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RESEARCH ARTICLE

Assessment of Cardiovascular Risk Factors in Prediabetic Subjects and Their Association with HbA1c

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Abstract

Background: Prediabetes is a high-risk intermediate metabolic state characterised by impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT), which significantly increases the risk of developing both type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). Glycated haemoglobin (HbA1c) serves as a valuable surrogate marker of glycaemic exposure and has been increasingly linked to subclinical cardiovascular risk. This study aimed to assess cardiovascular risk factors in prediabetic subjects and evaluate their association with HbA1c levels.

Methods: A hospital-based case-control study was conducted involving 100 prediabetic cases (HbA1c 5.7–6.4% or FPG 100–125 mg/dL) and 100 age- and sex-matched normoglycemic controls. Detailed anthropometric measurements, lipid profiles, blood pressure, and Framingham 10-year CVD risk scores were assessed. Pearson's correlation and binary logistic regression analyses were applied.

Results: Prediabetic subjects demonstrated significantly elevated systolic blood pressure (132.4 ± 14.2 vs. 118.6 ± 11.4 mmHg; $p<0.001$), higher LDL-C (138.4 ± 28.6 vs. 108.7 ± 23.5 mg/dL; $p<0.001$), reduced HDL-C (38.6 ± 7.2 vs. 48.4 ± 8.9 mg/dL; $p<0.001$), and elevated triglycerides compared to controls. HbA1c showed a significant positive correlation with total cholesterol ($r=0.52$), triglycerides ($r=0.46$), systolic blood pressure ($r=0.48$), and Framingham CVD risk score ($r=0.58$).

Conclusion: Prediabetes is associated with a significantly higher burden of cardiovascular risk factors even before the onset of overt diabetes. HbA1c demonstrates a strong positive correlation with multiple CVD risk markers, reinforcing its utility as a screening tool for early cardiovascular risk stratification.

Key words: Prediabetes, cardiovascular risk factors, HbA1c, dyslipidaemia, hypertension, case-control study, Framingham risk score, insulin resistance

1 | INTRODUCTION

Prediabetes is defined by the American Diabetes Association (ADA) as an intermediate glycaemic state between normal glucose homeostasis and overt type 2 diabetes mellitus. It encompasses impaired fasting glucose (IFG; fasting plasma glucose 100–125 mg/dL), impaired glucose tolerance (IGT; 2-hour post-load glucose 140–199 mg/dL), or a glycated haemoglobin (HbA1c) value between 5.7% and 6.4% (1). Globally, the prevalence of prediabetes has reached epidemic proportions, with the International Diabetes Federation estimating approximately 541 million affected adults in 2021, a figure projected to exceed 730 million by 2045 (2).

Despite its classification as a 'pre-disease' state, accumulating evidence indicates that prediabetes

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carries significant clinical consequences beyond the risk of progression to overt diabetes. Several large-scale epidemiological studies have demonstrated that cardiovascular morbidity and mortality are substantially elevated in prediabetic individuals, independent of conventional risk factors (3, 4). The pathophysiological mechanisms underlying this excess cardiovascular risk include chronic low-grade inflammation, oxidative stress, endothelial dysfunction, hypercoagulability, and insulin resistance — all of which are operative in the prediabetic state (5).

HbA1c, or glycated haemoglobin, reflects average blood glucose concentrations over the preceding 2–3 months. It is now established as a diagnostic criterion for both prediabetes and diabetes by major international organisations including the ADA and the World Health Organization (WHO) (1, 6). Beyond its glycaemic significance, HbA1c has emerged as a potential marker for cardiovascular risk. Studies suggest a graded association between HbA1c levels and adverse cardiovascular outcomes, even within the non-diabetic range (7, 8). However, the precise relationship between HbA1c and specific cardiovascular risk factors in a prediabetic South Asian population remains inadequately characterised.

The clustering of multiple cardiovascular risk factors — including dyslipidaemia, hypertension, central obesity, and prothrombotic states — in prediabetic subjects constitutes a significant public health challenge. Early identification and intervention during the prediabetic phase represent a critical therapeutic window to prevent or delay both diabetes and cardiovascular disease. The Framingham Risk Score (FRS) offers a validated, composite tool to estimate 10-year CVD risk based on measurable clinical parameters and has been applied in this study to provide a comprehensive risk assessment (9).

Given the paucity of dedicated case-control studies examining the full spectrum of cardiovascular risk factors in prediabetic individuals and their correlation with HbA1c, the present study was designed to address this gap. The objectives were to: (1) compare the prevalence and magnitude of cardiovascular risk factors between prediabetic subjects and normoglycemic controls; (2) determine the correlation between HbA1c and individual cardiovascular risk parameters; and (3) evaluate the Framingham 10-year CVD risk score across the two groups.

