ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

Biomarkers of Cardiovascular Injury, Cardiac Stress and Inflammation in Adults with and Without Diabetes Mellitus: A Comparative Study

Chinakila J. Musonda[1*]; Brian Mukuka[1]; Bislom C. Mweene[2]; Benson M. Hamooya[2]; Sepiso K. Masenga[2;3]; Badisa John Phafane[4]

[1]University of Lusaka, School of Medicine and Health Sciences, Lusaka, Zambia.
[2]Mulungushi University, School of Medicine and Health Sciences, Livingstone, Zambia.
[3]Department of Medicine, Vanderbilt University Medical Center, Nashville, TN, USA.
[4] Diagnofirm Medical Laboratories, Gaborone, Botswana.

Corresponding Author: Chinakila J. Musonda[1*]

Publication Date: 2026/10/30

Abstract:

- ***Background:***

Diabetes mellitus is associated with increased cardiovascular morbidity, but the pattern of selected inflammatory and cardiac biomarkers among people with and without diabetes may vary across populations. This study compared selected biomarkers between participants with and without diabetes mellitus.

- ***Methods:***

This cross-sectional comparative study included 133 clinic attendees in Gaborone, Botswana: 71 with diabetes and 62 without diabetes. Biomarkers measured were high-sensitivity C-reactive protein (hs-CRP), creatine kinase (CK), CK-MB, troponin I and N-terminal pro-B-type natriuretic peptide (NT-proBNP). Continuous variables were summarised using medians and interquartile ranges (IQRs) and compared using Mann–Whitney U tests. Holm adjustment was applied across five biomarker comparisons. Adjusted associations were examined using separate log-linear regression models for each biomarker, controlling for age, sex, recalculated body mass index and systolic blood pressure.

- ***Results:***

Participants with diabetes were older than those without diabetes (median 56.0 [IQR 43.5–66.0] vs 44.0 [30.5–57.0] years; p=0.001) and had higher systolic blood pressure (140 [134–144] vs 136 [128.25–142] mmHg; p=0.028). Median hs-CRP was higher in the diabetes group (9.04 [2.87–18.53] vs 2.48 [1.68–6.04]; p less than 0.001; Holm-adjusted p less than 0.001). CK, CK-MB, troponin I and NT-proBNP did not differ significantly between groups. After adjustment, hs-CRP remained higher in the diabetes group (adjusted geometric mean ratio 2.41, 95% confidence interval 1.56 to 3.72; Holm-adjusted p less than 0.001); adjusted differences in the other biomarkers were not statistically significant.

- ***Conclusion:***

In this clinic-based sample, diabetes status was associated with higher hs-CRP, consistent with greater systemic inflammation. The cross-sectional design and absence of a confirmed cardiovascular disease endpoint mean these findings do not establish cardiovascular disease or predict future cardiovascular events.

Keywords: *Cardiovascular Biomarkers; Diabetes Mellitus; hs-CRP; Inflammation; NT-proBNP; Troponin I.*

How to Cite: Chinakila J. Musonda; Brian Mukuka; Bislom C. Mweene; Benson M. Hamooya; Sepiso K. Masenga; Badisa John Phafane (2026) Biomarkers of Cardiovascular Injury, Cardiac Stress and Inflammation in Adults with and Without Diabetes Mellitus: A Comparative Study. *International Journal of Innovative Science and Research Technology*, 11(10), 2237-2242. https://doi.org/10.38124/ijisrt/26oct1004

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

I. INTRODUCTION

Diabetes mellitus is a major non-communicable disease and is associated with an increased burden of cardiovascular disease. International estimates have documented a substantial and growing number of adults living with diabetes, while epidemiological studies consistently identify diabetes as an important cardiovascular risk factor [1–3,7]. The mechanisms linking diabetes and cardiovascular disease include metabolic dysfunction, vascular injury, oxidative stress and chronic low-grade inflammation [4,5,7,14–18].

