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Research Article

Correlation of Blood Pressure with Glycemic Control and Lipid Profile among Patients with Type 2 Diabetes Mellitus: A Clinical Cross-Sectional Study

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ABSTRACT
Background: Patients with type 2 diabetes mellitus commonly exhibit concurrent abnormalities in glycaemic control, blood pressure, and lipid metabolism.
Objective: To evaluate the association of routinely measured glycaemic and lipid parameters with systolic and diastolic blood pressure in a hospital-based population with type 2 diabetes mellitus.
Methods: This cross-sectional study was conducted from January to June 2026 at Hayat Memorial Hospital, Lahore, Pakistan. A total of 100 adults with established type 2 diabetes were consecutively enrolled. Clinical, behavioural, anthropometric, blood pressure, glycaemic, glycated haemoglobin, and lipid parameters were recorded. Owing to incomplete data, 59 participants were included in the primary analysis, with variable-specific sample sizes applied where appropriate. Associations were assessed using Spearman correlation and multivariable linear regression adjusted for age, sex, body mass index, and diabetes duration.
Results: The mean age was 48.3 ± 10.8 years, and 34 participants (57.6%) were male. Elevated blood pressure was present in 42 (71.2%), glycated haemoglobin ≥7.0% in 44/54 (81.5%), and dyslipidaemia in 51 (86.4%). Systolic blood pressure correlated with fasting glucose (ρ=0.370, p=0.004) and glycated haemoglobin (ρ=0.388, p=0.004), while similar associations were observed for diastolic blood pressure. High-density lipoprotein cholesterol was inversely correlated with diastolic blood pressure (ρ=-0.271, p=0.038). After adjustment, each 10 mg/dL increase in fasting glucose was associated with 0.98 mmHg higher systolic and 0.51 mmHg higher diastolic blood pressure.
Conclusion: Fasting glucose showed the most consistent independent association with blood pressure. The frequent coexistence of poor glycaemic control, elevated blood pressure, and dyslipidaemia supports integrated cardiometabolic assessment in routine diabetes care.

Keywords: Type 2 Diabetes, Systolic Pressure, Diastolic Pressure, Fasting Glucose, Glycated Haemoglobin, Dyslipidaemia

INTRODUCTION

Type 2 Diabetes Mellitus is a condition of chronic abnormalities in sugar regulation that develops as a result of insulin resistance and a gradual loss of beta-cell function. Its clinical consequences are determined not only by hyperglycaemia, but also by the accumulation of vascular risk factors such as excess body weight, hypertension, dyslipidaemia, and physical inactivity[1,2]. When these abnormalities occur together, vascular injury is amplified, and the likelihood of coronary disease, stroke, renal impairment, and premature death rises substantially[3].

This burden is particularly relevant to Pakistan, which has a high prevalence of diabetes, and published evidence has documented the ongoing challenges in disease awareness, adherence to treatment, and metabolic control[4]. A patient whose glucose value is poorly controlled may also have untreated or inadequately controlled blood pressure and lipid abnormalities, making single-parameter assessment an incomplete approach to risk estimation[5].

Several biological pathways exist by which abnormal regulation of glucose and high arterial pressure might coexist. Increased sympathetic tone, increased sodium retention and altered renin-angiotensin-aldosterone activity are all associated with insulin resistance[6]. In addition, hyperglycaemia can lead to endothelial dysfunction, elevated oxidative stress, increased inflammatory signalling, non-enzymatic glycation of vascular proteins, and decreased arterial compliance, over the long

term. Such mechanisms may offer a tangible explanation underlying the parallel deterioration in glycaemic and haemodynamic control[7,8].

Many type 2 diabetics also have disorders in lipid metabolism. These often involve raised triglycerides, lowered high-density lipoprotein cholesterol, higher production of very low-density lipoprotein and a higher proportion of small dense low-density lipoprotein particles[8]. But there is variable correlation of different lipid fractions with blood pressure or glycaemic indices in different populations, and these may be influenced by various clinical factors such as obesity, drug treatment, diet, disease duration and other conditions[9].

Contemporary diabetes management therefore emphasises overall cardiovascular-risk reduction rather than correction of glucose alone[10-12]. Given that there is scarce evidence in the literature for the relationships of blood pressure with glycaemic measures and lipid indices in a clinical sample in the same population, this study aimed to examine the relationship of systolic and diastolic blood pressure with fasting glucose, postprandial glucose, glycated haemoglobin, total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol in adults with type 2 diabetes[13].

