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Association of Type 2 Diabetes Mellitus with Dyslipidemia in Hypertensive Patients: A Retrospective Cross-Sectional Study

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Abstract:

Objective: This study aimed to examine the association between type 2 diabetes mellitus (T2DM) and metabolic profile alterations—particularly dyslipidemia—in patients with hypertension, and to determine whether T2DM is independently associated with dyslipidemia in this population.

Methods: This retrospective cross-sectional study included 191 patients with a documented diagnosis of hypertension followed between September 1, 2024, and September 1, 2025. Participants were categorized according to T2DM status. Demographic, clinical, and laboratory data were retrieved from electronic medical records. Continuous variables are expressed as median (interquartile range) and categorical variables as counts and percentages. Group comparisons were performed using the Mann–Whitney U and chi-square tests. Associations between variables were evaluated using Spearman's rank correlation analysis, and factors independently associated with dyslipidemia were identified using binary logistic regression analysis.

Results: Participants had a median age of 50.0 (42.0–58.0) years; 66.0% were female and 24.1% had T2DM. Triglyceride levels were significantly higher in the T2DM group (P=0.039), as were C-reactive protein concentrations (P=0.025). Dyslipidemia was present in 56.5% of patients with T2DM compared with 13.8% of those without (P<0.001). In multivariable analysis, T2DM remained independently associated with dyslipidemia (odds ratio=4.47, 95% confidence interval: 1.58–12.67; P=0.005), as did age (odds ratio=1.038; P=0.049).

Conclusion: T2DM in hypertensive individuals was significantly and independently associated with dyslipidemia, most prominently reflected by elevated triglyceride levels. Lipid screening and cardiometabolic risk management should be approached more comprehensively in hypertensive patients with T2DM.

Keywords: Type 2 Diabetes Mellitus, Triglycerides, Cardiovascular Risk, Dyslipidemia, Hypertension

Hypertension and type 2 diabetes mellitus (T2DM) are chronic disorders that significantly contribute to the global burden of cardiovascular disease. Recent epidemiological reports show that hypertension remains highly prevalent worldwide and affects a considerable proportion of adults [1]. Similarly, the prevalence of T2DM is increasing, and its coexistence with

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hypertension has been widely reported. When hypertension and T2DM coexist, the risk of atherosclerotic cardiovascular disease becomes greater than that observed with either condition alone, thereby increasing the complexity of clinical management [2]. T2DM involves not only disturbances in glucose regulation but also significant changes in lipid metabolism. The dyslipidemic pattern typically observed in individuals with T2DM is characterized by elevated triglyceride (TG) concentrations, decreased high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol concentrations that are often within the normal range or only mildly increased [3]. TG rich lipoproteins and their circulating remnants contribute to atherogenesis and have been linked to a higher incidence of cardiovascular events [4]. Accordingly, T2DM may contribute to vascular injury through changes in both the composition and circulating levels of lipoproteins. According to contemporary European cardiovascular guidelines, the coexistence of hypertension and T2DM places patients in the high or very high cardiovascular risk category [2].

However, real-world clinical data evaluating the specific contribution of T2DM to metabolic profile alterations and the prevalence of dyslipidemia among hypertensive individuals remain limited. In routine outpatient settings, hypertensive patients are not uniformly screened for the full spectrum of cardiometabolic abnormalities, and the additional metabolic burden imposed by coexisting T2DM is often underappreciated despite its substantial implications for long-term cardiovascular risk. Clarifying how dyslipidemia is distributed among hypertensive individuals with and without T2DM is therefore directly relevant to risk stratification, the intensity of lipid screening, and the planning of follow-up strategies in everyday clinical practice. Assessing the presence of T2DM in the hypertensive population in conjunction with lipid parameters, inflammatory markers, and renal function indicators may enable more precise cardiovascular risk stratification. Real-world outpatient data, derived from unselected patients followed under routine conditions, can complement evidence from clinical trials by reflecting the metabolic profiles encountered in actual practice and may help identify subgroups in whom more comprehensive lipid evaluation is particularly warranted.

