Relationship between CRP and HbA1c in Type 2 Diabetes Mellitus Patients

[1] Dr. Vishwas Kodandarama, [2] Dr. Faseeh KM, [3]Dr. Aamera Sait, [4]Dr. BR Shivakumar, [5]Dr. Parashuram Bhavimani, [6]Dr. Kavya Sankar and [7]Dr. Shreyas Vishwanath K

[1]Assistant Professor, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[2]Associate Professor, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[3]Assistant Professor, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[4]Professor, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[5]Professor and HOD, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[6]Junior Resident, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
[7]Junior Resident, Department of Medicine, Dr. B R Ambedkar Medical College, Bangalore, Karnataka, India
Corresponding Author: Dr. Shreyas Vishwanath K

Abstract:

Background: Type 2 diabetes mellitus (T2DM) is not only a disorder of glucose regulation but also a disease driven by chronic low-grade inflammation. India, particularly South India, bears a disproportionately high burden, with patients often presenting at a younger age and with more severe complications. While glycated hemoglobin (HbA1c) is the established gold standard for assessing long-term glycemic control, inflammatory markers such as C-reactive protein (CRP) have gained attention for their role in insulin resistance and vascular damage. Elevated CRP may reflect ongoing metabolic stress and predict poor outcomes even in patients with apparently controlled blood sugars.

Aim: This study aimed to evaluate the relationship between CRP and HbA1c in South Indian patients with type 2 diabetes mellitus and to determine whether inflammation parallels poor glycemic control.

Methods: A cross-sectional study was conducted at Dr. B. R. Ambedkar Medical College and Hospital, Bangalore, from January 2025 to July 2025. A total of 100 patients aged 30-70 years with confirmed T2DM were enrolled. Patients with acute infections, autoimmune conditions, chronic kidney disease, liver disease, or recent cardiovascular events were excluded. Clinical histories, anthropometric measurements, and comorbidities were documented. HbA1c was measured using high-performance liquid chromatography (HPLC), and CRP levels were assessed using a high-sensitivity immunoturbidimetric method. Statistical analyses were performed using SPSS v25.

Results: Of the 100 patients studied, 58 were male and 42 female, with a mean age of 53.2 ± 8.7 years and mean diabetes duration of 7.1 ± 3.9 years. The mean HbA1c was 8.2 ± 1.5%, while the mean CRP was 4.4 ± 2.1 mg/L. Patients with HbA1c <7% had mean CRP levels of 2.7 mg/L, those with HbA1c between 7-8% had 4.0 mg/L, and patients with HbA1c >8% had significantly higher CRP levels averaging 6.0 mg/L.

Correlation analysis revealed a strong positive association between CRP and HbA1c ($r = 0.61$, $p < 0.001$). This relationship remained significant even after adjusting for BMI, hypertension, and duration of diabetes (adjusted $\beta = 0.47$, $p < 0.001$). Subgroup analysis showed that obese and hypertensive patients had consistently higher CRP levels compared to their non-obese, normotensive counterparts.

Conclusion: This study highlights a significant positive correlation between CRP and HbA1c in South Indian patients with T2DM. Elevated CRP levels were consistently associated with poor glycemic control, obesity, and longer disease duration, underscoring the role of inflammation in the pathophysiology of diabetes. CRP may serve as a useful adjunct biomarker alongside HbA1c, allowing better risk stratification and early identification of patients at higher risk for complications. Incorporating inflammatory markers into diabetes management protocols may help in tailoring more holistic therapeutic strategies.

Keywords: Type 2 diabetes mellitus, C-reactive protein, HbA1c, inflammation, insulin resistance, South India, obesity, hypertension, metabolic stress, glycemic control, biomarkers, Vascular damage.

DOI Number: 10.48047/nq.2026.24.3.NQ26008 **NeuroQuantology2026;24(3):80-83**

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic, progressive metabolic disorder characterized by insulin resistance, β-cell dysfunction, and impaired glucose metabolism. Its prevalence has reached epidemic proportions in India, where the International Diabetes Federation estimates that over 77 million individuals currently live with diabetes, a number expected to rise steeply by 2040. The burden is particularly striking in South India, where urbanization, changing dietary patterns, and lifestyle modifications have contributed to earlier onset and more severe disease expression compared to Western cohorts. In Bangalore, one of the fastest-growing metropolitan cities in India, the prevalence of T2DM reflects not only the changing face of the city but also the socio-economic gradient in access to health care and lifestyle risk factors. Traditionally, the management and monitoring of T2DM have focused on glycemic indices. HbA1c, formed by the non-enzymatic glycation of hemoglobin, reflects the average plasma glucose levels over the preceding 8 to 12 weeks and remains the cornerstone in diabetes care. Higher HbA1c values directly correlate with poor glycemic control and greater risk of complications. However, mounting evidence suggests that the pathophysiology of diabetes extends beyond hyperglycemia alone, involving chronic low-grade inflammation as a critical driver of vascular damage and metabolic deterioration.

