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# Influence of Hypertension and Other Chronic Comorbidities on the Clinical Profile of Diabetes Patients

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**Abstract:** Diabetic patients are often seen to have high blood pressure, which can further compromise diabetic control and lead to increased end-organ damage. The main purpose of the current study was to examine the clinical characteristics of hypertension and other long-term comorbidities in people with type 2 diabetes mellitus (T2DM). Cross-sectional research was conducted on 131 patients with type of diabetes Mellitus (DM) who attended the hospitals in Baghdad, Iraq. The follow-up time was defined as 12 months (from March 2025 to March 2026). There were 72.5% of patients with high blood pressure from the 131 patients. The HbA1C level (8.4 ± 1.6% vs. 7.2 ± 1.1%), the frequency of hyperglycemia, and the microvascular and macrovascular complications of diabetes were significantly higher in those who had diabetic hypertension. People with diabetes need integrated care as well as multidisciplinary care approaches.

**Keywords:** Hypertension, Diabetes, Dyslipidemia, Chronic Kidney Disease, and Complications.

## INTRODUCTION

Diabetes mellitus is the biggest public health problem in the world today and is defined as chronic hyperglycemia and massive metabolic dysregulation [Meo, S. A. *et al*., 2017]. The most common and most important associated condition is hypertension; up to 2/3 of diabetes patients are associated with hypertension [Ta, S. 2014]. These conditions act synergistically on each other and synergistically enhance end-organ damage, the difficulty of treatment, and early mortality [Diabetes, A. 2015].

Insulin resistance, a key characteristic of type 2 diabetes, leads to increased blood pressure by causing sodium retention in the kidneys and increasing sympathetic nervous system activity. On the other hand, hypertension over time causes more insulin resistance and dysfunction of the pancreatic beta cells [American Diabetes Association, 2010]. This is a bidirectional association that significantly affects the clinical characteristics of diabetic patients, especially for the development of microvascular (diabetic nephropathy and retinopathy) and macrovascular (myocardial infarction and ischemic stroke) events. Hypertension turns an easily controlled metabolic into an elevated-risk, accelerated cardiovascular state [Contreras, F. *et al*., 2000; Pavlou, D. I. *et al*., 2018].

Apart from hypertension, diabetics have a wide range of chronic comorbidities, such as Dyslipidaemia, obesity, and chronic kidney failure [Kemche, B. *et al*., 2020]. These disorders often occur together, and thus, the burden of metabolism becomes increased and exceeds what a person's body can compensate for, such as when they are involved in metabolic syndrome [De Boer, I. H. *et al*., 2017]. Moreover, the incidence of chronic kidney disease is a marker for advanced microvascular damage and significantly affects the renal clearance of glucose-lowering drugs, which requires careful dose titration to avoid unnecessarily high risk of hypoglycemic events and worsening renal function [Strain, W. D., & Paldánius, P. M. 2018]. In this present study, this was the case, and the authors sought to determine the clinical presentation and progression of diabetes in the presence of hypertension and other chronic conditions.

## PATIENTS AND METHODS

**Study's design and setting.**

This was a cross-sectional study in an outpatient diabetes clinic of hospitals in Baghdad, Iraq, for 12 months (January 2025 to January 2026). The study had informed consent of the participants.

**Study Population.**

131 people suffering from type 2 diabetes mellitus (T2DM) were included in a sequential series.





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Patients with type 1 diabetes, gestational diabetes, and acute infections, hospital stays within the last four weeks, cancer, or incapacity to give informed permission were not included. Patients who were eligible and presented throughout the trial period were enrolled one after the other.

**Data Collection**

Demographic data were collected on a standard data collection form. During clinical examination, blood pressure (average of two measures, 5 minutes apart, sitting position, using a calibrated mercury sphygmomanometer), anthropometric measures, and peripheral neuropathy (using 10-g monofilament and vibration perception threshold) were measured.

**Laboratory Investigations**

The laboratory measures were fasting blood glucose (FBG), Glycated hemoglobin (HbA1c) by using the high-performance liquid chromatography (HPLC), total cholesterol (TC), triglycerides (TG), cholesterol in low density (LDL-C), cholesterol with high density (HDL-C), serum creatinine (SCr), and estimated glomerular filtarion (eGFR) rate calculated according to the CKD-EPI equation. A fundoscopy was carried out by an oculist to evaluate the level of DR.

**Definitions of Complications**

Fundoscopic examination was used to diagnose diabetic retinopathy, which was then graded using the International Clinical Diabetic Retinopathy Severity Scale. DN was defined as UACR ≥ 30 mg/g on 2 occasions or eGFR < 60 mL/min/1.73 m². Peripheral neuropathy was diagnosed if both clinical symptoms (numbness, tingling, burning pain) and abnormal vibrating or monofilament testing were present.

