



Impact of Hyperglycaemia on Blood Pressure: A retrospective study of the impact of hyperglycaemia on blood pressure including potential mechanisms and implications for treatment.

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Abstract

Background: Diabetes mellitus and hypertension commonly coexist, leading to significant cardiovascular risk. Chronic hyperglycaemia may result in high blood pressure by both vascular and neurohormonal mechanisms. **Methods:** This retrospective observational study conducted on 100 adult patients in Internal Medicine Department in Prime Specialist Medical Centre between January 2024 and December 2025. The examination data collected included fasting blood glucose (FBG), postprandial blood glucose (PPBG), HbA1c, systolic blood pressure (SBP) and diastolic blood pressure (DBP). Patient population was divided into three groups as normoglycemic, prediabetic and diabetic. **Results:** The mean age was 52.4 ± 11.8 years, and 58% were male. Mean FBG was 146.8 ± 48.5 mg/dL, HbA1c $7.8 \pm 1.6\%$, SBP 136.5 ± 18.2 mmHg, and the DBP 84.7 ± 10.6 mmHg. Hypertension was present in the 54% of patients. HbA1c showed that the significant positive correlation with SBP ($r = 0.47$, $p < 0.001$). Diabetic patients had higher mean SBP (144.2 ± 16.8 mmHg) compared to normoglycemic individuals (124.6 ± 12.4 mmHg; $p < 0.001$). HbA1c independently predicted systolic blood pressure ($\beta = 0.38$, $p = 0.001$). **Conclusions:** Hyperglycaemia is highly associated with high blood pressure and appropriate measures to manage hyperglycaemia should be combined with the control of hypertension.

Keywords: Blood Pressure, Diabetes Mellitus, Hyperglycaemia, Hypertension, HbA1c.



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INTRODUCTION

Diabetes mellitus and hypertension are the two most common non-communicable diseases globally, both diseases are significant risk factors for global morbidity and mortality [1]. The epidemic of type 2 diabetes, primarily caused by urbanization, inactivity, obesity and dietary transitions has occurred in concert with the emerging burden of hypertension [2]. Hundreds of millions of adults around the world are now diagnosed with diabetes, and a large percentage of them also have high blood pressure. Coexistence of both diseases significantly increases the risk of cardiovascular events such as coronary heart disease, stroke or heart failure and chronic kidney disease [3]. Diabetes and hypertension are an interacting pathophysiological axis, promoting each other's adverse effects on vascular damage and healthcare burden in both short- and long-term.

The principal biochemical feature of diabetes, hyperglycaemia, is central to the vascular injury. Chronic hyperglycaemia results in metabolic and molecular alterations in a cascade that involves the formation of advanced glycation end-products (AGEs), activation of protein kinase C pathways, excessive oxidative stress and chronic low-grade inflammation [4,5]. Such mechanisms affect endothelial function, precursor cell mobilization, arterial stiffness and vascular reactivity [6,7]. Endothelial dysfunction (ED) especially loss of nitric oxide-mediated vessel relaxation in addition to reduced nitric oxide levels and increased peripheral vascular resistance as main contributors to the development of hypertension [8]. Hyperglycemia induces insulin resistance that leads to sympathetic nervous system's activation and renin angiotensin aldosterone system (RAAS) stimulation, both promoting sodium retention and vasoconstriction and resulting in the development of high blood pressure [9,10].

The relationship between hyperglycaemia and cardiovascular risk is not only macrovascular [11,12]. There is also evidence that even mild elevations in fasting glucose and HbA1c are linked with higher level of arterial stiffness and SBP. Clinical examination shows that patients with poorly controlled diabetes mellitus usually have higher BP than those with

good glycaemic control. This mutual interaction implies that hyperglycaemia could not only occur together with hypertension, but it might also result in development and perpetuation. Thus, understanding the impact of glycaemic status on BP control is important for broader assessment and management of cardiovascular risk.

