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COMPARISON OF GLYCEMIC AND METABOLIC PARAMETERS BETWEEN TYPE 2 DIABETES MELLITUS PATIENTS AND HEALTHY CONTROLS IN KASHMIR: A CASE-CONTROL STUDY

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Abstract

Background:
Type 2 diabetes mellitus (T2DM) is frequently associated with multiple metabolic derangements including insulin resistance, dyslipidemia, and hyperuricemia, which significantly elevate the risk of cardiovascular and renal complications. This study aimed to evaluate and compare glycemic, anthropometric, and metabolic parameters between T2DM patients and healthy controls in the Kashmiri population and to identify predictors of hyperuricemia among diabetic individuals.

Methods:
A total of 298 diagnosed T2DM patients and 310 age- and sex-matched healthy controls were enrolled. Anthropometric indices (BMI, waist circumference), fasting glucose, HbA1c, fasting insulin, HOMA-IR, lipid profile, and serum uric acid were measured. Multivariate logistic regression was used to identify predictors of hyperuricemia in T2DM.

Results:
T2DM patients had significantly higher BMI, waist circumference, fasting glucose, HbA1c, insulin levels, HOMA-IR, triglycerides, LDL-C, and serum uric acid, and lower HDL-C compared to controls ($p < 0.001$). Coexisting dyslipidemia and hyperuricemia were observed in 34.8% of T2DM patients. BMI ≥ 27 kg/m² (OR 1.84), HOMA-IR ≥ 5 (OR 2.03), and triglycerides ≥ 150 mg/dL (OR 1.77) were independent predictors of hyperuricemia. Poor glycemic control (HbA1c $\geq 7\%$) was significantly associated with increased prevalence of both dyslipidemia and hyperuricemia.

Conclusion:
T2DM patients in Kashmir exhibit significant insulin resistance and concurrent metabolic disturbances. Hyperuricemia is strongly associated with obesity, insulin resistance, and dyslipidemia. Integrated glycemic and metabolic management strategies are essential to mitigate long-term complications in this population.

Keywords: Type 2 diabetes mellitus, insulin resistance, dyslipidemia, hyperuricemia, Kashmir.

Introduction

Type 2 diabetes mellitus (T2DM) is characterized primarily by insulin resistance and dysfunction in insulin secretion, leading to chronic hyperglycemia and associated metabolic disturbances and emerged as a global health concern [1]. Diabetes mellitus is in fact a serious vascular disease with poor prognosis, and not only a disease characterized by elevated blood glucose [2]. People with diabetes comprise 8.8% of the world's population, and International Diabetes Federation (IDF) predicts that the number of cases of diabetes will rise to 642 million by 2040 [3]. Diabetes mellitus (DM) is one of the most common chronic diseases globally and continues to increase in numbers. It is among the top five causes of mortality [4]. The IDF estimates that worldwide, 415 million people have diabetes, 91% of whom have type 2 DM (T2DM) [5]. Worldwide, T2DM affects millions, contributing substantially to morbidity and mortality through cardiovascular diseases, kidney disorders, neuropathy, and other debilitating complications [6]. This increasing prevalence is exacerbated by lifestyle changes, urbanization, aging populations, and dietary transitions, particularly in developing and rapidly urbanizing regions [6].

Glycemic status, characterized by markers such as fasting blood glucose (FBG), postprandial glucose (PPG), glycated hemoglobin (HbA1c), inflammatory cytokines and insulin levels, remains central in the pathogenesis, monitoring, and therapeutic management of T2DM [7,8]. Persistent hyperglycemia induces oxidative stress and pro-inflammatory cascades, contributing to endothelial dysfunction, dyslipidemia, and hyperuricemia, further compounding cardiovascular risk [9,10]. Elevated insulin levels, reflective of insulin resistance, are associated with dysregulation of lipid metabolism, contributing significantly to the atherogenic lipid profile typically observed in T2DM patients [10].

