



Research Article

Integrated Assessment of C - Reactive Protein and Glycated Haemoglobin Dysregulation in Diabetic Patients attending Kwara State University Teaching Hospital, Ilorin

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ABSTRACT

Diabetes mellitus is associated with concurrent glycaemic and inflammatory dysregulation, but glycated haemoglobin (HbA1c) and C-reactive protein (CRP) are frequently assessed separately, and evidence on their concurrent evaluation among Nigerian diabetic patients remains limited. This hospital-based comparative study evaluated HbA1c and high-sensitivity CRP (Hs-CRP) among 50 diabetic patients and 50 non-diabetic controls attending Kwara State University Teaching Hospital (KWASUTH), Ilorin. HbA1c was determined by enzyme-linked immunosorbent assay, and Hs-CRP was measured by fluorescence immunoassay. Diabetic participants had significantly higher fasting blood sugar, HbA1c, and Hs-CRP than controls (all $p < 0.001$). Among diabetic participants, median HbA1c was 8.85% (IQR 7.80–10.25) and median Hs-CRP was 5.05 mg/L (IQR 1.88–5.10), and HbA1c correlated positively with Hs-CRP ($p = 0.514$, $p < 0.001$). Diabetic participants were significantly older than controls (56.4 ± 10.8 versus 50.7 ± 9.4 years, $p = 0.006$), while sex distribution did not differ significantly between groups ($\chi^2 = 0.04$, $p = 0.841$). Among the 50 non-diabetic controls, 14.0% had HbA1c $\geq 5.7\%$, 4.0% had fasting blood sugar ≥ 5.6 mmol/L, and 16.0% had Hs-CRP > 3.0 mg/L, 30.0% had at least one of these glycaemic or inflammatory risk markers. These findings demonstrate a significant positive relationship between glycaemic dysregulation and systemic inflammation among diabetic patients at KWASUTH, and show that a subset of apparently non-diabetic individuals already display glycaemic or inflammatory abnormalities, supporting the value of combined HbA1c and CRP assessment for risk stratification and diabetes management in this population.

Keywords: C-Reactive Protein; Diabetes Mellitus; Glycated Haemoglobin; Glycaemic Control; Inflammation; Nigeria

Citation: Yahya, B.A., Olaniyan, M.F., Garuba, W.O., & Hassan, S.O. (2026). Integrated Assessment of C – Reactive Protein and Glycated Haemoglobin Dysregulation in Diabetic Patients attending Kwara State University Teaching Hospital, Ilorin. *Sahel Journal of Life Sciences FUDMA*, 4(3): 335-341. DOI: <https://doi.org/10.33003/sajols-2026-0403-35>

INTRODUCTION

Diabetes mellitus is increasingly recognised as a complex metabolic-inflammatory disease rather than a disorder of glucose regulation alone. Persistent hyperglycaemia promotes oxidative stress, the

formation of advanced glycation end products, and activation of innate immune pathways, which together contribute to endothelial dysfunction and the development of diabetes-related complications (Donath, Dinarello, & Mandrup-Poulsen, 2019;

Garcia-Garcia *et al.*, 2020). Metabolic stress in diabetes can activate pro-inflammatory signalling, while the resulting low-grade inflammation may in turn impair insulin signalling and aggravate insulin resistance, creating a potentially self-sustaining interaction between hyperglycaemia and inflammation (Chinko & Efebo, 2025; Tsalamandris *et al.*, 2019).

