-CRP, serum ferritin, and lipoprotein(a) in diabetic patients reflect underlying chronic inflammation and heightened cardiovascular risk. These markers appear to act independently, with hs-CRP showing a significant association with atherogenic lipids. Incorporating inflammatory biomarkers into routine evaluation may improve risk stratification and management of patients with T2DM.

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