**Statistical Analysis**

Data analysis was done using SPSS version 26.0. The mean ± SD for normal data and the median (IQR) for skewed data were used to express all continuous variables. For categorical variables, percentages and frequencies were given. A logistic regression analysis was used to identify independent predictors for poor control of glycemic control (HbA1c > 7.0%).

## RESULTS

**Table 1**: Enroll the clinical and demographic features of 131 patients.

| Variable | Value |
|---|---|
| Age (years) | |
| Mean ± SD | 56.8 ± 10.4 |
| Median (IQR) | 57.0 (49.0–64.0) |
| Range | 32–79 |
| Sex, n (%) | |
| Male | 74 (56.5%) |
| Female | 57 (43.5%) |
| BMI (kg/m²) | |
| Mean ± SD | 29.3 ± 5.1 |
| Median (IQR) | 28.7 (25.6–32.4) |
| BMI Category, n (%) | |
| Normal (18.5–24.9) | 28 (21.4%) |
| Overweight (25.0–29.9) | 52 (39.7%) |
| Obese (≥ 30.0) | 51 (38.9%) |
| Diabetes Duration (years) | |
| Mean ± SD | 9.6 ± 5.8 |
| Median (IQR) | 8.0 (5.0–13.0) |
| Smoking Status, n (%) | |
| Never smoker | 71 (54.2%) |
| Former smoker | 38 (29.0%) |
| Current smoker | 22 (16.8%) |
| Family History of Diabetes, n (%) | |
| Yes | 89 (67.9%) |
| No | 42 (32.1%) |
| Diabetes Treatment, n (%) | |
| Oral hypoglycemics only | 68 (51.9%) |





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|---|---|
| Insulin only | 24 (18.3%) |
| Combined (oral + insulin) | 39 (29.8%) |