MATERIALS AND METHODS

Study Design and Setting

A clinical cross-sectional study was conducted in the Department of Medicine, Hayat Memorial Hospital Lahore, Pakistan, from January to June 2026. The recruitment of the respondents was done by consecutive non-probability sampling, which resulted in the inclusion of 100 adults whose type 2 diabetes mellitus was already known. The participants were assessed on one occasion. The data provided were then analysed by each variable; 59 records were available for the major descriptive analysis.

Eligibility

Participants were required to be at least 18 years old and to have had type 2 diabetes for six months or longer. Diagnosis was accepted if there was evidence in existing medical documents, a current glucose-lowering treatment, or biochemical evidence. Planned study procedures and written informed consent were also required.

Subjects with type 1 diabetes, gestational diabetes, stage IV and V chronic kidney failure, chronic liver disease, acute infection, diabetic ketoacidosis, hyperosmolar hyperglycaemic state and other acute diabetic emergencies were excluded. Patients were excluded if lipid-lowering treatment was not stable for at least three months to minimise the effect of recent changes in treatment. Measurements lacking for a particular analysis were excluded from that analysis.

Clinical and Behavioural Data

The age, sex, diabetes duration, smoking, physical activity, antidiabetic treatment, body mass index, systolic and diastolic pressure, fasting and postprandial glucose, glycated haemoglobin, total cholesterol, triglycerides, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol were documented on a case-record form designed for the purpose. Smoking was dichotomized as current, former, and never, with current smoking defined as people who were current smokers and/or smoked regularly in the past 30 days. Physical activity was considered adequate when there was at least 150 minutes of moderate intensity activity per week. Antidiabetic treatment was classified into one of four categories: oral antidiabetic drugs, insulin, oral and insulin therapies, and lifestyle modification alone (no glucose-lowering drugs).

Anthropometry and Blood-Pressure Measurement

Weight was obtained using a calibrated digital scale with the participants in light clothes and without shoes. Stadiometer height was measured to the nearest 0.1cm with the wall-mounted stadiometer, the head in the Frankfurt plane. Body mass index was calculated as kilograms divided by metres squared and was retained as a continuous covariate in the primary analyses.

Blood pressure was measured with an automated sphygmomanometer with proper cuff size. Participants refrained from smoking, drinking caffeine, and engaging in heavy exercise 30 minutes before measurement and were seated for at least five minutes with back supported, feet on the floor, arms at the level of the heart, and legs uncrossed. Two readings of the right arm were made approximately 2 minutes apart. If the difference between the systolic readings was >10 mmHg or the difference between diastolic readings was >5 mmHg, a third reading was made, and the two closest readings averaged. High blood pressure was defined for categorical comparisons as a

systolic blood pressure ≥140 mmHg, or a diastolic blood pressure ≥90 mmHg, or use of antihypertensive medication.

Biochemical Assessment

Venous blood was collected after an 8-12-hour overnight fast using aseptic technique. Fasting serum glucose was measured enzymatically by the glucose oxidase-peroxidase method. Postprandial glucose represented the two-hour value after the participant's usual meal. Glycated haemoglobin was measured with a standardised assay and reported as a percentage.

Total cholesterol, triglycerides, and high-density lipoprotein cholesterol were determined using enzymatic colorimetric methods. Low-density lipoprotein cholesterol was measured directly or calculated with the Friedewald equation when triglycerides were <400 mg/dL. Direct measurement was used when triglyceride concentration made calculation unsuitable. Where laboratory results were originally expressed in mmol/L, they were converted to mg/dL using factors of 18.018 for glucose, 38.67 for total/LDL/HDL cholesterol, and 88.57 for triglycerides.

Operational Definitions

Glycated haemoglobin was treated as the principal glycaemic measure and analysed both continuously and categorically. Values <7.0% were classified as controlled and values ≥7.0% as uncontrolled. Dyslipidaemia was defined by at least one of the following findings: total cholesterol ≥200 mg/dL, triglycerides ≥150 mg/dL, low-density lipoprotein cholesterol ≥100 mg/dL, high-density lipoprotein cholesterol <40 mg/dL in men, or <50 mg/dL in women. Continuous lipid measurements were retained for correlation and regression analysis.