The mechanistic basis of diabetic dyslipidemia is primarily related to insulin resistance, enhanced hepatic production of very-low-density lipoprotein (VLDL), and changes in lipoprotein lipase activity [3]. Nevertheless, the severity and clinical expression of these metabolic disturbances may vary considerably among hypertensive individuals. Therefore, clarifying the independent association between T2DM and dyslipidemia in patients with hypertension is of substantial importance for clinical screening and follow-up strategies.

This study examined the relationship between T2DM and alterations in the metabolic profile—particularly dyslipidemia—among individuals with hypertension, and assessed whether T2DM is independently associated with dyslipidemia in this population.

METHODS

Study Design and Population

This investigation followed a retrospective cross-sectional design. Data were obtained from medical records of patients with a documented diagnosis of hypertension who were followed at the General Internal Medicine Outpatient Clinic of Gaziantep University Sahinbey Research and Practice Hospital between September 1, 2024, and September 1, 2025. Individuals aged 18–65 years who had hypertension and documented glycemic and lipid measurements obtained during the same visit were included.

Patients with type 1 diabetes, secondary hypertension, pregnancy, active infection or malignancy, advanced renal failure, or missing essential clinical or laboratory data were excluded. Participants were divided into two groups based on the presence of a T2DM diagnosis in the medical records: patients with T2DM [T2DM(+)] and those without T2DM [T2DM(−)].

Data Collection and Definitions

Demographic data (age, gender), clinical measurements (systolic and diastolic blood pressure measurements), and laboratory parameters were obtained from the electronic patient record system. Glycemic status was assessed using fasting plasma glucose and HbA1c measurements. Within the lipid

profile, TG, LDL, and HDL levels were recorded, and the TG-to-HDL ratio was derived. Dyslipidemia was defined as the presence of either: (i) a prior physician-documented diagnosis of dyslipidemia in the electronic medical records, or (ii) at least one lipid parameter meeting the NCEP ATP III abnormal thresholds (low-density lipoprotein cholesterol ≥130 mg/dL, triglyceride ≥150 mg/dL, or high-density lipoprotein cholesterol <40 mg/dL in men or <50 mg/dL in women) [5]. C-reactive protein (CRP) levels and neutrophil-to-lymphocyte ratio (NLR), calculated from complete blood count results, were used as markers of systemic inflammation. Estimated glomerular filtration rate (eGFR) was used to assess renal function. Values of eGFR below 90 mL/min/1.73 m² were interpreted as indicative of reduced renal function. All data were anonymized for analysis.

Sample Size Calculation

An a priori power analysis was conducted to estimate the number of participants required to detect differences between two independent groups for a continuous outcome. Based on previously reported effect sizes from studies evaluating lipid profile differences in T2DM populations [6] in the literature, a medium standardized effect (Cohen's d=0.60) was assumed. Calculations were performed assuming a two-tailed α=0.05 and 80% power (1-β=0.80), determining that a total of at least 90 participants were needed, with at least 45 participants in each group. To compensate for possible missing data, the planned sample size was increased, and the final analysis was performed on 191 patients.

Ethical Approval

Ethical approval for this retrospective cross-sectional investigation was granted by the Gaziantep University Non-Interventional Clinical Research Ethics Committee (Decision No: 2026/98; Approval Date: 04 February 2026). Given the retrospective design of the study and the exclusive use of anonymized data extracted from existing electronic medical records, individual informed consent was not obtained from the participants. Institutional authorization to access electronic medical records was obtained from the chief physician's office at Gaziantep University Sahinbey Research and Practice Hospital. All patient information was anonymized before statistical analysis. All procedures were carried out in compliance with the principles of the Declaration of Helsinki.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was examined using the Shapiro–Wilk test. When normal distribution was not observed, non-parametric approaches were applied. Continuous data are presented as median and interquartile range (IQR), whereas categorical variables are expressed as frequencies and percentages. Group comparisons according to T2DM status were performed using the Mann–Whitney U test for continuous variables and the Chi-square test for categorical variables. For the Mann–Whitney U test, effect size was calculated as $r = Z/\sqrt{N}$. Associations between variables were explored using Spearman's rank correlation analysis. To identify factors independently associated with dyslipidemia, binary logistic regression analysis was applied. Variables reaching statistical significance in univariate analyses were entered into the multivariable model. Missing data were handled using pairwise deletion in descriptive and correlation analyses, so that each comparison was performed on the maximum number of patients with available data for the relevant variables. For the binary logistic regression analysis, listwise (complete-case) deletion was applied, resulting in a final analytic sample of 167 patients with complete data for all variables entered into the multivariable model. No imputation procedures were used. Multicollinearity was assessed using variance inflation factor (VIF). Odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were reported. Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. A two-sided P-value <0.05 was considered statistically significant.

