International Journal of Research in Medical Sciences
Varshney K et al. Int J Res Med Sci. 2026 Oct;14(10):xxx-xxx
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263379

Original Research Article

Evaluating the association between inflammatory markers and glycemic control status in type 2 diabetes mellitus patients: a tertiary care centre experience

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Received: 09 July 2026
Revised: 17 September 2026
Accepted: 18 September 2026

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ABSTRACT

Background: Type 2 diabetes mellitus is a chronic, multifactorial metabolic disease in which systemic inflammation plays a key role in the pathogenesis of insulin resistance. This study aimed to compare serum concentrations of ferritin, hs-CRP, IL-6, TNF-α, and IFN-γ between type 2 diabetes mellitus patients and matched healthy controls, and to determine their correlation with glycated haemoglobin (HbA1c) and fasting insulin levels.
Methods: A case-control study was conducted comprising 50 patients with type 2 diabetes mellitus and 50 age- and sex-matched healthy controls under fasting conditions. Serum levels of HbA1c, fasting insulin, ferritin, hs-CRP, IL-6, TNF-α, and IFN-γ were measured. Intergroup comparisons were evaluated using the Mann–Whitney U test, and correlations with HbA1c and insulin were assessed using Spearman's rank correlation.
Results: Serum levels of ferritin, hs-CRP, IL-6, and TNF-α were significantly higher in type 2 diabetic patients than in healthy controls ($p<0.001$), whereas IFN-γ levels showed no significant intergroup difference ($p=0.515$). Serum ferritin demonstrated a statistically significant positive correlation with both HbA1c (rho=0.38, p=0.007) and fasting insulin (rho=0.33, p=0.021). In contrast, serum hs-CRP (rho=0.13, p=0.368 with HbA1c; rho=0.19, p=0.193 with insulin), IL-6 (rho=-0.01, p=0.952 with HbA1c; rho=-0.11, p=0.462 with insulin), TNF-α (rho=0.11, p=0.448 with HbA1c; rho=0.13, p=0.364 with insulin), and IFN-γ (rho=0.20, p=0.154 with HbA1c; rho=0.09, p=0.538 with insulin) showed no significant correlation with either HbA1c or fasting insulin levels ($p>0.05$).
Conclusions: Chronic systemic inflammation is pronounced in type 2 diabetes mellitus. Among the evaluated inflammatory biomarkers, serum ferritin uniquely correlates with both glycemic control and circulating insulin levels, highlighting its practical clinical utility in monitoring subclinical metabolic decompensation, while acute-phase cytokines reflect tonic inflammatory activation rather than direct glycemic fluctuations.

Keywords: Diabetes mellitus, IL-6, IFN-γ, TNF-α, Ferritin, hs-CRP, HbA1c

INTRODUCTION

Type 2 diabetes mellitus is a heterogeneous metabolic disease characterized by peripheral insulin resistance and impaired insulin secretion from pancreatic β-cells.[1] According to estimates from the International Diabetes Federation, approximately 463 million adults aged 20–79 years were living with diabetes globally in 2019, with figures projected to exceed 700 million by 2045.[2] The vast majority of these patients have type 2 diabetes mellitus, the prevalence of which continues to rise significantly.

Type 2 diabetes mellitus represents a leading contributor to global morbidity and mortality. It is characterized by chronic low-grade systemic inflammation linked to microvascular and macrovascular complications, which cause multiorgan damage predominantly affecting the eyes, kidneys, heart, and vasculature.[1]

Systemic inflammation plays a central pathogenic role in mediating insulin resistance and disease progression in type 2 diabetes mellitus. Circulating inflammatory biomarkers may therefore serve as practical tools for risk stratification and monitoring long-term complications. Previous studies have reported elevated concentrations of various pro-inflammatory mediators and acute-phase reactants, including ferritin, high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ).[3-5] However, establishing which specific markers best correlate with ongoing glycemic instability and insulin levels remains an area requiring clear clinical definition.

Chronic inflammation also contributes to key features of the metabolic syndrome, including dyslipidemia and impaired glucose regulation, which collectively establish the substrate for atherogenesis and progressive insulin resistance.[6] Numerous investigations highlight the clinical utility of hs-CRP as an independent predictor of adverse cardiovascular outcomes in both diabetic and nondiabetic cohorts.[7]

The present study was designed to evaluate and compare serum levels of ferritin, hs-CRP, IL-6, TNF-α, and IFN-γ between type 2 diabetes mellitus patients and healthy matched controls. Furthermore, we assessed how these inflammatory parameters correlate with glycemic control (HbA1c) and serum insulin levels to clarify their relationship with subclinical disease progression.