The primary analytical objective was to quantify the relationship of systolic and diastolic pressure with glycated haemoglobin. Secondary analyses examined both pressure components in relation to fasting glucose, postprandial glucose, total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. Age, sex, body mass index, and diabetes duration were included as covariates in the reported adjusted regression models.

Statistical Analysis

Case-record forms completed were checked for completeness and internal inconsistencies. The electronic data set was audited for duplicate records, unit mismatch, transcription errors, and implausible values. Entries that were recorded as 'nil', 'nill' or a single period or as blank were coded as missing. Only questionable values were corrected if the original case form or laboratory report validated the desired value. Identifiers were de-identified before analysis and coded with study numbers.

Analysis was performed using the IBM SPSS Statistics 26.0 program. Histograms, quantile-quantile plots, and the Shapiro-Wilk test were used to assess the distributional characteristics. Data are presented as mean and standard deviation for normally distributed continuous data, median and interquartile range (IQR) for skewed continuous data, and frequencies and percentages for categorical data. The independent-samples t test, Mann-Whitney U test, one-way analysis of variance, Kruskal-Wallis test, chi-square test, or Fisher exact test was used for the comparisons, depending on the distribution and group number.

The relationships between blood pressure and metabolic parameters were initially examined graphically. It was decided to use Pearson correlation with approximately normal linear relationships, and Spearman rank correlation for skewed data or as affected by extreme observations. A multivariable linear regression model was obtained for each of the two measures: systolic and diastolic pressure. Variance inflation factors and tolerance were used to assess the collinearity, and the residual plots, quantile-quantile plots and Cook distance were used to assess the model assumptions and influential observations. All statistical tests were two-sided and $p<0.05$ was regarded as significant.

Ethical Considerations

The protocol was approved by the Institutional Review Board before recruitment, and administrative permission was obtained from Hayat Memorial Hospital. All the participants were explained the study and gave written informed consent. Participation was voluntary and refusal or withdrawal did not impact clinical care. The data was anonymised prior to analysis and only used for research in accordance with the Declaration of Helsinki.

RESULTS

Participant Characteristics

The principal analytic dataset comprised 59 participants. Their mean age was 48.3 ± 10.8 years, and 34 (57.6%) were men. Diabetes had

been present for a median of 4.5 years. Body mass index was available for 52 participants; the mean was 34.2 ± 5.9 kg/m² and 42/52 (80.8%) were obese. Twenty participants (33.9%) were current smokers and 30 (50.8%) were physically inactive.

Average systolic pressure was 137.9 ± 17.3 mmHg, whereas median diastolic pressure was 90 mmHg (IQR 81-100). Forty-two participants (71.2%) met the study definition of elevated blood pressure. Median glycated haemoglobin was 7.9% (IQR 7.1-9.0), and 44 of 54 participants with available measurements (81.5%) had glycated haemoglobin ≥7.0%. Dyslipidaemia was detected in 51 (86.4%). The complete descriptive profile is shown in Table 1.

Table 1. Clinical and Metabolic Characteristics of the Analysed Cohort

Measure	Observed value
Analysed participants	59
Age, years	48.3 ± 10.8
Men	34 (57.6%)
Women	25 (42.4%)
Duration of diabetes, years	4.5 (3.0–8.0)
Body mass index, kg/m²	34.2 ± 5.9
Obesity (BMI ≥30 kg/m²)	42/52 (80.8%)
Current smoking	20 (33.9%)
Former smoking	1 (1.7%)
Never smoked	38 (64.4%)
Meets activity threshold	28 (47.5%)
Below activity threshold	30 (50.8%)
Systolic pressure, mmHg	137.9 ± 17.3
Diastolic pressure, mmHg	90 (81–100)
Elevated pressure	42 (71.2%)
Fasting glucose, mg/dL	204.3 ± 73.2
Postprandial glucose, mg/dL	307 (195–368)
HbA1c, %	7.9 (7.1–9.0)
HbA1c ≥7.0%	44/54 (81.5%)
Total cholesterol, mg/dL	156.0 (119.9–210.5)
Triglycerides, mg/dL	146.0 (91.5–226.1)
LDL-C, mg/dL	90.0 (73.5–130.0)
HDL-C, mg/dL	38.7 (34.0–43.0)
Dyslipidaemia	51 (86.4%)

Values are reported as mean ± SD, median (IQR), or n (%), according to data type.