Glycated haemoglobin (HbA1c) and C-reactive protein (CRP) are two widely used laboratory indices that reflect these two related but distinct biological dimensions of diabetes. HbA1c is a stable glycated form of haemoglobin formed through the non-enzymatic attachment of glucose to circulating erythrocytes, and provides an integrated measure of glycaemic exposure over the preceding two to three months (Islam & Rahman, 2023). It is recognised by the American Diabetes Association Professional Practice Committee (2025) as a key biomarker for assessing glycaemic status and diagnosing diabetes; the same criteria define an HbA1c of 5.7–6.4% and a fasting plasma glucose of 5.6–6.9 mmol/L as indicating prediabetes. Gomez-Peralta *et al.* (2022) similarly emphasised the clinical value of HbA1c for evaluating prolonged glycaemic exposure, while noting that its interpretation should take into account factors that can influence the relationship between HbA1c and average glucose in individual patients. CRP, in contrast, is an acute-phase protein produced predominantly by the liver in response to inflammatory stimulation and is widely used as a marker of systemic inflammatory activity (Rakesh, 2017; Sproston & Ashworth, 2018). Elevated CRP has been linked to metabolic dysfunction and vascular abnormalities in diabetic patients, supporting its relevance to inflammatory assessment in this population (Stanimirovic *et al.*, 2022). Although HbA1c and CRP are interrelated, they are frequently assessed separately in clinical practice, which limits the ability to characterise the combined metabolic and inflammatory status of diabetic patients (Ibama & Uwikor, 2025; Obeagu, 2026).

Evidence from within Nigeria supports the presence of both glycaemic and inflammatory abnormalities among people living with diabetes. Ekun *et al.* (2022) reported significantly higher HbA1c and high-sensitivity CRP among 160 adults recently diagnosed with type 2 diabetes compared with 90 non-diabetic controls, while Agho *et al.* (2021) similarly reported higher salivary CRP concentrations among diabetic patients than healthy controls attending a Nigerian teaching hospital. At the population level, Ajayi *et al.* (2023) reported a diabetes prevalence of 14.2% in

Kwara State, the highest among the Nigerian states surveyed, indicating a substantial local burden of dysglycaemia. Because HbA1c assay performance can be affected by haemoglobin variants that are common in sub-Saharan African populations, locally generated evidence on HbA1c is particularly important for this setting (Balungi *et al.*, 2024).

Despite this evidence, relatively few Nigerian studies have concurrently assessed HbA1c and CRP within the same diabetic population, and evidence specifically from Kwara State University Teaching Hospital (KWASUTH) remains limited. This gap is particularly relevant given that Kwara State has been reported to have the highest prevalence of HbA1c-based dysglycaemia among the Nigerian states surveyed by Ajayi *et al.* (2023), while KWASUTH itself is a tertiary referral centre drawing diabetic patients from across Kwara State and neighbouring North-Central states; the demographic and clinical profile of this referral population has not previously been characterised for combined HbA1c-CRP evaluation. Moreover, existing Nigerian studies have generally reported HbA1c and CRP findings separately rather than as an integrated, dual-biomarker risk-stratification matrix capable of jointly flagging diabetic and at-risk non-diabetic individuals; to our knowledge, no study from North-Central Nigeria has combined HbA1c and CRP in this way. This creates a gap in local knowledge regarding the levels of HbA1c and CRP among diabetic patients at KWASUTH, their correlation, their distribution relative to non-diabetic controls, and the prevalence of glycaemic and inflammatory risk markers among apparently healthy individuals. This study therefore evaluated HbA1c and Hs-CRP among diabetic patients and non-diabetic controls attending KWASUTH, examined the correlation between the two biomarkers among diabetic participants, compared their levels between the two groups, and determined the prevalence of selected glycaemic and inflammatory risk markers among the non-diabetic controls.

MATERIALS AND METHODS

Study Design and Area

This was a hospital-based, comparative study with a cross-sectional data-collection design, conducted at Kwara State University Teaching Hospital (KWASUTH), Ilorin, Kwara State, Nigeria, a tertiary referral centre providing specialist diagnostic and clinical services to patients across Kwara State and neighbouring North-Central states.