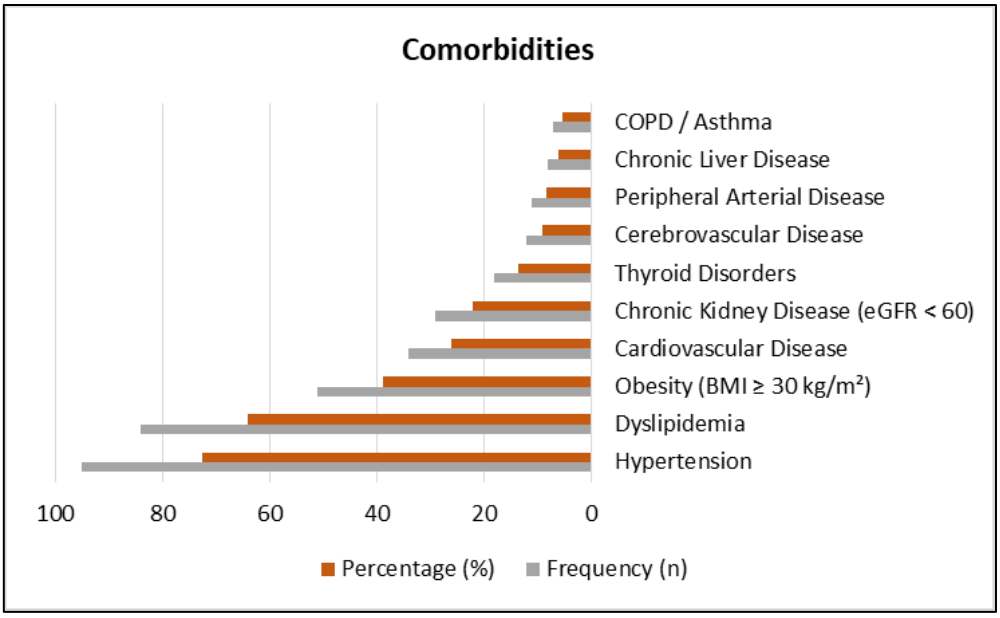


**Figure 1**: Classification of the chronic comorbidities in the 131-study population.

**Table 2**: Laboratory outcomes.

| Parameter | Hypertensive (n = 95) | Non-Hypertensive (n = 36) |
|---|---|---|
| Age (years), Mean ± SD | 58.9 ± 9.6 | 51.2 ± 10.8 |
| BMI (kg/m²), Mean ± SD | 30.1 ± 5.0 | 27.2 ± 4.8 |
| Diabetes Duration (years), Mean ± SD | 10.8 ± 5.9 | 6.4 ± 4.2 |
| HbA1c (%), Mean ± SD | 8.4 ± 1.6 | 7.2 ± 1.1 |
| HbA1c (%), Median (IQR) | 8.1 (7.2–9.4) | 7.0 (6.5–7.8) |
| FBG (mg/dL), Mean ± SD | 168.4 ± 48.2 | 139.6 ± 32.7 |
| FBG (mg/dL), Median (IQR) | 158.0 (132.0–196.0) | 134.0 (118.0–156.0) |
| Total Cholesterol (mg/dL), Mean ± SD | 214.6 ± 42.3 | 192.8 ± 35.1 |
| Triglycerides (mg/dL), Mean ± SD | 186.2 ± 68.4 | 152.3 ± 51.6 |
| Triglycerides (mg/dL), Median (IQR) | 172.0 (138.0–224.0) | 142.0 (112.0–182.0) |
| LDL-C (mg/dL), Mean ± SD | 132.4 ± 36.8 | 118.6 ± 30.2 |
| HDL-C (mg/dL), Mean ± SD | 41.2 ± 9.8 | 46.8 ± 11.4 |
| Serum Creatinine (mg/dL), Mean ± SD | 1.18 ± 0.42 | 0.94 ± 0.28 |
| eGFR (mL/min/1.73m²), Mean ± SD | 72.4 ± 22.6 | 88.6 ± 18.3 |
| UACR (mg/g), Median (IQR) | 68.4 (22.6–142.8) | 18.2 (8.4–42.6) |
| SBP (mmHg), Mean ± SD | 148.6 ± 16.4 | 124.2 ± 10.8 |
| DBP (mmHg), Mean ± SD | 92.4 ± 10.2 | 78.6 ± 7.4 |

**Table 3**: Classification of hospitalization outcomes in correlation with the number of comorbidities.

| Parameter | 0–1 Comorbidities (n = 32) | 2–3 Comorbidities (n = 62) | ≥ 4 Comorbidities (n = 37) |
|---|---|---|---|
| Age (years) | 50.4 ± 10.2 | 57.1 ± 9.8 | 62.4 ± 8.6 |
| BMI (kg/m²) | 26.4 ± 4.2 | 29.6 ± 4.8 | 31.8 ± 5.4 |
| Diabetes Duration (years) | 5.8 ± 3.6 | 9.4 ± 5.2 | 13.6 ± 6.4 |
| HbA1c (%) | 7.1 ± 1.0 | 8.0 ± 1.4 | 9.2 ± 1.7 |
| FBG (mg/dL) | 136.2 ± 30.4 | 158.6 ± 42.8 | 184.8 ± 54.6 |
| Total Cholesterol (mg/dL) | 188.4 ± 32.6 | 208.2 ± 38.4 | 226.8 ± 46.2 |
| Triglycerides (mg/dL) | 142.6 ± 44.8 | 176.4 ± 62.4 | 204.8 ± 78.6 |
| LDL-C (mg/dL) | 114.2 ± 28.4 | 128.6 ± 34.2 | 142.8 ± 40.6 |





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|---|---|---|---|
| HDL-C (mg/dL) | 48.6 ± 11.2 | 42.4 ± 9.6 | 38.2 ± 8.4 |
| eGFR (mL/min/1.73m²) | 92.4 ± 16.8 | 76.8 ± 20.4 | 62.4 ± 24.2 |
| Serum Creatinine (mg/dL) | 0.88 ± 0.22 | 1.08 ± 0.36 | 1.38 ± 0.52 |
| SBP (mmHg) | 126.4 ± 12.6 | 142.8 ± 16.2 | 156.2 ± 18.4 |
| DBP (mmHg) | 78.4 ± 8.2 | 88.6 ± 10.4 | 96.2 ± 11.8 |

**Table 4**: Determining diabetes complications according to hypertension status.

| Complications | Total (N = 131) | Hypertensive (n = 95) | Non-Hypertensive (n = 36) | p-value |
|---|---|---|---|---|
| Microvascular Complications | | | | |
| Diabetic Retinopathy | 48 (36.6%) | 42 (44.2%) | 6 (16.7%) | 0.003 |
| Diabetic Nephropathy | 41 (31.3%) | 36 (37.9%) | 5 (13.9%) | 0.008 |
| Peripheral Neuropathy | 56 (42.7%) | 46 (48.4%) | 10 (27.8%) | 0.032 |
| Autonomic Neuropathy | 19 (14.5%) | 16 (16.8%) | 3 (8.3%) | 0.218 |
| Macrovascular Complications | | | | |
| Coronary Artery Disease | 28 (21.4%) | 25 (26.3%) | 3 (8.3%) | 0.026 |
| Cerebrovascular Accident | 12 (9.2%) | 11 (11.6%) | 1 (2.8%) | 0.124 |
| Peripheral Arterial Disease | 11 (8.4%) | 10 (10.5%) | 1 (2.8%) | 0.156 |
| Other Complications | | | | |
| Diabetic Foot Ulcer | 14 (10.7%) | 12 (12.6%) | 2 (5.6%) | 0.242 |
| Erectile Dysfunction (males) | 22/74 (29.7%) | 19/54 (35.2%) | 3/20 (15.0%) | 0.092 |
| Any Microvascular Complication | 72 (55.0%) | 60 (63.2%) | 12 (33.3%) | 0.002 |
| Any Macrovascular Complication | 38 (29.0%) | 34 (35.8%) | 4 (11.1%) | 0.005* |