RESULTS

The final analysis comprised 191 patients with hypertension. Participants had a median age of 50.0 years (IQR: 42.0–58.0), and 66.0% were female.

TABLE 1. Demographic and Clinical Characteristics of the Patients (n=191)

Variable	Value
Age (years)	50.0 (42.0-58.0)
Sex, n (%)	
Male	65 (34.0)
Female	126 (66.0)
Systolic BP (mmHg)	132.0 (124.0-145.0)
Diastolic BP (mmHg)	78.0 (71.0-85.0)
T2DM, n (%)	46 (24.1)
Dyslipidemia, n (%)	46 (24.1)
Glycemic parameters	
Glucose (mg/dL)	95.0 (85.0-108.0)
HbA1c (%)	5.5 (5.1-5.9)
Lipid profile	
TG (mg/dL)	145.0 (101.0-209.0)
LDL (mg/dL)	132.0 (108.0-159.0)
HDL (mg/dL)	51.0 (43.0-60.0)
TG/HDL	2.8 (1.6-4.2)
Other parameters	
eGFR (mL/min/1.73 m²)	101.0 (90.0-111.0)
CRP (mg/L)	3.2 (1.7-6.5)
NLR	1.8 (1.4-2.3)

Data are shown as median (interquartile range; Q1–Q3) or counts (percentages) where appropriate. BP, blood pressure; T2DM, type 2 diabetes mellitus; HbA1c, glycated hemoglobin; TG, triglycerides; LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein; NLR, neutrophil-to-lymphocyte ratio.

T2DM was present in 24.1% of patients (n=46), and dyslipidemia was observed at the same rate (24.1%). Systolic blood pressure (BP) had a median value of 132.0 mmHg (IQR: 124.0–145.0), while diastolic BP showed a median of 78.0 mmHg (IQR: 71.0–85.0). Median fasting glucose was 95.0 mg/dL (IQR: 85.0–108.0), and median HbA1c was 5.5% (IQR: 5.1–5.9). TG levels showed a median of 145.0 mg/dL (IQR: 101.0–209.0), whereas LDL cholesterol and HDL cholesterol had median values of 132.0 mg/dL (IQR: 108.0–159.0) and 51.0 mg/dL (IQR: 43.0–60.0), respectively. The TG/HDL ratio had a median of 2.8 (IQR: 1.6–4.2). Among other biochemical parameters, median eGFR was 101.0 mL/min/1.73 m² (IQR: 90.0–111.0), CRP was 3.2 mg/L (IQR: 1.7–6.5), and NLR was 1.8 (IQR: 1.4–2.3) (Table 1).

Comparison by T2DM status showed that median glucose values were higher in the T2DM(+) group compared with the T2DM(−) group [123.0 (98.0–163.0) vs 92.0 (84.0–102.0) mg/dL; P<0.001, r=0.42]. Similarly, HbA1c levels were greater in the T2DM(+) group [6.7% (6.1–8.8) vs 5.4% (5.0–5.6); P<0.001, r=0.62], with a large effect size observed for HbA1c (r=0.62). With respect to lipid parameters, TG concentrations were higher among patients in the T2DM(+) group [159.0 (125.0–233.0) vs 140.0 (93.2–198.5) mg/dL; p=0.039, r=0.16]. CRP values were likewise higher in the T2DM(+) group [4.6 (2.2–10.0) vs 3.0 (1.6–5.8) mg/L; P=0.025, r=0.16]. Although the TG/HDL ratio tended to be higher in the T2DM(+) group, this difference approached but did not reach statistical significance (P=0.060). LDL, HDL, BP measurements, and eGFR did not differ significantly between groups (Table 2).