Study Participants

The study population comprised adult individuals aged 18–70 years attending the diabetic clinic at Kwasuth. Only patients with a clinical diagnosis of type 2 diabetes mellitus were eligible for inclusion, consistent with the predominance of type 2 diabetes within this adult age range; patients with type 1 diabetes mellitus were not included. Diabetic participants were diagnosed according to the American Diabetes Association Professional Practice Committee (2025) criteria (fasting plasma glucose ≥ 7.0 mmol/L, HbA1c $\geq 6.5\%$, or an existing clinical diagnosis of type 2 diabetes mellitus with a documented antidiabetic medication history) and provided informed consent; individuals with a history of acute or chronic infection, recent surgery or inflammatory disease, use of anti-inflammatory medication within the preceding two weeks, autoimmune disorders, or pregnancy were excluded. Non-diabetic control participants were recruited from hospital staff and accompanying relatives and were screened by clinical history, fasting blood sugar, and HbA1c to exclude diabetes mellitus, as well as for acute or chronic inflammatory conditions that could affect Hs-CRP. The minimum sample size was calculated using a formula for comparing two proportions (Charan & Biswas, 2013), based on an expected CRP positivity of 60% among diabetic participants and 30% among controls, a 95% confidence level ($Z\alpha=1.96$), and 80% power ($Z\beta=0.84$), giving a minimum requirement of 40 participants per group; this was increased to 44 per group after a 10% allowance for attrition, and further increased to 50 per group to enhance adequacy and ensure equal representation of the two study groups, giving a final sample of 100 participants (50 diabetic patients and 50 non-diabetic controls). Anthropometric measures such as body mass index and waist circumference, which are recognised determinants of both glycaemic control and CRP concentration, were not collected as part of this protocol; this is acknowledged as a limitation in the Discussion.

Ethical Considerations

Ethical approval for this study was obtained from the Kwara State University Teaching Hospital (Kwasuth) Institutional Review Committee (Kwasuth/IRC/246/VOL.III/38) and the Kwara State Ministry of Health Ethical Research Committee (ERC/MOH/2026/05/642). Written informed consent was obtained from all participants prior to enrolment, and participant confidentiality was maintained throughout using coded identifiers rather than personal identifiers.

Collection of Samples and Laboratory Analysis

Approximately 5 mL of venous blood was collected aseptically from each participant by antecubital venepuncture between 7:00 and 9:00 am after a minimum of 8 hours of fasting, and dispensed into an EDTA anticoagulant tube. Glycated haemoglobin (HbA1c) was determined by an enzyme-linked immunosorbent assay (Eldere HbA1c ELISA kit, Shanghai Aidele Medical Technology Co., Ltd, Shanghai, China): erythrocytes were lysed to release haemoglobin, and the resulting haemolysate, together with HbA1c standards and controls, was added to antibody-coated microplate wells; following incubation and washing, an enzyme-labelled conjugate and substrate were added, and absorbance was read spectrophotometrically at 450 nm and compared with the calibration curve to derive the HbA1c concentration, expressed as a percentage. High-sensitivity C-reactive protein (Hs-CRP) was measured by fluorescence immunoassay using the Finecare FIA system (Model W201): 8.5 μ L of whole blood was mixed with detection buffer, and 75 μ L of the resulting mixture was dispensed into the sample well of the test cartridge, which was inserted into the Finecare FIA meter after a 3-minute room-temperature incubation, with the quantitative result displayed automatically by the analyser. For both assays, lot-matched reagents, calibrators, and assay controls were used, and manufacturer expiry dates were verified, in accordance with the respective manufacturers' instructions.

Data Analysis

Data were analysed using IBM SPSS Statistics, version 25.0. Mann-Whitney U test was used to compare these variables between diabetic participants and non-diabetic controls, while the independent samples t-test was used to compare age between groups. The relationship between HbA1c and Hs-CRP among diabetic participants was assessed using Spearman's rank correlation, as the assumption of normality was not met for Hs-CRP. The chi-square test was used to compare the sex distribution between groups. Statistical significance was set at $p < 0.05$.