Biomarkers can provide complementary information about different biological processes. High-sensitivity C-reactive protein (hs-CRP) is a marker of systemic inflammation and has been studied in relation to vascular dysfunction and atherosclerotic processes [8–13,19]. Cardiac troponins are used to identify myocardial injury, whereas creatine kinase (CK) and its MB fraction (CK-MB) are enzymes that may rise with muscle injury but are not equally specific for cardiac injury. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is associated with myocardial wall stress and is used in the assessment of heart failure and related cardiac strain [6,20]. No single biomarker alone confirms cardiovascular disease in the absence of clinical assessment.

Although diabetes is associated with cardiovascular complications, biomarker patterns can differ according to the population, comorbidities and clinical context. Evidence from African populations has also highlighted the relevance of inflammatory markers to vascular dysfunction among people with and without diabetes [19,20]. However, biomarker differences should be distinguished from a confirmed cardiovascular diagnosis or validated prediction of future events.

This study therefore compared selected biomarkers of inflammation, cardiac stress and possible cardiac injury between participants with and without diabetes mellitus attending clinics in Gaborone, Botswana. The primary exposure was diabetes status, and the primary outcomes were hs-CRP, CK, CK-MB, troponin I and NT-proBNP concentrations. Secondary analyses examined whether differences in biomarker concentrations persisted after adjustment for age, sex, recalculated body mass index (BMI) and systolic blood pressure.

II. MATERIALS AND METHODS

➢ *Study Design and Setting*

This was a cross-sectional comparative study conducted in Gaborone, Botswana, among attendees of the Gaborone Diabetes Clinic and the Adventist Health Services Clinic. A total of 133 participants were included: 71 with diabetes mellitus and 62 without diabetes. Participants were recruited using convenience sampling because of the limited number of eligible attendees during the study period.

➢ *Eligibility Criteria*

Individuals with or without diabetes who were able to understand the study procedures and provided informed consent were eligible. Individuals described in the original protocol as psychotic or lacking capacity to provide informed consent were excluded. The supplied dataset included participants aged 15–88 years.

➢ *Data Collection and Laboratory Measurements*

Sociodemographic, lifestyle and symptom information was collected using a structured interviewer-administered questionnaire. Recorded variables included age, sex, education, employment, residence, alcohol intake, smoking, physical activity/sedentary lifestyle and self-reported symptoms. Height, weight and blood pressure were measured. BMI was recalculated as weight in kilograms divided by height in metres squared because the BMI field in the supplied spreadsheet contained discrepancies with the recorded height and weight. The source height and weight measurements were not altered.

Blood samples were collected in serum-separator tubes and transported to Diagnofirm Medical Laboratories for analysis. The original manuscript states that hs-CRP, CK, CK-MB, troponin I and NT-proBNP were measured using an Alinity ci analyser. D-dimer, HbA1c and sST2 were not included in the analyses for this study. The supplied spreadsheets did not contain complete unit metadata or assay-specific reference intervals; these should be confirmed against the laboratory records before final submission.

➢ *Statistical Analysis*

Data were analysed using Python-based statistical procedures. Continuous variables were summarised as medians and interquartile ranges (IQRs), and between-group comparisons used two-sided Mann–Whitney U tests. Categorical variables were summarised as counts and percentages and compared using Pearson chi-square tests; Fisher's exact test was used for sparse 2×2 comparisons. Holm adjustment was applied to the five primary biomarker comparisons to account for multiple testing.

For adjusted analyses, each biomarker was modelled separately as the outcome after natural-log transformation. Linear regression models included diabetes status (with the diabetes group coded 1 and the non-diabetes group coded 0), age, sex, recalculated BMI and systolic blood pressure. Heteroscedasticity-consistent HC3 standard errors were used. Exponentiated coefficients for diabetes status are presented as adjusted geometric mean ratios (GMRs) with 95% confidence intervals (CIs). Holm adjustment was also applied across the five adjusted biomarker models. All hs-CRP and NT-proBNP observations, including extreme values confirmed by the study author, were retained. A p-value <0.05 was considered statistically significant, with interpretation also considering multiple-testing adjustment and the study design.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

➢ *Ethical Considerations*

Ethical approval was obtained from the Mulungushi University Research Ethics Committee (SMHS-MU2-2022-48) on 12 January 2024. Additional clearances were reported from the Zambia National Health Research Authority on 7 March 2024, the Botswana Ministry of Health and Wellness on 24 May 2024, and the Princess Marina Ethics Committee on 10 July 2024. The original manuscript states that approvals were granted for one year and that all participants provided written informed consent before recruitment. Data were de-identified for analysis.