Analysis of categorical variables according to T2DM status demonstrated a strong association between diabetes and dyslipidemia (χ^2=32.574, P<0.001). Dyslipidemia was more frequent in the T2DM(+) group (56.5%) than in the T2DM(−) group (13.8%). Sex distribution and the prevalence of reduced renal function (eGFR <90 mL/min/1.73 m²) did not differ significantly between groups (Table 3). Correlation analysis indicated a weak positive association between HbA1c and TG levels (rho=0.213, P=0.007). Similarly, HbA1c showed a positive association with LDL cholesterol (rho=0.160, P=0.041), and glucose levels were weakly associated with TG concentrations (rho=0.178, P=0.019). TG levels were inversely associated with HDL cholesterol (rho=−0.259, P=0.037). HbA1c was inversely associated with eGFR (rho=−0.203, P=0.008) (Table 4). Logistic regression analyses were performed on 167 patients who had complete data for all variables entered into the model; 24 patients (12.6%) were excluded because of missing values for HbA1c or other model covariates. Univariate logistic regression analysis evaluating factors associated with dyslipidemia showed that T2DM (OR=8.471, 95% CI: 3.818–18.793, P<0.001), age (OR=1.045, P=0.007) and HbA1c (OR=1.928, P<0.001) were significantly associated with dyslipidemia. T2DM was linked to an

TABLE 2. Comparison of Clinical and Metabolic Parameters According to T2DM Status

Variables	T2DM(+) (n=46)	T2DM(-) (n=145)	Z	P-value	r (effect size)
Age (years)	52.0 (45.0-60.5)	49.0 (40.0-58.0)	1.70	0.090	0.12
Systolic BP (mmHg)	135.5 (124.0-149.5)	131.0 (124.0-143.0)	1.11	0.268	0.08
Diastolic BP (mmHg)	77.5 (72.0-81.0)	79.0 (71.0-86.0)	-0.58	0.560	0.04
Glucose (mg/dL)	123.0 (98.0-163.0)	92.0 (84.0-102.0)	5.74	**<0.001**	0.42
HbA1c (%)	6.7 (6.1-8.8)	5.4 (5.0-5.6)	8.10	**<0.001**	0.62
TG (mg/dL)	159.0 (125.0-233.0)	140.0 (93.2-198.5)	2.07	**0.039**	0.16
LDL (mg/dL)	129.0 (105.0-160.0)	133.5 (109.0-159.5)	-0.29	0.771	0.02
HDL (mg/dL)	50.5 (45.2-56.8)	51.0 (42.0-62.0)	-0.34	0.738	0.04
TG/HDL ratio	3.5 (2.4-4.7)	2.5 (1.4-4.0)	1.89	0.060	0.23
eGFR (mL/min/1.73 m²)	101.0 (88.0-109.0)	101.5 (91.0-112.0)	-0.56	0.574	0.04
CRP (mg/L)	4.6 (2.2-10.0)	3.0 (1.6-5.8)	2.24	**0.025**	0.16
NLR	1.8 (1.4-2.2)	1.8 (1.4-2.4)	-0.23	0.821	0.02

Data are shown as median (interquartile range). T2DM, type 2 diabetes mellitus; BP, blood pressure; HbA1c, glycated hemoglobin; TG, triglycerides; LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein; NLR, neutrophil-to-lymphocyte ratio.

Group comparisons were performed using the Mann–Whitney U test. Effect size was calculated as $r = Z/\sqrt{N}$ and interpreted as small (<0.30), moderate (0.30–0.50), or large (>0.50). Pairwise deletion was used for missing values; sample sizes shown reflect available data per group. Statistically significant P-values are shown in bold.

approximately 8.5-fold higher likelihood of dyslipidemia. In multivariable analysis, T2DM and age remained independently associated with dyslipidemia. After controlling for age, sex, and HbA1c, T2DM was associated with a 4.47-fold higher odds of dyslipidemia (OR=4.473, 95% CI: 1.580–12.668, P=0.005). Each additional year of age corresponded to a 3.8% rise in the odds of dyslipidemia (OR=1.038, 95% CI: 1.000–1.077, P=0.049). HbA1c did not retain statistical significance in the multivariable model (P=0.108), suggesting that its effect was largely explained by the presence of T2DM. Multicollinearity was not observed, with all variance inflation factor (VIF) values below 5. The