RESULTS

A total of 100 participants were studied, comprising 50 diabetic patients and 50 non-diabetic controls (Table 1). Diabetic participants were significantly older than controls (56.4 ± 10.8 versus 50.7 ± 9.4 years, $p = 0.006$), while sex distribution did not differ significantly between the two groups ($p = 0.841$). Diabetic participants had significantly higher fasting blood sugar, HbA1c, and Hs-CRP than non-diabetic

controls ($p<0.001$) (Table 2). Among diabetic participants, median HbA1c was 8.85% (IQR 7.80–10.25) and median Hs-CRP was 5.05 mg/L (IQR 1.88–5.10); among controls, median HbA1c was 5.30% (IQR 5.10–5.60) and median Hs-CRP was 0.85 mg/L (IQR 0.50–1.20).

The relationship between HbA1c and Hs-CRP among diabetic participants shows a moderate, statistically significant positive correlation (Table 3, Figure 1), indicating that participants with higher HbA1c levels tended to have higher Hs-CRP concentrations.

Among the 50 non-diabetic controls, 2 (4.0%) had fasting blood sugar ≥ 5.6 mmol/L, 7 (14.0%) had HbA1c $\geq 5.7\%$, and 8 (16.0%) had Hs-CRP >3.0 mg/L (Table 4). Overall, 35 (70.0%) of controls had none of the three selected risk markers, 13 (26.0%) had one marker, and 2 (4.0%) had two markers; no control participant met *all* three thresholds. In total, 15 (30.0%) of non-diabetic controls had at least one selected glycaemic or inflammatory risk marker.

Table 1: Demographic Characteristics of Diabetic Participants and Non-Diabetic Controls

Characteristic	Diabetic n (%)	Control n (%)	p-value
Age group (years)			0.006¹
<40	3 (6.0)	6 (12.0)	
40–49	11 (22.0)	16 (32.0)	
50–59	11 (22.0)	19 (38.0)	
60–69	20 (40.0)	9 (18.0)	
≥ 70	5 (10.0)	0 (0.0)	
Sex			0.841²
Female	27 (54.0)	25 (50.0)	
Male	23 (46.0)	25 (50.0)	
Total	50 (100)	50 (100)	

Note. ¹Independent samples t-test comparing mean age (diabetic 56.4 ± 10.8 vs control 50.7 ± 9.4 years). ²Chi-square test ($\chi^2=0.04$)

Table 2: Comparison of Fasting Blood Sugar, HbA1c, and Hs-CRP between Diabetic Participants and Non-Diabetic Controls

Variable	Diabetic (IQR) (n=50)	Control (IQR) (n=50)	Mann-Whitney U	p-value
FBS (mmol/L)	9.35 (8.45–11.35)	5.00 (4.70–5.38)	2500.0	<0.001
HbA1c (%)	8.85 (7.80–10.25)	5.30 (5.10–5.60)	2460.5	<0.001
Hs-CRP (mg/L)	5.05 (1.88–5.10)	0.85 (0.50–1.20)	1953.0	<0.001

$p<0.05$ is statistically significant

Table 3: Correlation between HbA1c and Hs-CRP Among Diabetic Participants

Variables Correlated	N	Spearman's ρ	p-value
HbA1c vs Hs-CRP (diabetic participants)	50	0.514	<0.001