III. RESULTS

➢ *Participant Characteristics*

The analysis included 133 participants, comprising 71 participants with diabetes and 62 without diabetes. Participants with diabetes were older (median 56.0 years, IQR 43.5–66.0) than those without diabetes (44.0 years, IQR 30.5–57.0; p=0.001). Median recalculated BMI did not differ significantly between groups. Median systolic blood pressure was higher in the diabetes group, whereas diastolic blood pressure did not differ significantly. Alcohol intake and smoking distributions differed between groups. Detailed selected characteristics are shown in Table 1.

Table 1 Selected Demographic, Clinical and Lifestyle Characteristics by Diabetes Status

Characteristic	Diabetes (n=71)	No diabetes (n=62)	p-value
Age, years	56.00 (43.50–66.00)	44.00 (30.50–57.00)	0.001
BMI, kg/m² (recalculated)	30.49 (27.58–33.41)	28.71 (26.70–33.40)	0.290
Systolic blood pressure, mmHg	140.00 (134.00–144.00)	136.00 (128.25–142.00)	0.028
Diastolic blood pressure, mmHg	90.00 (80.00–99.00)	88.00 (80.00–92.50)	0.113
Sex, male, n (%)	39 (54.9%)	31 (50.0%)	0.570
Employed, n (%)	57 (80.3%)	40 (64.5%)	0.041 (χ^2); Fisher 0.051
Urban residence, n (%)	49 (69.0%)	48 (77.4%)	0.276
Alcohol intake distribution, p-value	—	—	0.008
Current	19 (26.8%)	26 (41.9%)	
Former	9 (12.7%)	15 (24.2%)	
Never	43 (60.6%)	21 (33.9%)	
Smoking status distribution, p-value	—	—	0.006
Current	2 (2.8%)	11 (17.7%)	
Former	14 (19.7%)	16 (25.8%)	
Never	55 (77.5%)	35 (56.5%)	
Physically active, n (%)	26 (36.6%)	30 (48.4%)	0.170

Values are median (IQR) or n (%). Continuous variables were compared using Mann–Whitney U tests and categorical variables using Pearson chi-square tests. Employment status was borderline in a sensitivity comparison (Pearson χ^2 p=0.041; Fisher exact p=0.051). BMI was recalculated from recorded height and weight.

➢ *Biomarker Comparisons*

Median hs-CRP was higher in participants with diabetes than in those without diabetes (9.04, IQR 2.87–18.53 versus 2.48, IQR 1.68–6.04; unadjusted p<0.001; Holm-adjusted p<0.001). There were no statistically significant between-group differences in CK, CK-MB, troponin I or NT-proBNP. The extreme hs-CRP and NT-proBNP values were retained as provided and confirmed by the study author. Table 2 summarises the biomarker comparisons.

Table 2 Biomarker Concentrations by Diabetes Status

Biomarker	Diabetes, median (IQR)	No diabetes, median (IQR)	p-value	Holm-adjusted p
hs-CRP	9.04 (2.87–18.53)	2.48 (1.68–6.04)	<0.001	<0.001
CK	109.00 (75.00–174.00)	99.50 (82.75–139.75)	0.660	1.000
CK-MB	1.20 (0.70–2.40)	1.00 (0.70–2.22)	0.984	1.000
Troponin I	1.70 (1.50–20.70)	2.35 (1.50–15.20)	0.933	1.000
NT-proBNP	79.00 (24.00–315.50)	118.00 (66.00–146.75)	0.519	1.000

Biomarker units were not consistently specified in the supplied data extract and should be verified against laboratory records before submission. Holm adjustment was applied to the five biomarker comparisons.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

➢ *Adjusted Association Between Diabetes Status and Biomarker Concentrations*

In adjusted log-linear models controlling for age, sex, recalculated BMI and systolic blood pressure, hs-CRP was estimated to be 2.41 times as high in the diabetes group as in the non-diabetes group (95% CI 1.56–3.72; p<0.001; Holm-adjusted p<0.001). Adjusted differences in CK, CK-MB, troponin I and NT-proBNP were not statistically significant after adjustment. Results are presented in Table 3 as adjusted geometric mean ratios.