TABLE 3. Association Between T2DM and Categorical Variables

Variables	T2DM(+)	T2DM(-)	χ^2	P-value
Sex			0.000	1.000
Male	16 (34.8)	49 (33.8)		
Female	30 (65.2)	96 (66.2)		
Dyslipidemia			32.574	**<0.001**
Present	26 (56.5)	20 (13.8)		
Absent	20 (43.5)	125 (86.2)		
eGFR <90 mL/min/1.73m²			0.194	0.660
Present	13 (28.3)	34 (23.6)		
Absent	33 (71.7)	110 (76.4)		

Data are shown as n (%). T2DM, type 2 diabetes mellitus; eGFR, estimated glomerular filtration rate.

Comparisons between groups were performed using the chi-square test. Statistically significant P-value is shown in bold.

TABLE 4. Correlations Between Glycemic and Lipid Parameters

Variable Pair	n	rho	P-value
HbA1c - TG	159	0.213	**0.007**
HbA1c - LDL	164	0.160	**0.041**
HbA1c - HDL	163	0.032	0.806
Glucose - TG	173	0.178	**0.019**
Glucose - LDL	179	0.042	0.581
TG - HDL	165	-0.259	**0.037**
HbA1c - CRP	168	0.141	0.068
TG - CRP	172	0.042	0.586
HbA1c - eGFR	169	-0.203	**0.008**
TG - eGFR	174	-0.045	0.553

HbA1c, glycated hemoglobin; TG, triglycerides; LDL, low-density lipoprotein cholesterol; HDL, high-density lipoprotein cholesterol; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate.

Spearman's rank correlation analysis was used. ρ (rho) represents the Spearman correlation coefficient. Pairwise deletion was used; sample size varies slightly across pairs due to missing values for individual laboratory parameters.

Statistically significant P-value are shown in bold.

Hosmer–Lemeshow test suggested acceptable model calibration (χ^2=4.54, P=0.806). The model demonstrated overall statistical significance (Omnibus χ^2=37.29, P<0.001). The Nagelkerke R^2 value was 0.200, indicating that approximately 20% of the variance in dyslipidemia was accounted for by the model (Table 5).

DISCUSSION

In this cohort of hypertensive patients, T2DM demonstrated a strong and independent association with dyslipidemia. Multivariable modeling indicated that T2DM was associated with an approximately 4.5-fold higher odds of dyslipidemia, highlighting that the combination of diabetes and hypertension corresponds to a more adverse metabolic profile. These findings suggest that T2DM represents an important metabolic component within the hypertensive population rather than merely a coexisting condition.

The coexistence of hypertension and T2DM has long been associated with increased cardiovascular morbidity and mortality [1, 7, 8]. For this reason, current cardiovascular prevention and diabetes guidelines classify individuals with both conditions as having high or very high cardiovascular risk [7, 9]. However, in daily clinical practice, the biochemical pathways underlying this excess risk are not always explored in sufficient detail. These results indicate that dyslipidemia may constitute one of the metabolic alterations underlying this elevated cardiovascular risk.

The higher TG levels observed in the T2DM group are consistent with the well-known pattern of diabetic dyslipidemia [3, 4]. Although the underlying mechanisms were not directly assessed in the present study, this pattern has been attributed in the literature to insulin resistance, with proposed contributions from enhanced hepatic VLDL synthesis, diminished lipoprotein lipase activity, and greater circulation of TG-rich lipoproteins [3]. Such lipoproteins contribute to atherogenic processes and have been related to a higher incidence of cardiovascular events [10, 11]. Therefore, elevated TG levels in hypertensive individuals with T2DM may represent not only a biochemical abnormality but also a clinically relevant indicator of increased cardiovascular risk.