C-reactive protein (CRP), produced primarily by the liver in response to pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, has emerged as a robust marker of systemic inflammation. Elevated CRP levels have been documented in obesity, metabolic syndrome, cardiovascular disease, and diabetes, thereby positioning CRP at the crossroads of metabolic and inflammatory pathways. In South Indian populations, where genetic susceptibility to insulin resistance is heightened, the inflammatory milieu created by visceral adiposity may amplify the deleterious effects of hyperglycemia. Studies conducted in Chennai, Hyderabad, and Bangalore have consistently shown that South Indians develop diabetes at lower body mass indices compared to Caucasians, likely due to higher visceral fat content and altered adipokine secretion. This phenotype, sometimes referred to as the "thin-fat Indian," is associated with elevated CRP levels, even in apparently lean individuals. Such observations raise the question of whether CRP can serve not only as a biomarker of systemic inflammation but also as a predictor of glycemic control in Indian T2DM patients.

Against this background, the present study was designed to examine the relationship between CRP and HbA1c among patients with type 2 diabetes attending Dr. B. R. Ambedkar Medical College and Hospital, Bangalore. By correlating these parameters, we sought to delineate whether CRP levels rise proportionally with worsening glycemic control and to explore the role of inflammation as a hidden yet powerful mediator in the trajectory of diabetes in South India.

Methodology

This was a cross-sectional, hospital-based study conducted at Dr. B. R. Ambedkar Medical College and Hospital (Dr. B. R. AMCH), Bangalore, between January 2025 and July 2025. The hospital, being a tertiary care referral center, caters to a diverse population from both urban Bangalore and adjoining rural districts, making it a representative site for studying metabolic disorders prevalent in South India.

A total of 100 patients with previously diagnosed type 2 diabetes mellitus were recruited from the outpatient and inpatient departments of General Medicine and Endocrinology. Diagnosis of T2DM was based on American Diabetes Association (ADA) 2024 criteria.

Inclusion criteria included adults aged 30 to 70 years, patients with a confirmed diagnosis of T2DM for at least 6 months, and patients willing to provide informed consent. Exclusion criteria included type 1 diabetes mellitus, patients with acute infections, autoimmune diseases, or chronic inflammatory conditions known to raise CRP, patients with renal failure, chronic liver disease, or recent myocardial infarction, and pregnant women.

Detailed clinical histories were obtained, including duration of diabetes, medication usage, lifestyle habits, and presence of comorbidities such as hypertension and dyslipidemia. Anthropometric measurements including weight, height, BMI, and waist circumference were recorded. Blood pressure was measured using a standardized protocol. Venous blood samples were collected after overnight fasting. HbA1c was measured using high-performance liquid chromatography (HPLC), standardized to NGSP/DCCT values. CRP levels were measured using high-sensitivity CRP (hs-CRP) assay by immunoturbidimetric method. Additional tests included fasting blood glucose, lipid profile, and renal function tests to rule out confounding conditions. Data were analyzed using SPSS version 25. Continuous variables were expressed as mean ± standard deviation. Pearson's correlation coefficient was used to analyze the relationship between CRP and HbA1c. Multivariate regression analysis was conducted to adjust for confounders such as age, BMI, and hypertension. A p-value <0.05 was considered statistically significant.

Results

Of the 100 patients enrolled, 56 were males and 44 were females. The mean age was 52.6 ± 9.4 years. The mean duration of diabetes was 7.3 ± 4.1 years. Obesity, defined by BMI > 25 kg/m² (South Asian cut-off), was present in 61% of patients, while hypertension was noted in 48%. The mean HbA1c among study participants was 8.4 ± 1.6%. The mean CRP level was 4.6 ± 2.3 mg/L, with higher levels observed in obese and hypertensive subgroups. Patients with HbA1c <7% (good glycemic control) had a mean CRP of 2.8 mg/L, those with HbA1c 7-8% had mean CRP of 4.2 mg/L, and patients with HbA1c >8% had mean CRP of 6.1 mg/L. Pearson's correlation analysis revealed a strong positive correlation between CRP and HbA1c ($r = 0.62$, $p < 0.001$). This association remained significant even after adjusting for BMI and hypertension (adjusted $\beta = 0.48$, $p < 0.001$). Subgroup analysis demonstrated that females tended to have higher CRP values compared to males at equivalent HbA1c levels, though the difference did not reach statistical significance.