2 | MATERIALS AND METHODS

2.1 | Study Design and Setting

This was a hospital-based case-control study conducted over a period of 6 months at a tertiary care teaching hospital. The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants prior to enrolment. The study adhered to the principles of the Declaration of Helsinki (10).

2.2 | Study Participants

A total of 200 participants were enrolled: 100 prediabetic cases and 100 normoglycemic controls. Cases were recruited from the outpatient departments of Medicine and Endocrinology. Controls were age- and sex-matched healthy volunteers from the same catchment population.

Inclusion Criteria for Cases: Adults aged 30–65 years with confirmed prediabetes (HbA1c 5.7–6.4% and/or FPG 100–125 mg/dL on two occasions, as per ADA criteria) (1). **Inclusion Criteria for Controls:** Age and sex matched individuals with HbA1c <5.7% and FPG <100 mg/dL on two successive measurements.

Exclusion Criteria (Both Groups): established type 2 diabetes mellitus, known coronary artery disease, heart failure, renal disease (eGFR <60 mL/min/1.73m^2), hepatic disease, anaemia (haemoglobin <10 g/dL), use of lipid-lowering or antihypertensive medications that may confound lipid or blood pressure measurements, pregnancy, or any acute illness.

2.3 | Clinical and Anthropometric Assessment

A standardised questionnaire recorded demographic data, lifestyle habits (smoking status, physical activity, dietary patterns), family history of diabetes, and personal medical history. Body weight, height, and waist circumference were measured using calibrated instruments. Body mass index (BMI) was calculated as weight (kg)/height (m^2). Blood pressure was measured twice in the right arm after 10 minutes of seated rest using a mercury sphygmomanometer, and the mean of both readings was recorded.

2.4 | Laboratory Investigations

Venous blood samples were collected after an overnight fast of 10–12 hours. The following parameters were measured: fasting plasma glucose (FPG) by glucose oxidase method; HbA1c by high-performance liquid chromatography (HPLC); total cholesterol, HDL cholesterol, and triglycerides by enzymatic colorimetric methods on an automated analyser (Beckman Coulter AU480); LDL cholesterol was calculated using the Friedewald equation [LDL-C = Total Cholesterol − HDL-C − Triglycerides/5] (11); serum creatinine, urine albumin-to-creatinine ratio (ACR), and high-sensitivity C-reactive protein (hs-CRP) by nephelometry.

2.5 | Cardiovascular Risk Assessment

The 10-year cardiovascular disease risk was estimated using the Framingham Risk Score (FRS), incorporating age, sex, total cholesterol, HDL-C, systolic blood pressure, antihypertensive treatment status, and smoking history (9). Dyslipidaemia was defined according to National Cholesterol Education Program (NCEP-ATP III) criteria: total cholesterol ≥200 mg/dL, LDL-C ≥130 mg/dL, HDL-C <40 mg/dL (men)/<50 mg/dL (women), or triglycerides ≥150 mg/dL (12). Hypertension was defined as blood pressure ≥130/80 mmHg (AHA/ACC 2017 guidelines) (13).

2.6 | Statistical Analysis

Data were analysed using IBM SPSS Statistics version 26.0 (SPSS Inc., Chicago, IL, USA). Continuous variables are expressed as mean ± standard deviation (SD) and categorical variables as frequency and percentage. Independent samples t-test and Mann-Whitney U test were applied for normally and non-normally distributed continuous variables, respectively. Chi-square test was used for categorical variables. Pearson's correlation coefficient (r) was used to assess the association between HbA1c and continuous cardiovascular risk variables. Binary logistic regression was performed to identify independent predictors of prediabetes. A p-value of <0.05 was considered statistically significant.

3 | RESULTS

3.1 | Baseline Characteristics

The two groups were comparable in terms of age and sex distribution. Prediabetic subjects had significantly higher BMI, HbA1c, and FPG, and a greater prevalence of family history of diabetes mellitus compared to controls Table 1 .

Table 1. Baseline Demographic and Glycaemic Characteristics of Cases and Controls

Characteristic	Cases (Prediabetics) n=100	Controls (Normoglycemic) n=100	p-value
Age (years), Mean ± SD	44.8 ± 8.3	43.5 ± 7.9	0.21
Male (%)	58 (58%)	55 (55%)	0.64
BMI (kg/m^2), Mean ± SD	27.6 ± 3.8	23.4 ± 3.1	<0.001
HbA1c (%), Mean ± SD	6.1 ± 0.3	5.2 ± 0.2	<0.001
FPG (mg/dL), Mean ± SD	108.4 ± 9.6	88.3 ± 7.1	<0.001
Family history of DM (%)	44 (44%)	21 (21%)	<0.001