Correlation of Blood Pressure with Glycaemic and Lipid Parameters

Fasting glucose and glycated haemoglobin were the two metabolic measures most consistently related to arterial pressure. Systolic pressure correlated with fasting glucose (ρ=0.370, p=0.004) and glycated haemoglobin (ρ=0.388, p=0.004). The corresponding diastolic coefficients were ρ=0.342 (p=0.008) for fasting glucose and ρ=0.362 (p=0.007) for glycated haemoglobin. Postprandial glucose showed positive but non-significant correlations with both pressure components.

Total cholesterol, triglycerides, and low-density lipoprotein cholesterol were not significantly related to systolic or diastolic pressure. High-density lipoprotein cholesterol was inversely related to diastolic pressure (ρ=-0.271, p=0.038), but the analogous systolic association did not reach significance (ρ=-0.239, p=0.068). Table 2 provides the coefficients, confidence intervals, and p-values.

Table 2. Rank Correlations between Metabolic Measurements and Arterial Pressure

Metabolic measure	n	SBP: ρ (95% CI)	p	DBP: ρ (95% CI)	p
Fasting glucose	59	0.370 (0.134 to 0.562)	0.004	0.342 (0.096 to 0.546)	0.008
Postprandial glucose	39	0.260 (−0.048 to 0.527)	0.110	0.270 (−0.059 to 0.545)	0.097
HbA1c	54	0.388 (0.119 to 0.622)	0.004	0.362 (0.084 to 0.604)	0.007

Total cholesterol	59	−0.067 (−0.342 to 0.209)	0.613	−0.102 (−0.395 to 0.186)	0.440
Triglycerides	59	−0.107 (−0.357 to 0.166)	0.420	−0.196 (−0.450 to 0.070)	0.137
LDL-C	59	−0.043 (−0.309 to 0.232)	0.746	−0.064 (−0.345 to 0.210)	0.628
HDL-C	59	−0.239 (−0.480 to 0.030)	0.068	−0.271 (−0.497 to −0.021)	0.038

SBP, systolic blood pressure; DBP, diastolic blood pressure; CI, confidence interval. Spearman rank correlation; p<0.05 indicates statistical significance.

Blood Pressure According to Glycaemic-Control Status

Glycated-haemoglobin data were available for 54 participants: 44 had values ≥7.0% and 10 had values <7.0%. Median systolic pressure was 140 mmHg in the uncontrolled group and 121 mmHg in the controlled group (p=0.002). Median diastolic pressure was 91 versus 80 mmHg, respectively (p=0.001).

Median fasting glucose was 217.5 mg/dL among participants with HbA1c ≥7.0% and 110.0 mg/dL among those below 7.0% (p<0.001). Median postprandial glucose was 329 versus 130 mg/dL (p<0.001). No statistically significant between-group difference was observed for total cholesterol, triglycerides, low-density lipoprotein cholesterol, or high-density lipoprotein cholesterol.

Multivariable Analysis

Fasting glucose remained independently associated with both blood-pressure components after adjustment for age, sex, body mass index, and diabetes duration. For every 10 mg/dL increase in fasting glucose, systolic pressure was 0.98 mmHg higher (95% CI 0.44 to 1.53; p<0.001) and diastolic pressure was 0.51 mmHg higher (95% CI 0.20 to 0.82; p=0.001).

The unadjusted associations involving glycated haemoglobin were no longer significant in the multivariable models. Postprandial glucose retained positive coefficients but did not meet the prespecified significance threshold. None of the lipid fractions independently explained variation in systolic or diastolic pressure after adjustment (Table 3).

Table 3. Multivariable Estimates for Systolic and Diastolic Blood Pressure

Predictor	n	Adjusted SBP change, β (95% CI), mmHg	p	Adjusted DBP change, β (95% CI), mmHg	p
Fasting glucose, per 10 mg/dL	52	0.98 (0.44 to 1.53)	<0.001	0.51 (0.20 to 0.82)	0.001
Postprandial glucose, per 10 mg/dL	32	0.53 (−0.17 to 1.23)	0.139	0.38 (−0.04 to 0.81)	0.077
HbA1c, per 1%	50	2.52 (−2.75 to 7.79)	0.349	1.38 (−2.03 to 4.78)	0.428
Total cholesterol, per 10 mg/dL	52	−0.32 (−1.42 to 0.79)	0.576	−0.23 (−0.95 to 0.49)	0.528
Triglycerides, per 10 mg/dL	52	−0.52 (−1.11 to 0.08)	0.088	−0.34 (−0.74 to 0.07)	0.103
LDL-C, per 10 mg/dL	52	−0.09 (−1.60 to 1.42)	0.904	−0.05 (−1.00 to 0.90)	0.917
HDL-C, per 10 mg/dL	52	−2.12 (−9.12 to 4.88)	0.553	−2.40 (−5.96 to 1.16)	0.186

Models were adjusted for age, sex, BMI, and duration of diabetes. CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure. Statistical significance was set at p<0.05.