Diabetic patients are known to have high levels of serum low-density lipoprotein (LDL), serum triglyceride (TG), and low levels of serum high-density lipoprotein (HDL). Lipid disorders, particularly dyslipidemia characterized by elevated triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), reduced high-density lipoprotein cholesterol (HDL-C), and altered lipoprotein ratios, are frequent findings in patients with T2DM [10]. Dyslipidemia contributes markedly to cardiovascular morbidity and mortality, making lipid profile monitoring and management critical components of diabetic care [11]. Moreover, insulin resistance exacerbates dyslipidemia by promoting hepatic lipogenesis and suppressing lipoprotein lipase activity, resulting in elevated circulating triglycerides and LDL particles, alongside decreased HDL cholesterol [11.]

Hyperuricemia, defined by elevated serum uric acid levels, is increasingly recognized as an independent risk factor for cardiovascular disease, hypertension, and renal impairment in individuals with diabetes [12]. Elevated uric acid levels have been linked to insulin resistance, impaired glucose tolerance, endothelial dysfunction, and chronic inflammation, making it an essential marker for assessing metabolic disturbances in diabetic populations [13]. The pathophysiological relationship between hyperuricemia and T2DM remains complex, influenced by renal dysfunction, dietary habits, genetic predisposition, and insulin resistance [14].

The Kashmiri population, residing in the northernmost region of the Indian subcontinent, represents an ethnically and genetically distinct group with unique dietary patterns, lifestyle practices, and environmental exposures [15]. Studies specific to Kashmir have reported a rising prevalence of T2DM, dyslipidemia, and hyperuricemia, emphasizing the need for targeted research and region-specific healthcare strategies [16]. Insulin resistance contributes to hyperuricemia by altering renal clearance of uric acid and promoting systemic inflammation, thereby creating a vicious cycle of metabolic derangements [17]. Hyperuricemia itself has been implicated in exacerbating insulin resistance by inducing inflammatory responses and oxidative stress, further worsening glycemic control and lipid profiles [18]. Given these intricate relationships, elucidating how glycemic status and insulin levels interact with dyslipidemia and hyperuricemia is pivotal for a comprehensive understanding of metabolic disturbances in T2DM. Furthermore, studying these interrelations in specific ethnic and regional contexts such as Kashmir is essential, considering variations in genetic predisposition, environmental factors, lifestyle, and dietary habits. Hence, this study aims to investigate the

association and interplay of glycemic parameters (FBG, HbA1c, insulin), lipid disorders (total cholesterol, TG, LDL-C, HDL-C), and hyperuricemia in T2DM patients in Kashmir.

Methodology

Methodology

Study Design

This was case-control study aimed to conduct the association between glycemic status, insulin levels, lipid disorders, and hyperuricemia in patients with type 2 diabetes mellitus (T2DM) in Kashmir.

Study Setting

The study was carried out at Department of Biochemistry, Govt Medical College Srinagar, SMHS Hospital in collaboration with the Department of Endocrinology, Superspeciality Hospital Associated Hospital during July 2023 to September 2024.

Study Population

The study subjects consists of patients clinically diagnosed with T2DM according to the American Diabetes Association (ADA) criteria 2018 [19]. Participants was recruited from outpatient diabetic clinics, endocrinology units, and medical wards.

Table 1:- ADA 2018 Criteria for Diagnosing T2DM [19]

FPG ≥126 mg/dL (7.0 mmol/L)* Fasting is defined as no caloric intake for ≥8 hours
2-hr PG ≥200 mg/dL (11.1 mmol/L) during OGTT (75-g)* Using a glucose load containing the equivalent of 75g anhydrous glucose dissolved in water
A1C ≥6.5% (48 mmol/mol)* *Performed in a lab using NGSP-certified method and standardized to DCCT assay*
Random PG ≥200 mg/dL (11.1 mmol/L) In individuals with symptoms of hyperglycemia or hyperglycemic crisis
*In absence of unequivocal hyperglycemia, result to be confirmed by repeat testing.

Inclusion Criteria

1. Patients aged 30–70 years.
2. Diagnosed cases of T2DM for at least 1 year.
3. Patients who provide written informed consent.
4. Patients from ethnic population of Kashmir.