Even though the relationship of diabetes with hypertension is well-established, the direct influence of different levels of hyperglycaemia on blood pressure components in clinical practice has not been adequately investigated. Most of the published data on long-term cardiovascular outcomes or observational intervention-based trials, with less emphasis on correlation between retrospective hospital-based data and glycaemic indices and blood pressure measurements. The retrospective review permits examination of clinically collected data over long-term and illustrates patterns and associations applicable to routine practice. Such data is important for trend analysis, risk stratification, and early interventions strategies.

Objectives

- To assess the correlation between fasting blood glucose and blood pressure levels
- To compare blood pressure level in normoglycemic and hyperglycaemia groups
- To explore the association of HbA1c with SBP and DBP

MATERIALS AND METHODS

Study Design

This retrospective observational study aimed to assess the relationship between hyperglycaemia and blood pressure in patients attending at the Department of Internal Medicine, Prime Specialist Medical Centre. The retrospective nature of the study permitted assessment of pre-recorded data without any potential intervention/change in management of patients. The research aimed at finding associations and trends among glycaemic indexes, blood pressures over a structured time frame of 2 years.

Study Setting

This research was carried out in the Department of Internal Medicine, Prime Specialist Medical Centre. The service attends a broad spectrum of adult patients with metabolic, cardiovascular and systemic diseases. The electronic medical records in the department served as the main data source. Patients were tested in a standardized environment with access to laboratory investigations and vital parameters were recorded prospectively.

Study Duration

The research took place over two years from January 2024 to December 2025. All patients within this time span if meeting eligibility were screened and considered for inclusion. This period was chosen to allow a sufficient sample size and to observe the changes in glycaemic and blood pressure over time.

Study Population

The study included 100 patients. Inclusion and exclusion criteria for the patients were determined in advance. The cohort included adults attending the internal medicine department for regular examination or treatment of metabolic and cardiovascular diseases during the study period.

Inclusion Criteria

Patients who were ≥ 18 years of age with documented measurements of blood glucose and blood pressure entered in the system were considered. Patients with a complete medical record were included. This also ensured as an offline consistency check for analyzing the data.

Exclusion Criteria

Patients with secondary hypertension due to endocrine or renal artery stenosis were excluded from the analysis for confounding effect. Patients with chronic renal insufficiency stage IV or V were not included because severe decline of renal function significantly affects the blood pressure control. Due to physiological changes of glucose metabolism and blood pressure during pregnancy, pregnant women were also excluded. Moreover, records lacking essential data or with missing data were excluded from the analysis.

Data Collection

Data were obtained from patient records. All variables were recorded such as demographic features (age and gender) and anthropometric measurement (assessed by body mass index [BMI]). Glycaemic variables comprising fasting blood glucose (FBG), postprandial blood glucose (PPBG) and HbA1c. Blood pressure was measured in terms of clinic SBP and DBP. Other clinical information including duration diabetes, use of anti-hypertensive drugs, and the Values for lipid profile

(when available) were also noted. All the information's were analysed after having made anonymous them in order to safeguard privacy.

Grouping of Participants

Participants were classified to three groups according to their glucose tolerance. Group A: Normal range for blood sugar in normoglycemic persons. Group B consisted of prediabetic patients who had impaired fasting glucose or borderline HbA1c. Group C was made up of patients (hyperglycaemic) with diabetes mellitus who had an elevated fasting blood glucose or HbA1c level. This grouping allowed for relative comparisons of blood pressure between different glycaemic control groups.

Outcome Measures

The primary outcome of the study was to establish the relationship between blood glucose level (FBG, PPBG & HbA1c) and blood pressure parameters (SBP& DBP). The secondary outcome was to determine the prevalence of hypertension in hyperglycaemic patients as well as to compare the blood pressure distribution between normoglycemia and hyperglycemia.

Statistical Analysis

SPSS was used for data analysis. Demographic and clinical characteristics were presented by descriptive statistics, continuous variables were mean values $\pm$ standard deviation, whereas categorical variables were given as frequencies and percentages. Pearson or Spearman's correlation analysis was used for the relationship between glycaemic parameters and blood pressure values, depending on data distribution. The mean of the blood pressure in glycaemic groups was compared using independent t-tests or one-way ANOVA. A chi-square test was used to measure the relationship between categorical variables, including prevalence of hypertension in groups. Values of $p < 0.05$ were considered statistically significant.