HbA1c and Systolic Pressure

Figure 1 demonstrates substantial individual variation in systolic pressure across the observed HbA1c range, together with an overall upward trend. The unadjusted Spearman correlation was ρ=0.39 (p=0.004; n=54).

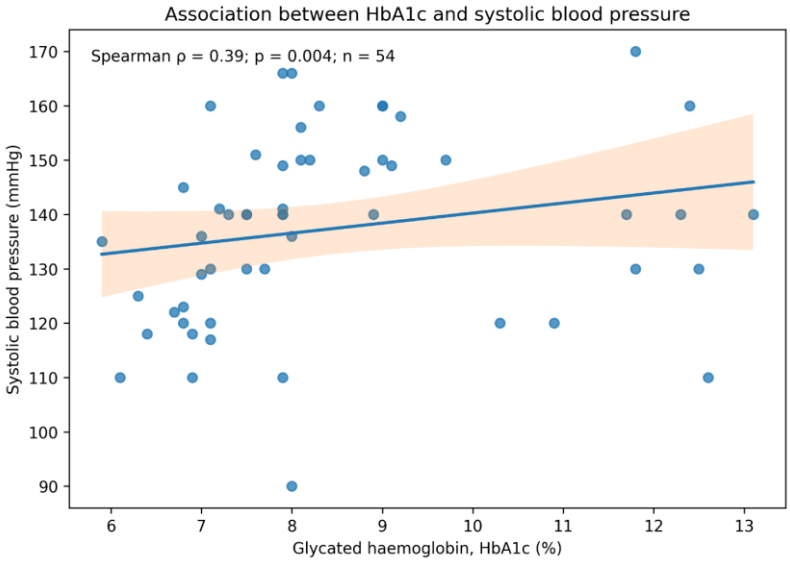

Figure 1. Scatterplot of Glycated Haemoglobin (Hba1c) Against Systolic Blood Pressure.

DISCUSSION

Three findings stand out from this analysis. First, uncontrolled glycaemia, elevated arterial pressure, and dyslipidaemia were simultaneously common. Second, both fasting glucose and glycated haemoglobin tracked with systolic and diastolic pressure before covariate adjustment[1]. Third, only fasting glucose remained independently associated with both pressure components in the adjusted models, while the observed lipid relationships were weak or absent. Together, these results suggest that a single clinic visit can reveal clustering of several cardiovascular-risk abnormalities rather than isolated hyperglycaemia[2,3].

The difficulty of achieving multiple treatment targets is not unique to this cohort. In a Jordanian cross-sectional study of 1,200 adults with type 2 diabetes, Hyassat et al. reported that 41.7% achieved HbA1c <7%, 61.9% achieved blood pressure <140/90 mmHg, and only 15.4% met glycaemic, blood-pressure, and low-density-lipoprotein goals at the same time[11]. The greater proportions of uncontrolled glycaemia and elevated pressure observed here may reflect differences in obesity burden, referral patterns, medication use, continuity of care, or access to repeated monitoring[4,5].

Our categorical findings also show that participants with HbA1c ≥7.0% had substantially higher median systolic and diastolic pressures[6,7]. Lou et al., studying 2,485 Chinese adults with type 2 diabetes, found that the combination of hypertension and dyslipidaemia was associated with greater odds of poor glycaemic control and demonstrated an additive interaction between the two conditions[8]. Although their outcome direction differed, both studies point toward a clinically meaningful tendency for metabolic and haemodynamic abnormalities to cluster[9,10].