$p<0.05$ is statistically significant

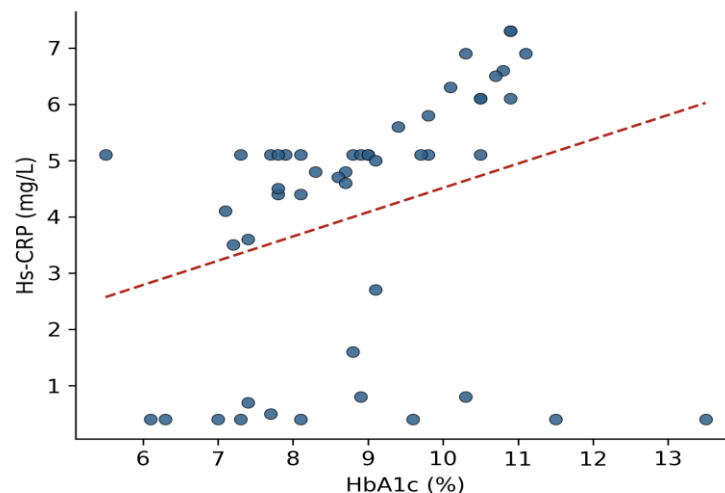


Figure 1: Scatter plot showing the correlation between HbA1c and Hs-CRP among diabetic participants (Spearman's $\rho=0.514$, $p<0.001$)

Note: a subset of Hs-CRP values cluster near the upper and lower ends of the fluorescence immunoassay's quantitative measuring range; these clustered points likely reflect the assay's ceiling and floor effects rather than a true biological plateau and should be interpreted with this measurement limitation in mind (see Discussion).

Table 4: Prevalence of Selected Glycaemic and Inflammatory Risk Markers among Non-Diabetic Controls

Risk Marker	Frequency, n (%)
Individual risk markers	
FBS ≥ 5.6 mmol/L	2 (4.0)
HbA1c $\geq 5.7\%$	7 (14.0)
Hs-CRP > 3.0 mg/L	8 (16.0)
Number of risk markers present	
0	35 (70.0)
1	13 (26.0)
2	2 (4.0)
3	0 (0.0)
≥ 1 (at least one marker)	15 (30.0)

DISCUSSION

Diabetic participants in this study were significantly older than non-diabetic controls, consistent with the pattern of type 2 diabetes presenting predominantly in middle and later adulthood, and with the substantial burden of dysglycaemia already reported among adult populations in Kwara State (Ajayi *et al.*, 2023). The sex distribution did not differ significantly between diabetic participants and controls, suggesting that the observed differences in glycaemic and inflammatory markers were unlikely to be driven by sex composition, although the age imbalance between groups should be borne in mind when interpreting the between-group comparisons, as age itself may independently influence both glycaemic and inflammatory status. Beyond age, body mass index, central obesity, lipid profile, and statin use are recognised independent determinants of CRP concentration, but these variables were not measured in the present study (see Limitations); their potential contribution to the observed between-group differences in Hs-CRP therefore cannot be excluded and should be addressed in future studies that incorporate full anthropometric and metabolic profiling.

Diabetic participants had significantly higher fasting blood sugar, HbA1c, and Hs-CRP than non-diabetic controls, findings that are consistent with previous Nigerian evidence. Ekun *et al.* (2022) similarly reported significantly higher HbA1c and high-sensitivity CRP among adults recently diagnosed with type 2 diabetes compared with non-diabetic controls, while Agho *et al.* (2021) reported higher salivary CRP among diabetic patients than healthy controls at a Nigerian teaching hospital. These consistent findings across different Nigerian populations and specimen types support the view that diabetes in this setting is

accompanied by measurable systemic inflammation in addition to glycaemic dysregulation, in keeping with the broader conceptualisation of diabetes as a combined metabolic-inflammatory disorder (Donath, Dinarello, & Mandrup-Poulsen, 2019; Galicia-Garcia *et al.*, 2020).

The moderate, statistically significant positive correlation observed between HbA1c and Hs-CRP among diabetic participants ($p=0.514$, $p<0.001$) supports the hypothesis that greater glycaemic dysregulation is associated with greater systemic inflammatory activity in this population. This is biologically consistent with the proposed hyperglycaemia-inflammation pathway, in which persistent hyperglycaemia promotes oxidative stress and the formation of advanced glycation end products, which in turn stimulate inflammatory signalling and hepatic acute-phase protein production, including CRP (Rakesh, 2017; Sproston & Ashworth, 2018; Stanimirovic *et al.*, 2022; Tsalamandris *et al.*, 2019). Because CRP can also be influenced by factors unrelated to glycaemic status, such as infection, adiposity, and other inflammatory conditions, the correlation observed here should be interpreted as evidence of a biologically plausible association rather than a causal relationship, particularly given the cross-sectional design of this study (Ibama & Uwikor, 2025; Obeagu, 2026).