RESULTS

Demographic Characteristics

The study enrolled 100 patients. The average age among the study group of patients was 52.4 ± 11.8 years, and most of them were between 41-60 years old. Men constituted 58% and women 42% of the participants. Patients were overweight (27.6 ± 4.3 kg/m²) with high proportions of patients being in the overweight and obese categories.

Table 1 Demographic Characteristics of Study Population (n = 100)

Variable	Value
Mean Age (years)	52.4 ± 11.8
Male	58 (58%)
Female	42 (42%)
Mean BMI (kg/m ²)	27.6 ± 4.3
Duration of Diabetes (years)*	6.2 ± 4.5

Glycaemic Profile of Patients

The average FBG was 146.8 ± 48.5 mg/dL and the average PPBG was 212.4 ± 62.7 mg/dL. The mean HbA1c in study population was $7.8 \pm 1.6\%$. According to glycaemic classification, 28 were normoglycemic, 24 prediabetic and 48 diabetics (hyperglycaemic). These results are presented in Table 2.

Table 2 Glycaemic Profile of Patients (n = 100)

Parameter	Mean $\pm$ SD / n (%)
Fasting Blood Glucose (mg/dL)	146.8 ± 48.5
Postprandial Blood Glucose (mg/dL)	212.4 ± 62.7
HbA1c (%)	7.8 ± 1.6
Normoglycemic	28 (28%)
Prediabetic	24 (24%)
Diabetic (Hyperglycaemic)	48 (48%)

Blood Pressure Distribution

Mean SBP of study population was 136.5 ± 18.2 mmHg and mean DBP was 84.7 ± 10.6 mmHg. 54% of all patients had hypertension (SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg or antihypertensive medication at entry). Distribution of blood pressure levels is shown in Table 3.

Table 3 Blood Pressure Distribution (n = 100)

Parameter	Mean $\pm$ SD / n (%)
Systolic BP (mmHg)	136.5 ± 18.2
Diastolic BP (mmHg)	84.7 ± 10.6
Normotensive	46 (46%)
Hypertensive	54 (54%)

Correlation Between Blood Glucose and Blood Pressure

Correlation analysis showed positive correlation between glycaemic parameters and blood pressure values. Fasting blood glucose was moderately associated with systolic blood pressure ($r = 0.42$, $p < 0.001$) and weakly correlated with diastolic blood pressure ($r = 0.29$, $p = 0.004$). Systolic and diastolic blood pressures were also significantly associated with HbA1c ($r = 0.47$, $P < 0.001$) and ($r = 0.31$, $P = 0.002$), respectively). These results imply increased glycaemic levels are related with increased blood pressure values.

Comparison Between Glycaemic Groups

Normoglycemic patients differed from those with diabetes (i.e. hyperglycaemia) in mean blood pressure when systolic and diastolic pressures were compared according to glycaemic status. The mean SBP among diabetics was 144.2 ± 16.8 mmHg and in non-diabetics was 124.6 ± 12.4 mmHg where $p < 0.001$). The mean DBP was 88.9 ± 9.8 mmHg among diabetics and 78.3 ± 8.6 mmHg in normoglycemic patients ($p = 0.002$). Values from prediabetic patients were intermediate between those of diabetic and control ones. These results suggest a stepwise rise in blood pressure with increasing severity of glycaemic state.

Multivariate Analysis

Multivariate linear regression analysis was conducted to assess whether hyperglycaemia independently contributed to elevated SBP after accounting for age, sex, BMI and diabetes duration. HbA1c continued to be an independent significant predictor of systolic blood pressure ($\beta = 0.38$, $p = 0.001$). BMI and age were also independently associated with increased SBP. After adjustment however, the correlation between HbA1c and diastolic blood pressure was slightly diminished although it remained significant ($p = 0.03$).