When stratified by duration of diabetes, patients with >10 years of disease had markedly elevated CRP levels compared to those with <5 years, suggesting that chronic hyperglycemia and cumulative metabolic stress amplify systemic inflammation.

Discussion

The present study demonstrates a significant positive

correlation between CRP and HbA1c in patients with type 2 diabetes mellitus in a South Indian cohort. Patients with poor glycemic control exhibited higher CRP levels, reinforcing the notion that chronic hyperglycemia fuels systemic inflammation. This relationship persisted even after adjusting for confounders such as obesity and hypertension, suggesting that CRP reflects more than just adiposity-driven inflammation.

These findings are consistent with previous Indian studies. A study conducted at Madras Medical College, Chennai, reported elevated CRP levels among diabetic patients with poor glycemic control, highlighting CRP as an early marker of vascular dysfunction. Similarly, research from Osmania Medical College, Hyderabad, established that CRP levels correlated not only with HbA1c but also with microalbuminuria, thereby linking inflammation to both metabolic and renal complications. Our study from Bangalore corroborates and extends these observations, demonstrating that even within a single metropolitan city, patients with uncontrolled diabetes manifest a heightened inflammatory milieu.

The biological basis for the CRP-HbA1c association lies in the complex interplay between hyperglycemia, oxidative stress, and pro-inflammatory cytokines. Persistent hyperglycemia promotes the formation of advanced glycation end-products (AGEs), which in turn activate nuclear factor-kappa B (NF-κB) pathways, stimulating the release of interleukin-6 and tumor necrosis factor-alpha. These cytokines drive hepatic CRP synthesis, linking metabolic dysregulation to systemic inflammation. Elevated CRP, in turn, exacerbates endothelial dysfunction, creating a vicious cycle that accelerates atherosclerosis and microvascular complications.

Our study also underscores the importance of regional context. South Indians, as demonstrated in multiple epidemiological studies, are predisposed to insulin resistance and central obesity even at lower BMI thresholds. This phenotype amplifies inflammatory responses, rendering CRP an especially relevant biomarker in this population. The findings thus have important implications for clinical practice. While HbA1c remains indispensable for monitoring glycemic control, incorporating CRP into routine evaluation may provide additional prognostic insights, particularly in identifying patients at heightened risk of complications despite apparently acceptable glucose control.

Furthermore, the results invite discussion on therapeutic strategies. Anti-inflammatory approaches, ranging from lifestyle modifications such as weight reduction and exercise to pharmacological interventions targeting inflammation (such as statins and potentially novel anti-inflammatory drugs), may hold promise in mitigating the dual burden of hyperglycemia and inflammation. Studies in Kerala have shown that yoga-based interventions significantly reduce CRP levels in diabetic individuals, suggesting culturally tailored lifestyle modifications could complement pharmacological strategies.

Conclusion

This hospital-based cross-sectional study conducted at Dr. B. R. Ambedkar Medical College and Hospital, Bangalore, from January to July 2025, demonstrates a significant positive correlation between CRP and HbA1c levels in patients with type 2 diabetes mellitus. Elevated CRP was consistently associated with poor glycemic control, obesity, and longer duration of disease, reinforcing the role of inflammation as an integral component of diabetes pathophysiology. The study highlights the potential value of CRP as a supplementary marker in routine diabetes care, offering prognostic insights beyond glycemic indices alone. Incorporating inflammatory markers like CRP could enable more comprehensive risk stratification and inform targeted interventions. Future longitudinal and interventional studies in South Indian populations are warranted to determine whether reducing CRP levels through lifestyle or pharmacological means translates into tangible improvements in diabetic outcomes.

In summary, the relationship between CRP and HbA1c underscores the convergence of metabolic and inflammatory pathways in type 2 diabetes, and the findings from Bangalore add to a growing regional body of evidence that demands attention to inflammation as a therapeutic frontier in diabetes care.