The risk-marker analysis among the 50 non-diabetic controls showed that 30.0% had at least one selected glycaemic or inflammatory risk marker, most commonly Hs-CRP > 3.0 mg/L (16.0%) and HbA1c $\geq 5.7\%$ (14.0%). The American Diabetes Association Professional Practice Committee (2025) recognises an HbA1c of 5.7–6.4% as indicating prediabetes, so the presence of this threshold in 14.0% of apparently non-diabetic participants may reflect early, sub-

clinical glycaemic dysregulation that would not otherwise have been detected. Similarly, the presence of elevated Hs-CRP in a subset of controls may be relevant to cardiovascular risk assessment, since Hs-CRP reflects systemic inflammation that has been linked to vascular and metabolic dysfunction independent of diagnosed diabetes (Stanimirovic *et al.*, 2022). The elevated Hs-CRP observed in this subset of controls may reflect early features of the metabolic syndrome, such as undiagnosed visceral adiposity or dyslipidaemia, which are common precursors of both dysglycaemia and low-grade systemic inflammation in Nigerian populations; dietary patterns high in refined carbohydrate and saturated fat, and less commonly, unrecognised subclinical infection, may also contribute. These potential drivers were not directly assessed in the present study and warrant targeted investigation, given their relevance to cardiovascular risk stratification in this population. These findings support the value of opportunistic glycaemic and inflammatory screening in hospital populations, and suggest that individuals identified with one or more risk markers may benefit from closer clinical follow-up, lifestyle counselling, and monitoring for the future development of diabetes and its complications, in keeping with the diabetes-management implications previously highlighted for this study population. The consistent direction of findings across multiple comparisons, and their concordance with previous Nigerian evidence, support the overall conclusion that glycaemic and inflammatory dysregulation are significantly associated among diabetic patients at Kwasuth.

CONCLUSION

This study demonstrated that diabetic patients attending Kwasuth had significantly higher fasting blood sugar, HbA1c, and Hs-CRP than non-diabetic controls, and that HbA1c and Hs-CRP were moderately and significantly positively correlated among diabetic participants, supporting a significant relationship between glycaemic dysregulation and systemic inflammation in this population. Additionally, approximately three in ten apparently non-diabetic controls displayed at least one selected glycaemic or inflammatory risk marker, indicating that sub-clinical abnormalities may be present even among individuals without a diabetes diagnosis. These findings support the value of the combined assessment of HbA1c and CRP for characterising the metabolic and inflammatory status of diabetic patients, and for identifying apparently healthy

individuals who may benefit from closer monitoring, in this Nigerian hospital population. Based on these findings, routine dual screening with HbA1c and Hs-CRP is recommended for patients attending the Kwasuth diabetic clinic, and for apparently healthy individuals presenting with other cardiometabolic risk factors, as a practical, low-cost strategy for the early identification of combined glycaemic and inflammatory risk and for guiding timely lifestyle counselling and clinical follow-up before overt diabetes or cardiovascular disease develops.

Author Contributions: Yahya Abubakar Bilkisu conceived the study, recruited participants, collected the biological samples, and performed the HbA1c and Hs-CRP laboratory analyses. Mathew Folaranmi Olanayan supervised the study and its design. Wasii Olanrewaju Garuba contributed to the laboratory analysis. Seidat Omolabake Hassan carried out the statistical analysis, drafted, reviewed and edited the manuscript. All authors read and approved the final manuscript.

Conflicts of Interest: The authors declare no conflict of interest.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors.

Data Availability: The dataset analysed during the current study is available from the corresponding author on reasonable request.

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