Aims and objectives

Aim of this study was to compare the serum levels of ferritin, hs-CRP, IL- 6, TNF-α and IFN-γ in type 2 diabetes mellitus and in healthy controls. Also to find the correlation of inflammatory markers (ferritin, hs-CRP, IL-6, TNF-α, IFN-γ) with HbA1c and serum insulin levels in type 2 diabetes mellitus cases.

METHODS

A case control study was conducted in the Departments of Pathology and Medicine in Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, over a period of 18 months (December 2020 to May 2022) after obtaining clearance from Institute Ethics Committee. Informed written consent was obtained from all participants prior to enrollment. The study comprised 50 clinically confirmed cases of type 2 diabetes mellitus and 50 age- and sex-matched healthy controls.

Inclusion criteria for cases were patients aged >18 years diagnosed with type 2 diabetes mellitus according to the American Diabetes Association (ADA) 2020 criteria.[8] The control group comprised healthy volunteers with normal glycemic parameters and no personal history of dysglycemia. Exclusion criteria for both groups included type 1 diabetes mellitus, treatment with insulin therapy or oral hypoglycemic agents, iron deficiency anemia, active or chronic systemic diseases, acute coronary syndrome, recent myocardial infarction, or any active or recent infectious illness.

Venous blood samples were obtained from all participants under fasting conditions after an overnight fast of 8–12 hours. A 2 ml sample was collected in an EDTA vial for HbA1c estimation. An additional 5 ml of blood was collected into two plain vacutainers for serum separation (centrifuged at 3000 rpm for 10 minutes) to evaluate fasting insulin, ferritin, hs-CRP, IL-6, TNF-α, and IFN-γ levels.

HbA1c was quantified using the Hemo One analyzer. Serum hs-CRP was measured on a Biocell Medicare BNII nephelometer. Serum ferritin and fasting insulin concentrations were measured via chemiluminescence immunoassay on the ADVIA Centaur CP system. Serum concentrations of IL-6, TNF-α, and IFN-γ were quantified using commercial enzyme-linked immunosorbent assay (ELISA) kits in accordance with the manufacturer's protocol.

Normal reference cutoff ranges were defined as follows: HbA1c <6.5%, fasting insulin <25 mIU/l, ferritin 15–250 ng/ml for males and 10–125 ng/ml for females, hs-CRP <3 mg/l, IL-6 <5–15 pg/ml, TNF-α <8 pg/ml, and IFN-γ <5.5 pg/ml.

Data analysis was performed using IBM statistical package for the social sciences (SPSS) statistics version 21.0. Continuous variables were evaluated for distributional normality using the Shapiro–Wilk test. Parametric variables are presented as mean±standard deviation (SD), and non-normally distributed variables are expressed as median and interquartile range (IQR). Group comparisons for non-normally distributed parameters were performed using the Mann-Whitney U test (Wilcoxon rank-sum test). Correlation analyses between inflammatory markers, HbA1c, and fasting insulin were evaluated using Spearman's rank correlation coefficient. A two-tailed p value ≤0.05 was considered statistically significant.

RESULTS

Statistical analysis was done in SPSS licensed version 21.0. Mean and standard deviation was calculated. For quantitative non-normally distributed data, Mann-Whitney test was applied. P value ≤0.05 was considered as a level of statistical significance.

Baseline demographics

A total of 100 participants were enrolled in the study, comprising 50 type 2 diabetes mellitus cases and 50 age- and sex-matched healthy controls (Figure 1). The mean (SD) age was 45.34 (8.26) years (range: 28–62 years) in the diabetic cohort and 43.52 (12.86) years (range: 19–75 years) in the control group (Figure 2). Both groups had

identical sex distributions, with 22 males (44.0%) and 28 females (56.0%) in each cohort (Figure 3). There were no statistically significant differences between the two groups regarding age (t=0.842, p=0.402) or sex (χ^2=0.000, p=1.000), confirming successful matching.

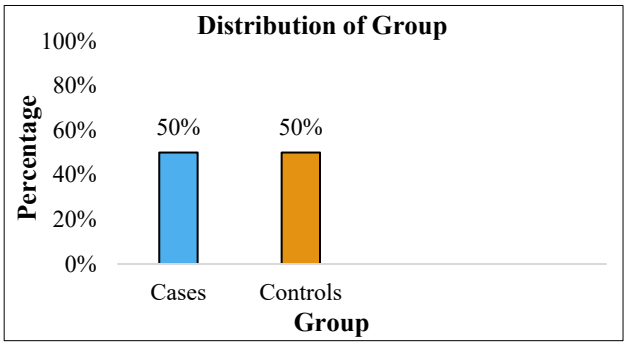

Figure 1: Bar graph depicts the distribution of participants in terms of group.