Exclusion Criteria

1. Patients with type 1 diabetes mellitus or gestational diabetes.
2. Individuals on uric acid-lowering drugs or lipid-lowering therapy initiated within the last 3 months.
3. Patients with chronic kidney disease (stage 3 and above) or liver failure.
4. Pregnant and lactating women.
5. Patients with acute infections or inflammatory conditions.

Criteria for Controls: Non-diabetic group (NDM) who had no family history of T2DM and were recruited from an unselected population undergoing routine health check-ups.

Sample Size

The sample size was calculated using statistical power analysis, considering a 95% confidence level and 80% power to detect significant correlations between the studied variables. Based on prior regional prevalence data for dyslipidemia and hyperuricemia in diabetic populations, an estimated

sample size of 298 T2DM and 310 healthy controls was targeted to ensure sufficient statistical robustness.

Ethical Considerations

Ethical clearance was obtained from the Institutional Ethics Committee. Written informed consent was collected from each participant. The study adhered to the Declaration of Helsinki guidelines, ensuring confidentiality and the right to withdraw without any consequences.

Data Collection

Demographic and Clinical Data

A structured questionnaire was used to collect demographic information (age, sex, occupation), clinical history (duration of diabetes, medication use, comorbidities), lifestyle factors (dietary habits, physical activity, smoking), and family history of diabetes or metabolic disorders.

Anthropometric Measurements

1. Body weight (kg) and height (cm) was measured using calibrated instruments.
2. Body mass index (BMI) was calculated using the formula: BMI = weight (kg) / height (m^2).
3. Waist and hip circumferences was recorded to calculate waist-to-hip ratio (WHR).
4. Blood pressure was measured using a standardized digital sphygmomanometer.

Sample collection

5 ml blood was collected by phlebotomists from T2DM patients through venipuncture after an overnight fast of at least 10–12 h. 3 ml of collected blood was immediately transferred into lithium heparin green top BD vacutainers and centrifuged at 4000 rpm for 1 min and plasma/ serum was aliquoted into 2 ml microfuge tubes. Further, 2 ml of whole blood was collected in EDTA containing vacutainers for estimation of glycosylated hemoglobin (HbA1c).

Biochemical analysis

Estimation of glucose (by hexokinase G-6-PDH method), HbA1c (by enzymatic method), TG (by glycerol phosphate oxidase method), total cholesterol (by enzymatic method), low density lipoprotein-cholesterol (LDL-C; by measured liquid selective detergent) and high density lipoprotein-cholesterol (HDL-C; by accelerator selective detergent) was performed using standard commercially available kits (Abbott, USA), employing the principle of spectrophotometry. Samples were processed and analysed on ALLINITY CI fully automated biochemistry analyser (Abbott, USA) in the Biochemistry Diagnostic Laboratory, SMHS Hospital Srinagar within 1–2 h after collection. The normal values of biochemical parameters were as; fasting blood glucose: 100–125 mg/dl; HbA1c: <6.5%, total cholesterol: ≤200 mg/dl; TG: ≤200 mg/dl; LDL-C: ≤120 mg/dl; HDL-C: ≤40 mg/dl (M) and ≤50 mg/dl (F). Serum uric acid levels determined using the uricase-peroxidase method. UA<7.0 mg/dl.

Quantitative estimation of fasting insulin levels and HOMA-IR

Fasting Insulin levels (µIU/ml) of T2DM cases were measured using chemiluminescent micro particle immunoassay (CMIA) technology with flexible assay protocols, referred to as Chemiflex. Serum samples were quantitatively analysed on ALLINITY CI fully automatic immunoassay analyser (Abbott, USA) within 1–2 h after collection, following the ALLINITY CI insulin reagent kit (Abbott, USA) instructions. Insulin levels of 5–25 µIU/ml are considered to be normal. Insulin resistance (IR) was determined by HOMA-IR (homeostatic model assessment – insulin resistance) was calculated by using the formula: HOMA-IR = fasting serum insulin (µU/ml) × fasting plasma glucose (mg/dl)/405 . Following HOMA score was used as reference for classification of insulin resistance.