3.2 | Cardiovascular Risk Factor Profile

Prediabetic subjects demonstrated a significantly adverse cardiovascular risk profile compared to normoglycemic controls across all major parameters. The prevalence of hypertension was 48% in cases versus 22% in controls (p<0.001), and dyslipidaemia was detected in 62% of cases versus 31% of controls (p<0.001). Mean systolic blood pressure, LDL-C, and triglycerides were significantly higher in prediabetics, while HDL-C was significantly lower Table 2

Table 2. Comparison of Cardiovascular Risk Factors between Prediabetic Cases and Normoglycemic Controls

CVD Risk Factor	Cases (Prediabetics)	Controls (Normoglycemic)	p-value
SBP (mmHg), Mean ± SD	132.4 ± 14.2	118.6 ± 11.4	<0.001
DBP (mmHg), Mean ± SD	84.7 ± 9.3	76.3 ± 8.1	<0.001
Total Cholesterol (mg/dL)	214.6 ± 32.8	182.3 ± 27.4	<0.001
LDL-C (mg/dL)	138.4 ± 28.6	108.7 ± 23.5	<0.001
HDL-C (mg/dL)	38.6 ± 7.2	48.4 ± 8.9	<0.001
Triglycerides (mg/dL)	172.8 ± 44.6	124.3 ± 36.2	<0.001
Hypertension (%)	48 (48%)	22 (22%)	<0.001
Dyslipidaemia (%)	62 (62%)	31 (31%)	<0.001
Current Smokers (%)	29 (29%)	18 (18%)	0.07

The mean 10-year Framingham CVD Risk Score was significantly higher in prediabetic subjects (14.8 ± 6.2%) compared to controls (8.3 ± 4.1%; p<0.001), indicating a higher probability of a major cardiovascular event over the subsequent decade.

3.3 | Correlation of HbA1c with Cardiovascular Risk Factors

Pearson's correlation analysis within the prediabetic group revealed significant positive correlations between HbA1c and systolic blood pressure, total cholesterol, LDL-C, triglycerides, BMI, and the Framingham CVD risk score. A significant negative correlation was observed between HbA1c and HDL-C Table 3

Table 3. Pearson's Correlation of HbA1c with Cardiovascular Risk Factors in Prediabetic Subjects

CVD Risk Factor	Pearson's r	p-value
Systolic Blood Pressure	0.48	<0.001
Diastolic Blood Pressure	0.41	<0.001
Total Cholesterol	0.52	<0.001
LDL Cholesterol	0.44	<0.001
HDL Cholesterol	-0.39	<0.001
Triglycerides	0.46	<0.001
BMI	0.51	<0.001
10-year Framingham CVD Risk Score	0.58	<0.001

The strongest correlations were observed with the 10-year Framingham CVD risk score (r=0.58, p<0.001) and total cholesterol (r=0.52, p<0.001), suggesting that higher HbA1c values within the prediabetic range are associated with a progressively greater cardiovascular risk burden.

3.4 | Logistic Regression Analysis

On binary logistic regression analysis with prediabetes as the outcome variable, BMI (OR: 2.14; 95% CI: 1.56–2.93; p<0.001), LDL-C (OR: 1.82; 95% CI: 1.34–2.47; p<0.001), family history of DM (OR: 2.88; 95% CI: 1.63–5.10; p<0.001), and hypertension (OR: 2.21; 95% CI: 1.28–3.82; p=0.004) emerged as significant independent predictors of prediabetes.

4 | DISCUSSION

The present case-control study provides compelling evidence that cardiovascular risk factors are significantly more prevalent and severe in prediabetic subjects compared to normoglycemic controls, even after controlling for age and sex. Furthermore, HbA1c demonstrated significant positive correlations with all major cardiovascular risk variables

examined, supporting its role not only as a glycaemic marker but also as a cardiovascular risk indicator.

Our finding of significantly higher BMI in the prediabetic group is consistent with the well-established association between adiposity and insulin resistance. Central obesity promotes ectopic fat deposition and impairs insulin signalling, leading to compensatory hyperinsulinemia and dysglycaemia (14). A meta-analysis by Bellou et al. confirmed obesity as one of the most powerful modifiable risk factors for incident type 2 diabetes (15). The substantially higher proportion of individuals with family history of diabetes among cases (44% vs. 21%) underscores the contribution of genetic susceptibility to prediabetes, a finding corroborated by Tuomilehto et al. (16).

The significantly elevated blood pressure observed in our prediabetic cohort aligns with existing literature demonstrating that hypertension and insulin resistance frequently coexist through shared mechanisms, including sodium retention, sympathetic nervous system activation, and impaired endothelial nitric oxide synthesis (17). The DECODE study established that IGT is independently associated with increased cardiovascular mortality, and elevated blood pressure was a key mediating factor (18). Our finding that 48% of prediabetic subjects had hypertension compared to only 22% of controls ($p<0.001$) highlights the need for blood pressure monitoring in this population.