These results indicate that hyperglycaemia is independently associated with high blood pressure other than traditional risk factors, and emphasize the need for integrated approach of glycaemic control with BP in patients with diabetes.

DISCUSSION

In the present retrospective study, the effect of hyperglycaemia on blood pressure in 100 patients were analysed. The results revealed a significant positive relationship between glycaemic parameters especially fasting blood glucose and HbA1c, with both systolic and diastolic blood pressure. When patients were grouped as diabetic (hyperglycaemic), the mean Systolic and Diastolic blood pressure values were significantly higher in comparison to normoglycemic subjects, with prediabetic group showing intermediate levels. Multivariate analysis also demonstrated that HbA1c was still an independent predictor for systolic blood pressure in the presence of age, BMI and other confounding factors. These findings imply that a progressive rise in blood pressure is related to the deterioration of glycaemic control.

Comparison with Previous Studies

The results are in accordance with the previous epidemiological and clinical studies, where a high prevalence of hypertension among diabetic patients has been reported. Several prospective large cohort studies have shown that patients with uncontrolled diabetes are more probable to develop hypertension [13]. It has been reported that increased HbA1c levels were correlated with arterial stiffness and the systolic blood pressure in previous studies. Consistent with this results, previous research has shown a modest positive association between fasting glucose and SBP [14]. Conflicting results and confounders such as obesity, insulin resistance have been proposed [15]. The current study adds further evidence by documenting departmental-level real-world data over a 2-year period, demonstrating the clinical significance of this association in everyday practice.

Mechanisms Linking Hyperglycaemia and Hypertension

There are multiple pathophysiological mechanisms through which hyperglycaemia and hypertension correlate. Activation of the RAAS is a key mediator, because chronic hyperglycaemia promotes angiotensin II production, which causes vasoconstriction and sodium reabsorption. Furthermore, hyperglycaemia leads to the overactivity of the sympathetic nervous system with associated increases in the heart rate as well as peripheral vascular resistance. Another important mechanism is endothelial dysfunction, high glucose levels are known to inhibit nitric oxide production, leading to decreased vasodilation then increased vascular stiffness. The production of advanced glycation end products (AGEs) also contributes to the vascular damage by crosslinking of collagen and elastin which results in diminished arterial compliance. Hyperglycaemia promotes vascular inflammation and oxidative stress leading to an accelerated atherosclerosis and disturbance of normal vascular function. These mechanisms collectively represent a biological rationale for the association between poor glycaemic control and high blood pressure.

Strengths of the Study

This study has several strengths. It employed a 2-year follow-up data that provided sufficient patient representation and diversity in clinical variables. The real-world clinical data application makes the findings more relevant in practice. Moreover, it is a departmental study which represents daily practice and yields data that can be extrapolated to similar settings.

Limitations

The study has several limitations despite its strengths. The retrospective nature of the study does not allow us to determine causality between hyperglycaemia and hypertension. Generalisability may be limited by the modest sample size (100 patients). As a single centre research, the results may not be generalizable. Some potential confounders including dietary patterns, physical activity and socioeconomic status were not recorded in medical records and were missing from the analysis.

CONCLUSION

The current retrospective study showed a strong positive relationship between hyperglycaemia and the higher blood pressure for the previous two years. Such hyperglycaemic patients recorded higher systolic blood pressure, and diastolic blood pressure with an apparent more hypertensive prevalence compared to normoglycemic. These results underscore that glycaemic status is relevant to the aetiology of blood pressure variance and conferring over-all cardiovascular risk.

These findings have important clinical implications for glycaemic control and blood pressure management by indicating that to prevent long-term cardiovascular complications, comprehensive therapeutic approach should be considered to lower both hyperglycaemia and hypertension. Reduced blood pressure can be partly due to better monitoring and control of blood glucose. Nevertheless, prospective, multicentre studies should be performed to examine causality and investigate long-term consequences of glycaemic control on blood pressure dynamics due to the retrospective design and small sample size in this study.

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