Table 3 Adjusted Geometric Mean Ratios for Biomarker Concentrations Associated with Diabetes Status

Biomarker outcome	Adjusted GMR	95% CI	p-value	Holm-adjusted p
hs-CRP	2.41	1.56–3.72	<0.001	<0.001
CK	1.20	0.93–1.53	0.156	0.626
CK-MB	0.78	0.47–1.29	0.334	1.000
Troponin I	1.11	0.68–1.81	0.670	1.000
NT-proBNP	1.15	0.72–1.84	0.554	1.000

Models used natural-log-transformed biomarker concentrations as outcomes and included diabetes status, age, sex, recalculated BMI and systolic blood pressure. GMR >1 indicates a higher adjusted geometric mean in the diabetes group. HC3 robust standard errors were used.

IV. DISCUSSION

The principal finding was a higher hs-CRP concentration among participants with diabetes compared with those without diabetes. This difference remained statistically significant after adjustment for age, sex, BMI and systolic blood pressure and after correction for multiple comparisons. In contrast, CK, CK-MB, troponin I and NT-proBNP did not differ significantly between groups in the primary rank-based comparisons or in the adjusted models. The results therefore support an association between diabetes status and systemic inflammation in this clinic-based sample, but they do not demonstrate a higher prevalence of cardiovascular disease or establish that cardiac injury was present.

The higher hs-CRP observed in the diabetes group is biologically plausible because diabetes and related metabolic disturbances are associated with chronic low-grade inflammation. Prior literature has discussed inflammatory pathways in atherosclerosis and diabetes-related vascular dysfunction [4,5,8–13,19]. Nevertheless, hs-CRP is a nonspecific inflammatory marker. Its elevation cannot, by itself, diagnose coronary artery disease or determine an individual's future cardiovascular event risk. Other inflammatory conditions and comorbidities may also influence hs-CRP concentrations.

The absence of statistically significant differences in NT-proBNP, troponin I, CK and CK-MB should be interpreted cautiously. NT-proBNP reflects myocardial wall stress rather than myocardial injury alone, and its concentration may be influenced by age, renal function, cardiac status and other clinical factors. Troponin is primarily interpreted in a clinical context and with assay-specific thresholds; a single measurement without a defined clinical endpoint cannot establish or exclude all forms of cardiac disease [6,20]. CK and CK-MB are also not fully specific to the heart. In this dataset, no independently confirmed cardiovascular diagnosis or longitudinal cardiovascular event was available, so the study cannot estimate cardiovascular disease prevalence or validate biomarker-based prediction.

Participants with diabetes were older and had higher systolic blood pressure. Age is an established correlate of diabetes and cardiovascular burden, and blood pressure is clinically relevant to cardiovascular risk [3,7,14–18]. The regression models adjusted for age, sex, recalculated BMI and systolic blood pressure to account for selected differences between groups. Residual confounding remains possible because other factors, including renal function, lipid levels, glycaemic control, diabetes duration and treatment, were not included in the adjusted biomarker models. The study was not designed to establish causal pathways.

The recalculation of BMI was necessary because the supplied BMI field was inconsistent with height and weight for several records, including one implausibly high recorded value. Recalculated BMI was used in the analysis, while source height and weight values were preserved. The affected records should be checked against the original case-report forms or measurement records as part of data verification. Biomarker units and assay-specific reference intervals should also be confirmed with the laboratory before the manuscript is submitted.

➢ *Strengths and Limitations*

Strengths include the use of a non-diabetic comparison group, measurement of multiple biomarkers reflecting different biological processes, and adjustment for selected demographic and clinical covariates. The analyses retained all reported hs-CRP and NT-proBNP values after the study author confirmed that the extreme values were correct; skewed biomarker outcomes were log-transformed for adjusted modelling rather than deleting observations.