The loss of statistical significance for glycated haemoglobin after multivariable adjustment is informative. It suggests that the apparent association of longer-term glycaemic exposure with blood pressure may be partly due to shared factors, including duration of diabetes, age or body size[11]. However, the association between fasting glucose remained independent. In 1,715 Chinese adults with T2DM, Zhang et al. found no increase in the risk of hypertension with FPG and a negative correlation between HbA1c and hypertension[12]. This between study variation has a plausible explanation as treatment patterns, renal function, body composition, diseases severity, and definition of HTN vary between settings[13].

The lipid findings were also complicated. There was no significant relationship between total cholesterol, triglycerides, and low-density lipoprotein cholesterol and either component of blood-pressure in this sample[14,15]. In contrast, Sharahili et al. found some relationship between HbA1c with total cholesterol and triglycerides in 988 Saudi patients, while Nnakenyi et al. reported on relationship between glycaemic control and lipid abnormalities in a resource-limited population in Nigeria[12,16]. These studies addressed lipid-glycaemia relationships rather than blood-pressure prediction, which may partly explain the contrasting pattern[17].

High-density lipoprotein cholesterol was the only lipid fraction associated with pressure in the unadjusted analysis, showing an inverse

relationship with diastolic pressure[18]. This association did not remain significant after adjustment, so it is better interpreted as part of a broader adverse metabolic profile rather than as an independent blood-pressure determinant. Kumar et al. likewise reported that lipid fractions do not change uniformly across glycaemic categories, with stronger differences for triglycerides and very-low-density lipoprotein cholesterol than for several other conventional fractions[6]. Similar heterogeneities have also been described in other diabetic cohorts[17].

Although the adjusted fasting-glucose coefficient was small on a per-10 mg/dL basis, there are potentially large differences across the wide range of fasting glucose observed that may be clinically important. This may be explained by shared mechanisms: insulin resistance could raise sympathetic activation and renal sodium retention, and hyperglycaemia could reduce endothelial function and arterial compliance[7,18]. The current design can only be used to conclude that high glucose levels were associated with high levels of pressure at the same time and that either one or both were the cause of the other, or that the two were due to some other metabolic disturbance[19].

These findings have a significant role in the context of daily practice of diabetes care in Pakistan. There is a high disease burden reported both nationally and regionally, and awareness, adherence and metabolic control remain ongoing challenges[3,4]. A pragmatic approach is to evaluate glucose, blood pressure, lipid status, body weight, lifestyle factors and medication use in parallel at every meeting. Major cardiovascular-risk advice for diabetes also calls for coordinated care in these areas[6].

This study has methodological strengths, including simultaneous measurement of several cardiometabolic variables, standardisation of mixed laboratory units, and use of adjusted regression in addition to simple correlation. Its limitations should temper interpretation[6-9]. The design was cross-sectional, the effective sample size was modest and varied because of missing observations, and recruitment was confined to a single hospital. Detailed information on antihypertensive and lipid-lowering therapy was insufficient for comprehensive pharmacological adjustment. Diet, renal function, socioeconomic circumstances, medication adherence, and central adiposity may also have contributed residual confounding. Finally, a clinic-based blood-pressure measurement does not fully represent long-term blood-pressure exposure[18,19].

Future multicentre prospective studies should use repeated blood-pressure measurements, complete medication histories, and more comprehensive metabolic phenotyping to clarify the temporal sequence of these relationships. The signal most consistently reproduced for the current cohort was the independent association of fasting glucose with both systolic and diastolic pressure; adjusted relationships of glycated haemoglobin and conventional lipid fractions were less consistent[20,21].

CONCLUSION

The study demonstrates substantial coexistence of poor glycaemic control, elevated blood pressure, and dyslipidaemia among adults with type 2 diabetes treated in a hospital setting. Fasting glucose was independently related to both systolic and diastolic pressure, when age, sex, BMI and diabetes duration were adjusted. The glycated haemoglobin also showed an association with pressure before adjustments, but none of the individual lipid fractions were independently associated with blood pressure. These observations are in favor of a holistic cardiometabolic strategy, where the glucose, arterial pressure, lipid status, weight, lifestyle and adherence to treatment are considered as a group.

Declarations

Funding: No external funding was received for this study.

Competing Interests: The authors declare that they have no competing interests.

Authors' Contributions: FS and MA contributed to study conception and design. HAB and KA contributed to data collection and organization. SS contributed to statistical analysis and interpretation of the findings. AH contributed to literature review and manuscript drafting. FS and MA critically revised the manuscript. All authors reviewed and approved the final manuscript.

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