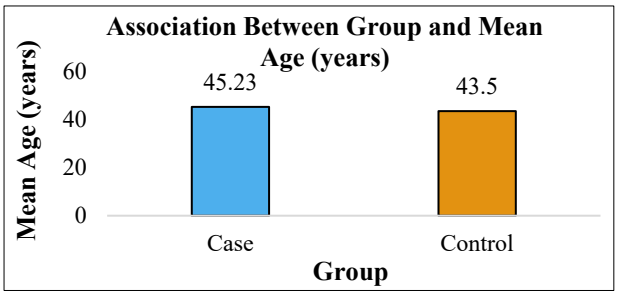

Figure 2: Bar graph depicts comparison of the cases and controls in terms of mean age (years).

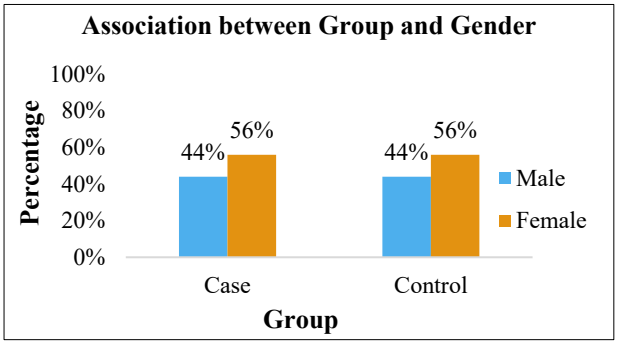

Figure 3: Bar graph depicts comparison of the cases and controls in terms of gender.

Comparison of inflammatory biomarkers between cases and controls

Because circulating inflammatory markers exhibited non-normal distributions, the Mann–Whitney U test was used for intergroup comparisons. Serum levels of ferritin, hs-CRP, IL-6, and TNF-α were significantly elevated in type 2 diabetes mellitus patients compared to healthy controls (p<0.001), whereas IFN-γ levels did not differ significantly between the groups (p=0.515) (Table 1 and Figure 4).