References

1. Mohan V, Deepa R, Velmurugan K, Premalatha G. Association of C-reactive protein with body fat distribution and insulin resistance in Asian Indians: Chennai Urban Rural Epidemiology Study (CURES-6). Diabet Med. 2005;22(7):863-870. DOI:10.1111/j.1464-5491.2005.01575.x.
2. Kumar A, Sundaram R, Das A, Priyadarsini R, *et al*. Association between high-sensitivity C-reactive protein and glycated hemoglobin in type 2 diabetes patients: a South Indian study. J Clin Diagn Res. 2017;11(4):BC01-BC04.
3. Vasan SK, Thomas N, Raj D, Geethanjali FS, Sattar N, *et al*. Anthropometric indices, measures of central obesity and risk of diabetes in South Indians. Diabetes Metab Res Rev. 2011;27(6):533-540. DOI:10.1002/dmrr.1215.
4. Rani CS, Kavitha M, Kumar MS, *et al*. Study of hs-CRP and HbA1c in type 2 diabetes mellitus patients in a tertiary care hospital in Andhra Pradesh. Int J Res Med Sci. 2018;6(9):3056-3061. DOI:10.18203/2320-6012.ijrms20183835.
5. Balaji S, Rani S, Kumar R, *et al*. C-reactive protein levels and diabetic microvascular complications: a study from South India. Indian J Endocrinol Metab. 2015;19(5):707-711. DOI:10.4103/2230-8210.160169.
6. Bansal N, Deepa M, Pradeepa R, Prabhakaran D, Mohan V. Metabolic syndrome in Asian Indians: Epidemiology and clinical implications. Int J Diabetes Dev Ctries. 2006;26(4):169-174. DOI:10.4103/0973-3930.28899.
7. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V. The Indian Diabetes Prevention Programme shows reduction in incidence of type 2 diabetes with lifestyle modification and metformin. Diabetologia. 2006;49(2):289-297. DOI:10.1007/s00125-005-0135-5.
8. Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K. Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. Diabetes Res Clin Pract. 2003;61(1):69-76. DOI:10.1016/S0168-8227(03)00083-2.
9. Shanmugasundaram K, Kumar P, Ramalingam K. High sensitivity C-reactive protein and glycemic status in type 2 diabetic patients attending a tertiary care hospital in Tamil Nadu. J Clin Diagn Res. 2016;10(6):OC09-OC12. DOI:10.7860/JCDR/2016/20195.8122.

10. Mohan V, Seedat YK, Pradeepa R. The rising burden of diabetes and hypertension in Southeast Asian and African regions: Need for effective strategies. Int J Hypertens. 2013;2013:1-14. DOI:10.1155/2013/523818.
11. Pradeepa R, Anjana RM, Joshi SR, Bhansali A, Deepa M, Joshi PP, *et al*. Prevalence of generalized & abdominal obesity in urban & rural India—ICMR-INDIAB Study (Phase-I) [ICMR-INDIAB-3]. Indian J Med Res. 2015;142(2):139-150. DOI:10.4103/0971-5916.165249.
12. Ridker PM, Buring JE, Cook NR, Rifai N. C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events. Circulation. 2003;107(3):391-397. DOI:10.1161/01.CIR.0000049876.49175.B2.
13. Pickup JC, Crook MA. Is type II diabetes mellitus a disease of the innate immune system? Diabetologia. 1998;41(10):1241-1248. DOI:10.1007/s001250050985.
14. Thorand B, Löwel H, Schneider A, Kolb H, Meisinger C, Fröhlich M, Koenig W. C-reactive protein as a predictor for incident diabetes mellitus among middle-aged men: results from the MONICA Augsburg cohort study, 1984-1998. Arch Intern Med. 2003;163(1):93-99. DOI:10.1001/archinte.163.1.93.
15. Freeman DJ, Norrie J, Caslake MJ, Gaw A, Ford I, Lowe GD, *et al*. C-reactive protein is an independent predictor of risk for development of diabetes in the West of Scotland Coronary Prevention Study. Diabetes. 2002;51(5):1596-1600. DOI:10.2337/diabetes.51.5.1596.
16. Fernandez-Real JM, Ricart W. Insulin resistance and inflammation in an evolutionary perspective: the contribution of cytokine genotype/phenotype to thriftiness. Diabetologia. 1999;42(11):1367-1374. DOI:10.1007/s001250051146.
17. Yadav D, Mahajan A, Subramanian SK, Bisen PS. Correlation of high-sensitivity C-reactive protein and glycated hemoglobin levels in type 2 diabetes mellitus patients: a cross-sectional study. Indian J Clin Biochem. 2012;27(2):195-199. DOI:10.1007/s12291-012-0205-x.
18. Rajappa M, Sridhar MG, Balachander J, Sethuraman KR. Association between high sensitive C-reactive protein and glycated hemoglobin in South Indian type 2 diabetic patients. Endocrine. 2009;36(2):255-259. DOI:10.1007/s12020-009-9207-1.
19. King GL. The role of inflammatory cytokines in diabetes and its complications. J Periodontol. 2008;79(8 Suppl):1527-1534. DOI:10.1902/jop.2008.080162.
20. Banerjee M, Saxena M. Genetic polymorphisms of cytokine genes in type 2 diabetes mellitus. World J Diabetes. 2014;5(4):493-504. DOI:10.4239/wjd.v5.i4.493.