Several limitations should be acknowledged. The cross-sectional design does not establish temporality or causation. Convenience sampling from two clinic settings may limit generalisability and may introduce selection differences between groups. The sample size was modest, particularly for adjusted models with several covariates. No confirmed cardiovascular disease diagnosis, renal function measure, lipid profile or longitudinal cardiovascular outcome was available in the analysis dataset. Biomarker units and assay-specific reference intervals were not fully documented in the supplied spreadsheet. Finally, inconsistencies in the original

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

BMI field required recalculation from height and weight, and source measurements for affected records should be verified.

V. CONCLUSION

Among participants attending clinics in Gaborone, hs-CRP concentrations were higher in participants with diabetes than in those without diabetes, and this difference persisted after adjustment for age, sex, recalculated BMI and systolic blood pressure. CK, CK-MB, troponin I and NT-proBNP did not show statistically significant between-group differences. These findings suggest an association between diabetes status and systemic inflammation in this sample, but they do not establish cardiovascular disease or predict future cardiovascular events. Larger studies with verified assay units, clinically confirmed cardiovascular outcomes and longitudinal follow-up are needed to determine the clinical implications of these biomarker patterns.

ACKNOWLEDGMENT

The authors thank the lecturers of the Mulungushi University School of Medicine and Health Sciences, including Dr Benson Hamooya and Prof Sepiso Masenga, and Mr Bislom C. Mweene, who are affiliated with the HANDS research group. The authors also acknowledge the Botswana Ministry of Health and Wellness for support and ethical clearance, and Diagnofirm Medical Laboratories, Botswana, for performing the laboratory tests.

- *Author Contributions*

Conceptualization: Chinakila J.C. Musonda and Sepiso K. Masenga. Data curation: Chinakila J.C. Musonda, Sepiso K. Masenga and Benson M. Hamooya. Formal analysis: Chinakila J.C. Musonda, Sepiso K. Masenga and Benson M. Hamooya. Investigation, methodology, project administration, resources, software and visualization: Chinakila J.C. Musonda. Supervision: Sepiso K. Masenga. Validation: Chinakila J.C. Musonda, Sepiso K. Masenga and Benson M. Hamooya. Writing—original draft: Chinakila J. Musonda. Writing—review and editing: Sepiso K. Masenga, Bislom C. Mweene, Chinakila J.C. Musonda, Brian Mukuka and Badisa John Phafane.

- *Ethics Approval And Consent To Participate*

Ethical approval and additional national/site clearances are detailed in Section 2.5. The original study report states that written informed consent was obtained from all participants before recruitment.

REFERENCES

[1]. Home, Resources, diabetes L with, Acknowledgement, FAQs, Contact, et al. 9th edition | IDF Diabetes Atlas [Internet]. [cited 2025 Jan 24]. Available from: https://diabetesatlas.org/atlas/ninth-edition/

[2]. Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017. Cardiovasc Diabetol [Internet]. 2018 Jun 8 [cited 2025 Jan 27];17:83. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5994068/

[3]. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics—2020 Update: A Report From the American Heart Association. Circulation [Internet]. 2020 Mar 3 [cited 2025 Jan 27];141(9):e139–596. Available from: https://www.ahajournals.org/doi/full/10.1161/CIR.0000000000000757

[4]. Libby P, Loscalzo J, Ridker P, Farkouh ME, Hsue PY, Fuster V, et al. Inflammation, Immunity, and Infection in Atherothrombosis: JACC Review Topic of the Week. J Am Coll Cardiol [Internet]. 2018 Oct 23 [cited 2025 Jan 27];72(17):2071–81. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6196735/

[5]. The Emerging Risk Factors Collaboration. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. Lancet [Internet]. 2010 Jun 26 [cited 2025 Jan 29];375(9733):2215–22. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2904878/

[6]. Apple FS, Collinson PO, for the IFCC Task Force on Clinical Applications of Cardiac Biomarkers. Analytical Characteristics of High-Sensitivity Cardiac Troponin Assays. Clinical Chemistry [Internet]. 2012 Jan 1 [cited 2025 Jan 29];58(1):54–61. Available from: https://doi.org/10.1373/clinchem.2011.165795