- < 3= Normal IR b) Between 3 and 5 = Moderate IR c) >5 =Severe IR

Statistical Analysis

All data were analyzed using SPSS version 21.0 (Chicago, IL) for Windows 10. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as percentages. The independent sample t-test was used to compare continuous variables between T2DM cases and controls, while the Chi-square test was applied for categorical variables. Multivariate logistic regression analysis was performed to predict role of variables. A p-value of <0.05 was considered statistically significant.

Results

A group of 298 T2DM patients and 310 healthy controls were taken for study (Table 2). Among 298 T2DM patients are 178 Males and 120 Females and in 310 healthy controls are 170 Males and 140 females. The mean age of T2DM (49.2 ±9.6 Years) and the controls were aged (47.1.±8.7).

Table 2. Demographic and Clinical Characteristics

Variable	T2DM (n=298)	Controls (n=310)	p-value
Age (years)	49.2 ± 9.6	47.1 ± 8.7	0.18
Male: Female ratio	178:120	170:140	0.23
BMI (kg/m²)	28.7 ± 4.5	24.3 ± 3.7	<0.001
Waist circumference (cm)	97.4 ± 11.3	87.5 ± 8.2	<0.001
Duration of DM (years)	8.2 ± 5.1	—	—
Hypertension (%)	54.3	17.4	<0.001*
Smokers (%)	27.2	24.6	0.48

Table 2 presents the demographic and clinical characteristics of the study participants, highlighting key differences between T2DM patients and healthy controls. The mean age and sex distribution between the two groups were comparable, with no statistically significant differences observed (p = 0.18 for age and p = 0.23 for gender ratio), indicating appropriate group matching. However, significant differences were noted in anthropometric and clinical parameters. The mean body mass index (BMI) was significantly higher in T2DM patients (28.7 ± 4.5 kg/m²) compared to controls (24.3 ± 3.7 kg/m²), with a p-value <0.001. Similarly, waist circumference was significantly elevated in T2DM individuals (97.4 ± 11.3 cm) versus controls (87.5 ± 8.2 cm), indicating increased central adiposity (p < 0.001).

Hypertension was significantly more prevalent among T2DM patients (54.3%) compared to controls (17.4%), with a p-value <0.001. No significant difference was observed in smoking status (p = 0.48).

Table 3. Glycemic Parameters and Insulin Levels

Parameter	T2DM (n=298)	Controls (n=310)	p-value
Fasting Glucose (mg/dL)	156.5 ± 35.2	89.3 ± 8.7	<0.001
HbA1c (%)	8.2 ± 1.5	5.4 ± 0.5	<0.001
Fasting Insulin (μU/mL)	19.7 ± 7.6	10.8 ± 3.2	<0.001
HOMA-IR	7.6 ± 3.1	2.4 ± 0.8	<0.001

Table 3 reveals significant differences in glycemic parameters and insulin resistance markers between T2DM patients and healthy controls. Fasting glucose levels were markedly elevated in the T2DM group (156.5 ± 35.2 mg/dL) compared to controls (89.3 ± 8.7 mg/dL), with a p-value of <0.001.

Likewise, HbA1c levels were significantly higher in T2DM subjects (8.2 ± 1.5%) than in controls (5.4 ± 0.5%). Fasting insulin levels were also significantly elevated in T2DM patients (19.7 ± 7.6 μU/mL) relative to controls (10.8 ± 3.2 μU/mL), with a p-value <0.001. Correspondingly, HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) values were substantially higher in T2DM patients (7.6 ± 3.1) compared to controls (2.4 ± 0.8).

Table-4. Lipid Profile Comparison in T2DM cases and controls.