The lack of a statistically significant difference in LDL cholesterol levels warrants attention. In diabetic dyslipidemia, LDL levels often remain within the normal range, while alterations in particle composition—including decreases in particle size and increases in density—have been described in the literature [4, 12]. Although LDL particle size and density were not directly measured in this study, small, dense LDL particles have been described as more atherogenic and have been associated with endothelial dysfunction in previous studies. Therefore, conventional LDL measurements may not fully reflect cardiovascular risk. Our results highlight the relevance of lipid composition beyond LDL quantity. Although the TG/HDL ratio was higher in the T2DM group, this difference approached statistical significance and may still be clinically relevant. The TG/HDL ratio serves as a practical and widely available marker of insulin resistance and atherogenic dyslipidemia [13] and has been linked to adverse cardiometabolic risk profiles. The increasing trend observed in our study may reflect an underlying biological relationship that could be more clearly demonstrated in larger or prospective studies. Although correlations between glycemic

TABLE 5. Logistic Regression Analysis for Dyslipidemia (n=167)

Variables	B	SE	Wald	P-value	OR	95% CI
Univariate analysis						
T2DM	2.137	0.407	27.62	**<0.001**	8.471	3.818-18.793
Age	0.044	0.016	7.16	**0.007**	1.045	1.012-1.078
Sex (male)	0.143	0.375	0.15	0.703	1.154	0.553-2.408
Systolic BP	0.007	0.011	0.37	0.543	1.007	0.985-1.029
HbA1c	0.657	0.157	17.42	**<0.001**	1.928	1.417-2.624
eGFR	-0.009	0.010	0.80	0.370	0.991	0.972-1.011
CRP	0.002	0.017	0.02	0.903	1.002	0.968-1.037
Multivariable analysis						
T2DM	1.498	0.531	7.96	**0.005**	4.473	1.580-12.668
Age	0.037	0.019	3.87	**0.049**	1.038	1.000-1.077
Sex (male)	0.201	0.430	0.22	0.640	1.222	0.527-2.836
HbA1c	0.281	0.174	2.59	0.108	1.324	0.941-1.864

B, regression coefficient; SE, standard error; BP, blood pressure; HbA1c, glycated hemoglobin; eGFR, estimated glomerular filtration rate; T2DM, type 2 diabetes mellitus; OR, odds ratio; CI, confidence interval.

The multivariable logistic regression model was performed on 167 patients with complete data for all variables (T2DM, age, sex, HbA1c) using listwise (complete-case) deletion; 46 dyslipidemia events were included in the analysis.

Multicollinearity was assessed using variance inflation factors (all <5), and model calibration was evaluated using the Hosmer–Lemeshow goodness-of-fit test (χ^2=4.54, P=0.806). Statistically significant P-value are shown in bold.

parameters and lipid levels were weak, they were statistically significant. The positive correlations between HbA1c and TG and LDL are consistent with the reported effects of chronic hyperglycemia on lipid metabolism [3, 14]. However, the loss of HbA1c's independent significance in multivariable analysis is noteworthy. This finding suggests that dyslipidemia in T2DM may not be explained solely by the degree of glycemic control but may reflect a broader spectrum of metabolic and hormonal alterations. In the literature, T2DM has been characterized as a systemic metabolic condition involving insulin resistance, adipokine imbalance, and chronic inflammation; however, these specific pathways were not directly evaluated in the present study.

The higher CRP levels observed in the T2DM group are consistent with this interpretation. Chronic low-grade inflammation has been described as a key component in the pathophysiology of both T2DM and atherosclerosis [15], and hypertension has similarly been associated with endothelial stress and vascular inflammatory processes in previous studies. As specific inflammatory mediators were not assessed in the present analysis beyond CRP, the coexistence of these two conditions may be associated with a greater inflammatory burden, together with dyslipidemia, potentially contributing to increased cardiovascular risk.

The negative correlation between HbA1c and eGFR suggests that renal function may be adversely affected in the setting of poorer glycemic control. Diabetic nephropathy represents a prominent clinical expression of microvascular injury [16, 17]. Although no statistically significant difference in eGFR was observed between groups, the identified correlation highlights the relevance of renal monitoring in hypertensive patients with T2DM.