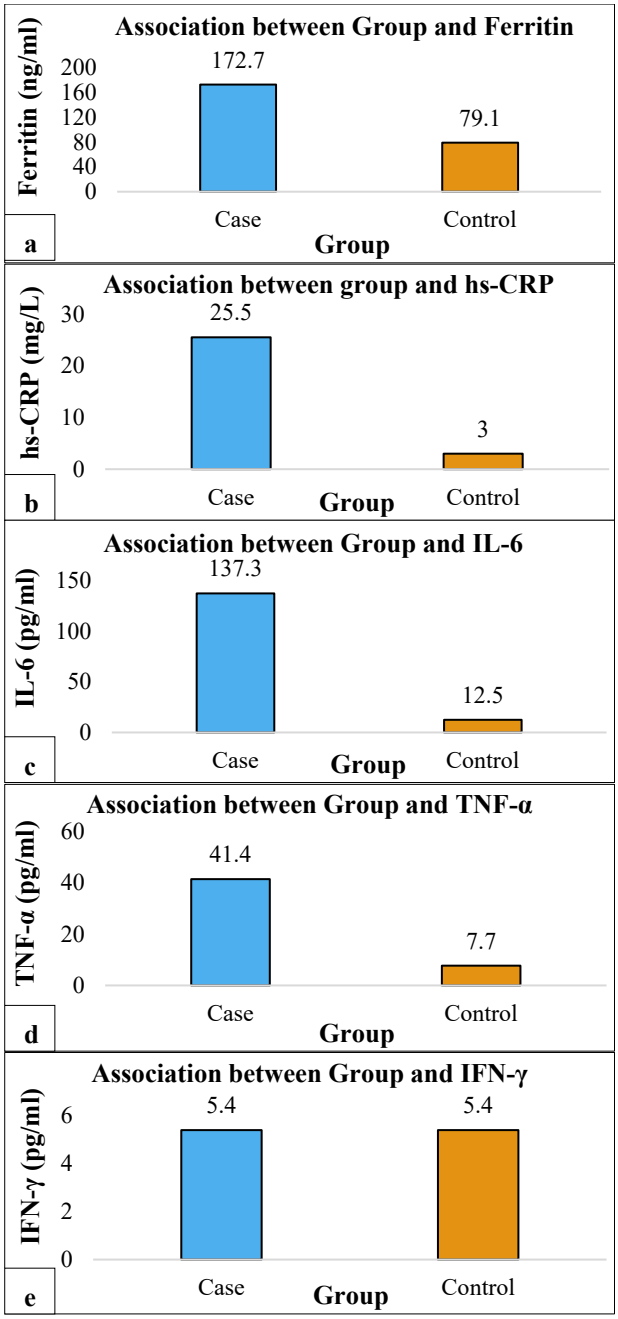

Figure 4: Comparison of the cases and controls in terms of inflammatory markers (ferritin, hs-CRP, IL-6, TNF-α, IFN-γ) (n=100). The bar graph depicts the means of (a) ferritin, (b) hs-CRP, (c) IL- 6, (d) TNF-α, and (e) IFN-γ in the cases and controls.

Ferritin

Median (IQR) ferritin in the diabetic cohort was 131.85 (104.45–223.20) ng/ml [mean±SD: 172.65±145.63; range: 30.9–933.3] compared to 71.25 (39.17–106.43) ng/ml [mean±SD: 79.11±57.11; range: 6.2–229.5] in controls (W=1959.500, p<0.001) (Table 1 and Figure 4a). Strength of association (point-biserial correlation) =0.39 (large effect size).

hs-CRP

Median (IQR) hs-CRP was 14.37 (6.21–30.18) mg/l [mean±SD: 25.55±31.71; range: 0.81–170.0] in cases versus 1.97 (1.07–3.10) mg/l [mean ± SD: 3.00 ± 4.36; range: 0.12–27.3] in controls (W=2225.000, p<0.001) (Table 1 and Figure 4b). Strength of association (point-biserial correlation) =0.45 (large effect size).

IL-6

Median (IQR) IL-6 was 68.75 (24.00–153.80) pg/ml [mean±SD: 137.28±193.33; range: 5.3–850.9] in cases compared to 10.05 (5.60–14.80) pg/ml [mean±SD: 12.48±9.99; range: 3.9–59.2] in controls (W=2195.500, p<0.001) (Table 1 and Figure 4c). Strength of association (point-biserial correlation) =0.42 (large effect size).

TNF-α

Median (IQR) TNF-α was 16.45 (10.72–64.78) pg/ml [mean±SD: 41.38±47.92; range: 2.7–207.3] in cases versus 5.90 (4.35–7.17) pg/ml [mean±SD: 7.73±11.25; range: 1.4–79.1] in controls (W=2193.000, p<0.001) (Table 1 and Figure 4d). Strength of association (point-biserial correlation) =0.44 (large effect size).

IFN-γ

Median (IQR) IFN-γ levels were comparable between cases [4.60 (3.50–6.47) pg/ml; mean±SD: 5.36±2.91; range: 2.2–16.4] and controls [4.98 (3.70–6.99) pg/ml; mean±SD: 5.37±2.27; range: 2.6–12.9], showing no significant difference (W=1155.000, p=0.515) (Table 1 and Figure 4e). Strength of association (point-biserial correlation) =0 (insignificant/no association).

Correlation of inflammatory markers with HbA1c and fasting insulin

Spearman rank correlation was performed to determine associations between inflammatory parameters, HbA1c, and fasting insulin in patients with type 2 diabetes mellitus.

Correlation with HbA1c

Serum ferritin exhibited a statistically significant positive correlation with HbA1c (rho=0.38, p=0.007) (Table 2 and Figure 5a). In contrast, no significant correlations were observed between HbA1c and hs-CRP (rho=0.13, p=0.368), IL-6 (rho=-0.01, p=0.952), TNF-α (rho=0.11, p=0.448), or IFN-γ (rho=0.20, p=0.154) (Table 2 and Figure 5b-e).

Correlation with fasting insulin

Serum ferritin also demonstrated a significant positive correlation with fasting serum insulin (rho=0.33, p=0.021) (Table 3 and Figure 6a). No significant associations were identified between fasting insulin and hs-CRP (rho=0.19, p=0.193), IL-6 (rho=-0.11, p=0.462), TNF-α (rho=0.13, p=0.364), or IFN-γ (rho=0.09, p=0.538) (Table 3 and Figure 6b-e).