[7]. Dal Canto E, Ceriello A, Rydén L, Ferrini M, Hansen TB, Schnell O, et al. Diabetes as a cardiovascular risk factor: An overview of global trends of macro and micro vascular complications. European Journal of Preventive Cardiology [Internet]. 2019 Dec 1 [cited 2025 Jan 31];26(2_suppl):25–32. Available from: https://doi.org/10.1177/2047487319878371

[8]. Willerson JT, Ridker PM. Inflammation as a Cardiovascular Risk Factor. Circulation [Internet]. 2004 Jun [cited 2025 Jan 29];109(21_suppl_1):II–2. Available from: https://www.ahajournals.org/doi/10.1161/01.CIR.0000129535.04194.38

[9]. Li L, Roumeliotis N, Sawamura T, Renier G. C-reactive protein enhances LOX-1 expression in human aortic endothelial cells: relevance of LOX-1 to C-reactive protein-induced endothelial dysfunction. Circ Res. 2004 Oct 29;95(9):877–83.

[10]. Scherer PE, Hill JA. Obesity, Diabetes, and Cardiovascular Diseases: A Compendium. Circ Res [Internet]. 2016 May 27 [cited 2025 Jan 31];118(11):1703–5. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4888905/

[11]. Rohm TV, Meier DT, Olefsky JM, Donath MY. Inflammation in Obesity, Diabetes and related Disorders. Immunity [Internet]. 2022 Jan 11 [cited 2025 Jan 29];55(1):31–55. Available from:

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26oct1004

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8773457/

[12]. de Luca C, Olefsky JM. Inflammation and Insulin Resistance. FEBS Lett [Internet]. 2008 Jan 9 [cited 2025 Jan 29];582(1):97–105. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2246086/

[13]. Zatterale F, Longo M, Naderi J, Raciti GA, Desiderio A, Miele C, et al. Chronic Adipose Tissue Inflammation Linking Obesity to Insulin Resistance and Type 2 Diabetes. Front Physiol [Internet]. 2020 Jan 29 [cited 2025 Jan 29];10:1607. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7000657/

[14]. Matheus AS de M, Tannus LRM, Cobas RA, Palma CCS, Negrato CA, Gomes M de B. Impact of Diabetes on Cardiovascular Disease: An Update. Int J Hypertens [Internet]. 2013 [cited 2025 Jan 30];2013:653789. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3603160/

[15]. Leon BM, Maddox TM. Diabetes and cardiovascular disease: Epidemiology, biological mechanisms, treatment recommendations and future research. World J Diabetes [Internet]. 2015 Oct 10 [cited 2025 Jan 30];6(13):1246–58. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4600176/

[16]. Borén J, Öörni K, Catapano AL. The link between diabetes and cardiovascular disease. Atherosclerosis [Internet]. 2024 Jul 1 [cited 2025 Jan 30];394. Available from: https://www.atherosclerosis-journal.com/article/S0021-9150(24)00167-9/fulltext

[17]. Petrie JR, Guzik TJ, Touyz RM. Diabetes, Hypertension, and Cardiovascular Disease: Clinical Insights and Vascular Mechanisms. Can J Cardiol [Internet]. 2018 May [cited 2025 Jan 30];34(5):575–84. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5953551/

[18]. Martín-Timón I, Sevillano-Collantes C, Segura-Galindo A, del Cañizo-Gómez FJ. Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? World J Diabetes [Internet]. 2014 Aug 15 [cited 2025 Jan 30];5(4):444–70. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4127581/

[19]. Hayfron-Benjamin CF, Maitland-van der Zee AH, van den Born BJ, Amoah AGB, Meeks KAC, Klipstein-Grobusch K, et al. Association between C reactive protein and microvascular and macrovascular dysfunction in sub-Saharan Africans with and without diabetes: the RODAM study. BMJ Open Diabetes Res Care [Internet]. 2020 Jul 14 [cited 2025 Jan 31];8(1):e001235. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7365428/

[20]. Bachmann KN, Wang TJ. Biomarkers of cardiovascular disease: contributions to risk prediction in individuals with diabetes. Diabetologia [Internet]. 2018 May [cited 2025 Jan 31];61(5):987–95. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5874155/