**Table 5**: Analysis of logistic regression related to poor glycemic control indicators.

| Variable | OR (95% CI) | p-value |
|---|---|---|
| Hypertension (yes vs. no) | 3.12 (1.41–6.89) | 0.005 |
| Number of Comorbidities (per unit increase) | 1.78 (1.22–2.60) | 0.003 |
| Age (per 10-year increase) | 1.18 (0.86–1.62) | 0.304 |
| Diabetes Duration (per 5-year increase) | 1.46 (1.04–2.05) | 0.028* |
| BMI (per 5 kg/m² increase) | 1.32 (0.92–1.89) | 0.128 |
| Sex (male vs. female) | 1.14 (0.54–2.42) | 0.724 |
| Smoking (current vs. never/former) | 1.38 (0.54–3.52) | 0.498 |
| Dyslipidemia (yes vs. no) | 1.52 (0.68–3.40) | 0.306 |
| CKD (eGFR < 60) (yes vs. no) | 1.64 (0.62–4.34) | 0.318 |
| Insulin Use (yes vs. no) | 2.24 (1.02–4.92) | 0.044 |

## DISCUSSION

The most common comorbidities were hypertension (72.5%) and a mean of 2.7 comorbidities per patient. Our results indicate that there is significantly poorer glycemic control, adverse lipid profiles, and impaired renal function in the hypertensive diabetic patients as compared to their non-hypertensive counterparts [Tatsumi, Y., & Ohkubo, T. *2017*].

Moreover, the relationship between hypertension and poor glycemic control (HbA1c > 7.0%) was comparable with some studies [Mills, K. T. *et al*., 2016; Mills, K. T. *et al*., 2016; Mills, K. T. *et al*., 2016] carried out in the USA (adjusted OR 3.12). The UK studies [Mills, K. T. *et al*., 2016; Mills, K. T. *et al*., 2016] have consistently demonstrated the pathophysiological pathways shared by hypertension. The higher HbA1c (8.4% vs. 7.2%), fasting blood glucose, and atherogenic lipid profiles (increased LDL-C and triglycerides, decreased HDL-C) we observed in the hypertensive patients were similar to those results from the ACCORD trial, which emphasized the hazardous association of metabolic syndrome components.

In addition, our data showed that patients with ≥4 comorbidities had significantly higher HbA1c, lower eGFR, and elevated urine albumin-to-creatinine ratios (UACR) than patients with 0–1 comorbidities. This dose-dependent reduction in renal function supports recent epidemiological evidence of the increased speed of progression of





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diabetic nephropathy in individuals with multimorbidity [Mills, K. T. *et al*., 2016]. Furthermore, the much higher rates of microvascular complications (retinopathy, nephropathy, and neuropathy) and coronary artery disease in hypertensive patients are similar to those of long-term cohort studies such as those in Candia [Piette, J. D., & Kerr, E. A. 2006].

Furthermore, hypertension and the burden of comorbidities were independent predictors of poor glycemic control, emphasizing the need for regular screening and intensive control of blood pressure, lipids, and renal function to prevent disease progression. Longitudinal studies [Kerr, E. A. *et al*., 2007; Piette, J. D., & Kerr, E. A. 2006; Pentakota, S. R. *et al*., 2012] are warranted in the future to determine if targeted intensive multimorbidity management can slow the rate of progression of diabetic complications in this vulnerable population.

## CONCLUSION

In conclusion, the clinical profile along with health outcomes of patients suffering from diabetes are further adversely affected by hypertension and a rising load of chronic comorbidities. Patients with hypertension and multiple comorbidities have significantly worse glycemic control, worse lipid parameters, and faster renal failure; in addition, they have a much higher risk of microvascular and macrovascular complications. Impaired glycemic control (HbA1c > 7.0%) is significantly and significantly correlated with hypertension, a greater number of comorbidities, longer duration of diabetes, and insulin use.

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