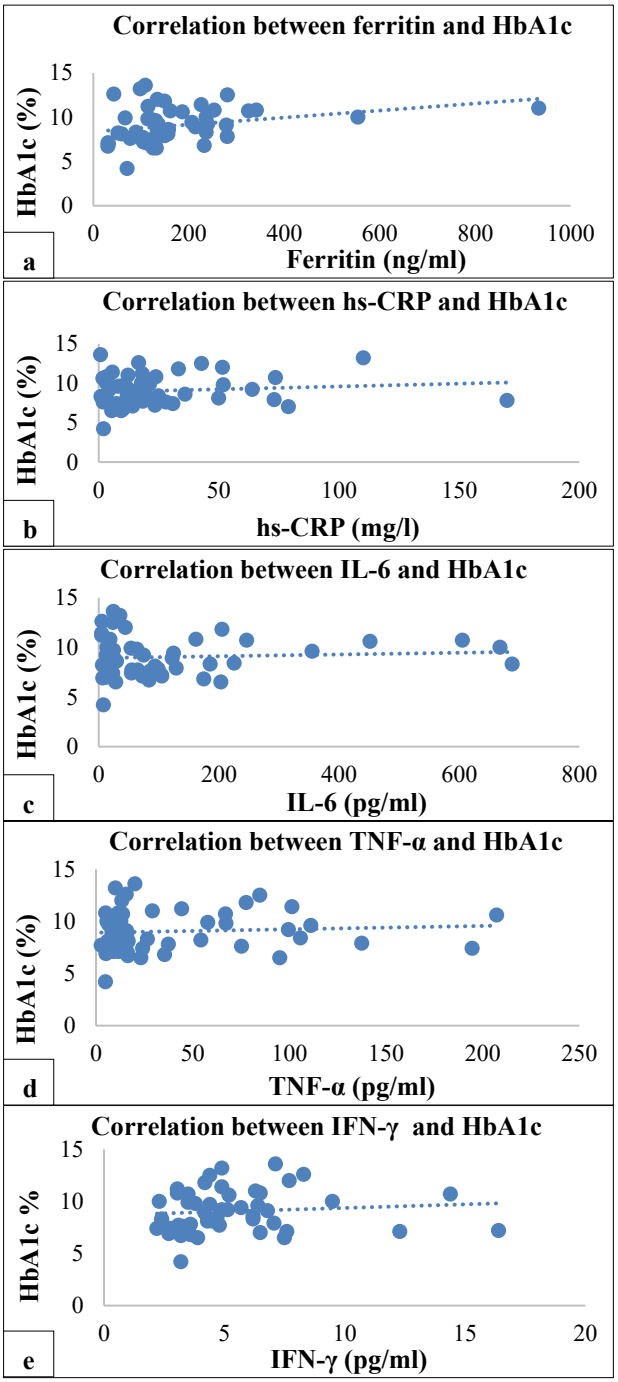

Figure 5: Correlation between inflammatory markers (ferritin, hs-CRP, IL-6, TNF-α, IFN-γ) and HbA1c (%) in type 2 diabetes mellitus (case) (n=50). The scatterplot depicts the correlation between inflammatory markers (a) ferritin, (b) hs-CRP, (c)IL-6, (d) TNF-α, and (e) IFN-γ and HbA1c (%).

The scatterplot in Figures 5 and 6 depicts the correlation between ferritin, hs-CRP, IL-6, TNF-α, IFN-γ with HbA1c and insulin. Individual points represent individual cases.

The blue trendline represents the general trend of correlation between the two variables. The shaded grey area represents the 95% confidence interval of this trendline.