The atherogenic dyslipidaemia characterised by elevated LDL-C, elevated triglycerides, and reduced HDL-C observed in prediabetic subjects in our study closely mirrors the metabolic dyslipidaemia described in insulin-resistant states. The strong correlation between HbA1c and total cholesterol ($r=0.52$) and LDL-C ($r=0.44$) is particularly noteworthy. Elevated HbA1c in the prediabetic range is associated with increased hepatic VLDL synthesis, impaired lipolytic enzyme activity, and reduced LDL receptor expression, collectively fostering an atherogenic lipoprotein profile (19). Similar findings were reported by Botas et al. in a Spanish population, where HbA1c correlated significantly with total cholesterol and triglycerides in non-diabetic adults (20).

The significant negative correlation between HbA1c and HDL-C ($r = -0.39$) observed in this study is consistent with prior evidence that glycation impairs HDL particle functionality and promotes its catabolism. HDL-C plays a critical protective role in reverse cholesterol transport; its reduction in prediabetes thus represents a quantifiable increase in atherogenic risk (21). The Atherosclerosis Risk in Communities (ARIC) study demonstrated that lower HDL-C was significantly associated with insulin resistance and predicted incident coronary heart disease (22).

One of the most clinically significant findings of our study is the substantially elevated mean Framingham 10-year CVD risk score in prediabetic subjects (14.8%) compared to controls (8.3%), and its strong correlation with HbA1c ($r=0.58$). This composite risk index aggregates multiple individual risk factors and provides a validated probabilistic estimate of hard CVD events. The finding that HbA1c — even within the prediabetic range — correlates strongly with this composite measure suggests that each incremental rise in HbA1c, though sub-threshold for diabetes, contributes meaningfully to overall cardiovascular risk. Khaw et al. reported that every 1% rise in HbA1c was associated with an increased risk of cardiovascular events, independent of other risk factors (7).

From a mechanistic perspective, the association between HbA1c and CVD risk in prediabetes is mediated through several pathways. Chronic hyperglycaemia promotes glycation of structural proteins such as collagen and elastin, leading to arterial stiffness and increased cardiovascular events (23). Advanced glycation end-products (AGEs) activate their specific receptor (RAGE), triggering nuclear factor-kappa B (NF-kB) signalling, endothelial dysfunction, and pro-inflammatory cytokine release (24). Oxidative stress, as evidenced by elevated hs-CRP levels in our cases, further amplifies endothelial damage and accelerates atherosclerotic plaque formation (25).

Our logistic regression findings identified BMI, LDL-C, hypertension, and family history of diabetes as independent predictors of prediabetes, collectively highlighting the multifactorial nature of this condition. These factors represent actionable targets for lifestyle and pharmacological intervention. The Diabetes Prevention Program (DPP) conclusively demonstrated that intensive lifestyle modifi-

cation can reduce the incidence of type 2 diabetes by 58% and may also attenuate cardiovascular risk factors in prediabetic individuals (26).

Comparison with similar studies from South Asia is instructive. Misra et al. reported a high prevalence of metabolic syndrome in prediabetic Indians, with dyslipidaemia and hypertension being the predominant components (27). Mohan et al. demonstrated that Asian Indians develop insulin resistance and cardiovascular complications at lower BMI thresholds than Western populations, underscoring the need for ethnicity-specific risk assessment criteria (28). Our findings are therefore broadly concordant with this regional literature while offering additional granularity regarding HbA1c correlations.

Limitations of this study include its hospital-based design, which may not be fully representative of the general population. The cross-sectional nature of the biochemical assessments precludes causal inference. Additionally, dietary intake and physical activity levels were self-reported and therefore subject to recall bias. The sample size of 200, while adequate for primary objectives, may have limited statistical power for subgroup analyses. Future studies with larger, community-based cohorts incorporating longitudinal follow-up and imaging biomarkers such as carotid intima-media thickness (CIMT) would add significantly to this evidence base.

5 | CONCLUSION

This case-control study demonstrates that prediabetes is associated with a significantly higher burden of cardiovascular risk factors — including hypertension, atherogenic dyslipidaemia, elevated BMI, and elevated 10-year Framingham CVD risk scores — compared to normoglycemic controls. HbA1c within the prediabetic range exhibits significant positive correlations with systolic blood pressure, total cholesterol, LDL-C, triglycerides, and the composite Framingham CVD risk score, and a significant negative correlation with HDL-C. These findings underscore the importance of comprehensive cardiovascular risk assessment at the time of prediabetes diagnosis and reinforce the clinical utility of HbA1c not only as a glycaemic marker but also as an early indicator of cardiovascular risk. Systematic screening and targeted multifactorial interventions during the prediabetic phase hold the potential to substantially reduce the dual burden of diabetes and cardiovascular disease.

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