The observation that T2DM remained independently associated with dyslipidemia in logistic regression analysis constitutes one of the principal findings of this study. The relationship between age and dyslipidemia aligns with the progressive accumulation of cardiometabolic risk over time [18]. However, the persistence of this association after adjustment for age, sex, and HbA1c may suggest that T2DM is closely interrelated with other components of the metabolic risk cluster in hypertensive

individuals, although the cross-sectional design precludes inferences regarding the directionality or relative weight of these relationships.

Clinical Implications

These findings suggest that hypertensive patients with T2DM may benefit from routine screening for atherogenic dyslipidemia, even in the absence of elevated LDL cholesterol levels. Lipid assessment may need to consider not only LDL levels but also parameters such as TG and the TG/HDL ratio. Current guidelines recommend comprehensive and aggressive risk management in individuals with multiple risk factors [7, 9, 19]. Our study provides real-world data that are consistent with this approach.

Strengths and Limitations

Several strengths of this study should be acknowledged. First, it is based on real-world clinical data from a clearly defined cohort of hypertensive patients, which enabled the assessment of metabolic interactions, T2DM, and dyslipidemia in routine clinical practice. Second, both univariate and multivariable analytical approaches were applied, allowing the independent association between T2DM and dyslipidemia to be evaluated after adjustment for potential confounders. In addition, the inclusion of multiple biochemical parameters, including TG and inflammatory markers, allowed a more comprehensive metabolic characterization beyond conventional lipid measures.

Nevertheless, certain limitations warrant acknowledgment. The retrospective cross-sectional nature of the study does not allow causal conclusions and restricts assessment of temporal relationships between T2DM and dyslipidemia. The study population was derived from a single center, which may reduce the external validity of the results. Importantly, detailed information on the use of lipid-lowering agents (e.g., statins, fibrates), antihypertensive medications, and antidiabetic therapies was not consistently available in the electronic records, and could not be incorporated into the analyses. As these agents may directly modify lipid concentrations, residual confounding by medication use cannot be excluded. Similarly, anthropometric data such as body mass index and waist circumference—both well-established correlates of insulin resistance and atherogenic dyslipidemia—were not systematically recorded, precluding their inclusion as covariates. Finally, information on lifestyle factors including dietary patterns, physical activity levels, smoking status, and treatment adherence was not accessible, and these factors may have additionally influenced the observed lipid profiles. Future prospective, multicenter investigations incorporating comprehensive data on medication use, anthropometric measures, and lifestyle factors would be valuable to further validate these findings and to more clearly elucidate the longitudinal metabolic interactions between hypertension, T2DM, and dyslipidemia.

CONCLUSION

In this retrospective cross-sectional study, T2DM was independently associated with dyslipidemia among hypertensive patients, with this association mainly reflected by higher triglyceride levels. These findings support the importance of careful lipid evaluation and comprehensive cardiometabolic risk assessment in hypertensive patients with T2DM.

Ethics Approval and Consent to Participate

This study was approved by the Gaziantep University Non-Interventional Clinical Research Ethics Committee (Decision No: 2026/98; date: 04.02.2026). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. The study was conducted retrospectively using anonymized patient data, and institutional permission was obtained from the chief physician's office following ethics committee approval and prior to data collection.

Clinical Trial Registration

Not Available.

Data Availability

The data supporting the findings of this study are not publicly available due to institutional and ethical

restrictions. The data were obtained from hospital electronic medical records and analyzed in an anonymized form. Access to the data may be considered upon reasonable request to the corresponding author, subject to approval by the hospital administration.

Authors' Contribution

Study Conception: KOL, SB, AB; Study Design: KOL, SB; Supervision: KOL, SB; Funding: N/A; Materials: KOL, SB, AB; Data Collection and/or Processing: KOL, AB; Statistical Analysis and/or Data Interpretation: SB; Literature Review: KOL, AB; Manuscript Preparation: KOL, SB, AB; and Critical Review: KOL, SB, AB.

Conflict of Interest

The author(s) disclosed no conflict of interest during the preparation or publication of this manuscript.

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Generative Artificial Intelligence Statement

ChatGPT (GPT-5.4 Thinking, Open AI) was used solely for language polishing and grammar correction. No artificial intelligence tools were used for data analysis, data interpretation, or the generation of scientific conclusions. The authors take full responsibility for the final content of the manuscript.

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