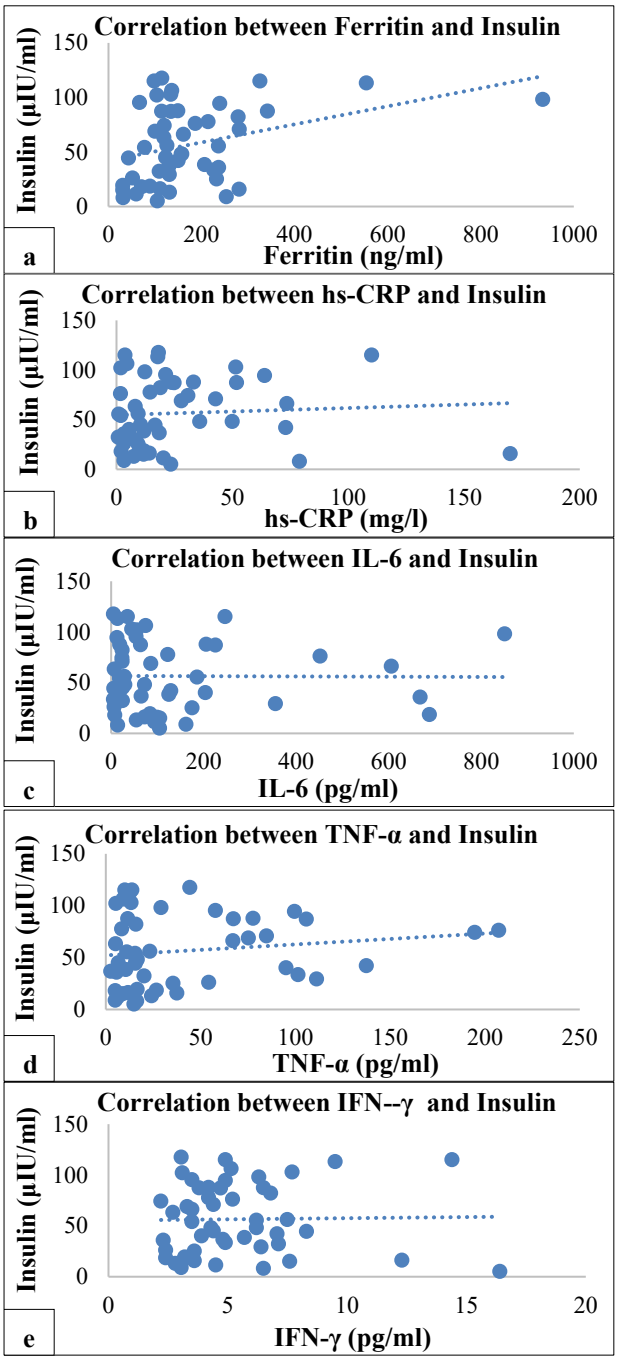

Figure 6: Correlation between inflammatory markers (ferritin, hs-CRP, IL-6, TNF-α, IFN-γ) and insulin (µIU/ml) in type 2 diabetes mellitus (Case) (n=50). The scatterplot depicts the correlation between inflammatory markers (a) ferritin, (b) hs-CRP, (c) IL-6, (d) TNF-α, and (e) IFN-γ and insulin (µIU/ml).

Hence, in this study we observed statistically significantly increased serum levels of ferritin, hs-CRP, IL-6 and TNF-α in type 2 diabetes patients compared to normal healthy individuals ($p<0.001$). Also, serum ferritin levels are positively correlated with serum HbA1c (rho=0.38, p=0.007) and insulin levels (rho=0.33, p=0.021) in type 2 diabetes patients. However, serum levels of hs-CRP, IL-6, TNF-α, and IFN-γ show no correlation with serum HbA1c and insulin levels in type 2 diabetes patients ($p>0.05$).

Table 1: Comparison of the cases and controls in terms of inflammatory markers (ferritin, hs-CRP, IL-6, TNF-α, IFN-γ).

Parameters	Group		P value
	Case (n=50)	Control (n=50)	
Ferritin (ng/ml)	172.65± 145.63	79.11± 57.11	<0.001
hs-CRP (mg/l)	25.55± 31.71	3.00± 4.36	<0.001
IL-6 (pg/ml)	137.28± 193.33	12.48± 9.99	<0.001
TNF-α (pg/ml)	41.38± 47.92	7.73± 11.25	<0.001
IFN-γ (pg/ml)	5.36± 2.91	5.37± 2.27	0.515

Table 2: Association between HbA1c (%) and parameters in type 2 diabetes mellitus (case) (n=50).

Parameters	Correlation coefficient (rho)	P value
Ferritin (ng/ml)	0.38	0.007
hs-CRP (mg/l)	0.13	0.368
IL-6 (pg/ml)	-0.01	0.952
TNF-α (pg/ml)	0.11	0.448
IFN-γ (pg/ml)	0.20	0.154

Table 3: Association between insulin (µIU/ml) and parameters in type 2 diabetes mellitus (case) (n=50).

Parameters	Correlation coefficient (rho)	P value
Ferritin (ng/ml)	0.33	0.021
hs-CRP (mg/l)	0.19	0.193
IL-6 (pg/ml)	-0.11	0.462
TNF-α (pg/ml)	0.13	0.364
IFN-γ (pg/ml)	0.09	0.538

DISCUSSION

Type 2 diabetes mellitus is characterized by impaired insulin secretion, peripheral insulin resistance, excessive hepatic glucose production, and abnormal lipid metabolism. While chronic low-grade systemic inflammation is recognized as a key driver of insulin resistance and diabetic angiopathy, the precise extent to which distinct circulating inflammatory biomarkers parallel glycemic control remains an important clinical question. In the present study, we demonstrated that

circulating concentrations of ferritin, hs-CRP, IL-6, and TNF-α were significantly elevated in type 2 diabetic patients compared to healthy controls (all $p<0.001$), whereas IFN-γ levels showed no significant difference ($p=0.515$). Furthermore, serum ferritin was the sole parameter among the evaluated panel that demonstrated a statistically significant positive correlation with both HbA1c ($\rho=0.38$, $p=0.007$) and fasting insulin levels ($\rho=0.33$, $p=0.021$).

Glycated hemoglobin monitoring in diabetic subjects reflects the integrated mean blood glucose level over the preceding 6 to 8 weeks, as the non-enzymatic glycation of hemoglobin is directly proportional to circulating ambient glucose concentrations.[1,9] A key clinical advantage of HbA1c measurement is its stability against daily glycemic fluctuations, physical exertion, and acute dietary intake, rendering it the benchmark for evaluating long-term glycemic regulation and complication risk. In physiological glucose homeostasis, an intricate balance exists between exogenous energy intake, endogenous hepatic glucose output, and peripheral uptake by insulin-sensitive tissues. Insulin serves as the central regulator of this metabolic equilibrium, acting in concert with glucagon, counter-regulatory hormones, and autonomic neural inputs. In the basal fasting state, low insulin concentrations coupled with appropriate glucagon tone stimulate hepatic glycogenolysis and gluconeogenesis while mobilizing gluconeogenic precursors, such as amino acids and free fatty acids, from muscle and adipose depots.[10] In type 2 diabetes, sustained low-grade inflammation disrupts this coordinated signaling, facilitating the development of dyslipidemia, impaired glucose tolerance, and accelerated atherogenesis.[6]

The inflammatory process and factors that contribute to chronic low-grade inflammation have become a central focus of cardiovascular and diabetic research. It is widely accepted that inflammation plays a major role in the pathogenesis and progression of atherosclerosis in persons with diabetes. A prominent feature of systemic inflammatory activity is the increase in circulating plasma concentrations of acute-phase proteins. hs-CRP provides an accurate measurement of CRP, a sensitive pentameric acute-phase reactant synthesized by hepatocytes in response to circulating pro-inflammatory stimuli, mainly IL-6 and TNF-α, with a plasma half-life of approximately 19 hours, serving as an established independent predictor of cardiovascular risk.[5,7]

Ferritin is the primary storage form of iron across tissues, sequestering approximately 1 g of total body iron under physiological conditions. Beyond its fundamental role in iron homeostasis, ferritin is an established positive acute-phase reactant whose transcription is upregulated in inflammatory states. At the cellular level, iron accumulation promotes reactive oxygen species generation via Fenton chemistry, causing oxidative damage to pancreatic β-cell membranes and worsening peripheral insulin resistance.

IL-6 is a multifunctional pro-inflammatory cytokine associated with chronic inflammation, autoimmune disorders, and metabolic derangements. Synthesized at sites of inflammation, IL-6 travels to the liver via the circulation, inducing the synthesis of major acute-phase proteins—including CRP, serum amyloid A, fibrinogen, and haptoglobin—while reducing hepatic production of fibronectin, albumin, and transferrin. In addition, IL-6 directly stimulates hepatic hepcidin production, which degrades ferroportin in the gut and reticuloendothelial macrophages, thereby decreasing serum iron availability and promoting intracellular iron retention.[11]

Similarly, TNF-α is a potent pro-inflammatory cytokine secreted by activated macrophages, monocytes, T-cells, smooth muscle cells, and adipocytes. Signaling via TNF receptor-1 (TNFR1) directly interferes with glucose homeostasis: it impairs the insulin receptor signaling pathway by stimulating inhibitor-κB kinase (IKK), which induces inhibitory serine phosphorylation of insulin receptor substrate-1 (IRS-1), thereby converting IRS-1 into an inhibitor of insulin receptor tyrosine kinase activity.

Furthermore, TNF-α stimulates lipolysis within adipose tissue, generating an excess flux of circulating free fatty acids that triggers peripheral insulin resistance, promotes hepatic gluconeogenesis, and indirectly disrupts insulin sensitivity by downregulating adiponectin and altering leptin action.[12,13]

Regarding interferon-gamma (IFN-γ), our findings revealed no statistically significant difference between diabetic cases and controls ($p=0.515$). IFN-γ plays a pivotal role in systemic inflammation and autoimmune pathology.[14] However, it also exerts counter-regulatory anti-inflammatory effects through the induction of IL-1 receptor antagonist (IL-1Ra) and IL-18 binding protein, modulation of pro-inflammatory cytokine production, and activation of apoptosis.[15] Although experimental evidence shows that IFN-γ can contribute to pancreatic β-cell dysfunction, our clinical observations suggest that metabolic inflammation in routine T2DM is primarily an innate, macrophage-driven process within visceral adipose tissue (predominantly mediated by IL-6 and TNF-α) rather than a classical Th1 lymphocyte-driven systemic cascade.[14-16]

Several studies have evaluated inflammatory markers to assess metabolic control and complication risks in diabetic subjects. Son et al evaluated the influence of ferritin levels and inflammatory markers on HbA1c in type 2 diabetic patients, demonstrating elevated ferritin in diabetic cohorts and confirming its positive correlation with glycemic parameters.[17] Our findings concord with Son et al as serum ferritin was the sole biomarker positively correlated with both HbA1c ($\rho=0.38$, $p=0.007$) and fasting insulin ($\rho=0.33$, $p=0.021$), highlighting its dual role in subclinical reticuloendothelial inflammation and iron-mediated metabolic stress.

In contrast, although hs-CRP, IL-6, and TNF-α were markedly elevated in our diabetic group ($p<0.001$), they showed no statistically significant linear correlation with HbA1c or fasting insulin ($p>0.05$). This observation clarifies contrasting findings reported in earlier literature. While Sattar et al reported a positive correlation of bio-inflammatory markers (CRP, IL-6) with glucose levels in obese diabetic individuals, and Malenica et al observed higher hs-CRP and IL-6 concentrations among patients with elevated HbA1c, our data demonstrate that circulating cytokine levels signify an established, tonically active inflammatory state rather than a parameter that fluctuates linearly with 3-month glycemic exposure. Pro-inflammatory cytokines like IL-6 and TNF-α exhibit pulsatile secretion, short circulating half-lives, and rapid autocrine/paracrine receptor clearance, whereas HbA1c provides an integrated index of chronic glucose exposure over several months.[18,19]

Furthermore, while Shukaili et al observed shifting inflammatory mediators with decreased IL-6 and TNF-α but elevated CRP and IFN-γ in their cohort, our study demonstrated consistent, robust elevations in both IL-6 and TNF-α alongside hs-CRP, while IFN-γ remained unchanged. These data reaffirm that circulating IFN-γ is not a reliable surrogate of metabolic deterioration in routine T2DM.[20]

A limitation of our study is the relatively modest sample size (n=100) from a single tertiary care center and the cross-sectional case-control design, which precludes establishing causal temporality. Nevertheless, because our diabetic cohort consisted of newly diagnosed, medication-naive individuals, potential pharmacotherapeutic confounding from insulin or oral hypoglycemics was eliminated. Our findings establish widespread innate inflammatory activation in type 2 diabetes and suggest that serum ferritin provides greater practical utility than soluble cytokine panels when assessing concurrent glycemic and insulinemic status.

CONCLUSION

This study substantiates the presence of marked chronic low-grade systemic inflammation in patients with type 2 diabetes mellitus, demonstrated by significantly elevated serum concentrations of ferritin, hs-CRP, IL-6, and TNF-α compared to healthy controls ($p<0.001$), while circulating IFN-γ showed no significant intergroup variation ($p=0.515$). Notably, serum ferritin was the only biomarker that correlated positively and significantly with both glycemic control HbA1c (rho=0.38, $p=0.007$) and fasting hyperinsulinemia (rho=0.33, $p=0.021$). In contrast, soluble pro-inflammatory cytokines (IL-6, TNF-α) and hs-CRP exhibited no linear association with either parameter ($p>0.05$), indicating that while they signify an established, tonically active inflammatory background, they do not fluctuate proportionally with intermediate-term glycemic control. Therefore, serum ferritin uniquely parallels both chronic glycemic exposure and insulinemic burden, underscoring its clinical utility as a reliable, cost-effective surrogate biomarker for assessing metabolic strain and subclinical disease decompensation in routine pathology practice.

ACKNOWLEDGEMENTS

The authors would like to thank the patients who participated in the study and the staff members of the Department of Pathology and other collaborating departments for their valuable support and cooperation during the study. The authors also acknowledge the institutional support and facilities provided for conducting this research.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Varshney K, Singh M, Ranga S. Evaluating the association between inflammatory markers and glycemic control status in type 2 diabetes mellitus patients – a tertiary care centre experience. Int J Res Med Sci 2026;14:xxx-xx.