Parameter	T2DM (n=298)	Controls (n=310)	p-value
Total Cholesterol (mg/dL)	202.7 ± 37.1	168.5 ± 28.3	<0.001
Triglycerides (mg/dL)	183.2 ± 53.5	124.8 ± 41.2	<0.001
LDL-C (mg/dL)	122.8 ± 28.9	96.2 ± 22.7	<0.001
HDL-C (mg/dL)	38.7 ± 8.5	48.5 ± 10.2	<0.001

Table 4 demonstrates significant alterations in lipid profile parameters among T2DM patients compared to healthy controls. Total cholesterol levels were significantly higher in T2DM subjects (202.7 ± 37.1 mg/dL) than in controls (168.5 ± 28.3 mg/dL), with a p-value of <0.001. Similarly, triglyceride levels were elevated in the T2DM group (183.2 ± 53.5 mg/dL) versus controls (124.8 ± 41.2 mg/dL). Low-density lipoprotein cholesterol (LDL-C), the atherogenic component of the lipid profile, was also significantly higher in T2DM patients (122.8 ± 28.9 mg/dL) compared to controls (96.2 ± 22.7 mg/dL), while high-density lipoprotein cholesterol (HDL-C), the protective lipid fraction, was significantly lower in T2DM patients (38.7 ± 8.5 mg/dL) than in controls (48.5 ± 10.2 mg/dL), both with p-values <0.001.

Table 5. Comparison of Serum Uric Acid Levels in T2DM cases and controls.

Parameter	T2DM (n=298)	Controls (n=310)	p-value
Uric Acid (mg/dL)	7.2 ± 1.4	5.6 ± 1.2	<0.001
Hyperuricemia (%)	38.9	13.5	<0.001

Table 5 highlights a statistically significant difference in serum uric acid levels between T2DM patients and healthy controls. The mean serum uric acid concentration in T2DM patients (7.2 ± 1.4 mg/dL) was markedly higher than in controls (5.6 ± 1.2 mg/dL), with a p-value of <0.001.Furthermore, the prevalence of hyperuricemia was significantly greater in the T2DM group (38.9%) compared to controls (13.5%), also with a p-value of <0.001.

Table 6. Correlation analysis among T2DM Patients

Correlation	r-value	p-value
Fasting Glucose & TG	0.41	<0.001
Fasting Glucose & Uric Acid	0.33	<0.001
HbA1c & LDL-C	0.27	<0.001
Fasting Insulin & TG	0.38	<0.001
HOMA-IR & LDL-C	0.31	<0.001
Uric Acid & TG	0.35	<0.001
Uric Acid & HOMA-IR	0.37	<0.001

The correlation analysis presented in Table 6 reveals several statistically significant associations ($p < 0.001$) among biochemical and metabolic parameters in patients with type 2 diabetes mellitus (T2DM). A moderate positive correlation was observed between fasting glucose and triglycerides (TG) ($r = 0.41$). Fasting glucose also showed a weaker but significant positive correlation with uric acid ($r = 0.33$).Hemoglobin A1c (HbA1c), a marker of long-term glycemic control, demonstrated a positive correlation with low-density lipoprotein cholesterol (LDL-C) ($r = 0.27$). Fasting insulin and TG were also positively correlated ($r = 0.38$). Additionally, HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) showed a significant correlation with LDL-C ($r = 0.31$).Moreover, uric acid exhibited significant correlations with both TG ($r = 0.35$) and HOMA-IR ($r = 0.37$).

Table-7. Prevalence of Combined Metabolic Derangements in T2DM

Metabolic Derangement	**Prevalence (%)**
Dyslipidemia only	29.2
Hyperuricemia only	7.4
Both Dyslipidemia & Hyperuricemia	34.8
Neither	28.6

Table 7 illustrates the distribution of metabolic derangements—dyslipidemia and hyperuricemia—among T2DM patients. A notable 34.8% of patients exhibited both dyslipidemia and hyperuricemia. Additionally, 29.2% had dyslipidemia alone, while 7.4% presented with isolated hyperuricemia. Only 28.6% of the T2DM patients had neither of these derangements, suggesting that more than 70% of the diabetic cohort had at least one metabolic abnormality.

Table-8. Multivariate Regression Analysis (Predictors of Hyperuricemia in T2DM)

Predictor	**Adjusted OR (95% CI)**	**p-value**
BMI ≥ 27 kg/m²	1.84 (1.21–2.81)	0.004
HOMA-IR ≥ 5	2.03 (1.33–3.10)	0.001
TG ≥ 150 mg/dL	1.77 (1.15–2.71)	0.009
Male sex	1.23 (0.82–1.85)	0.31

Table 8 presents the results of multivariate logistic regression analysis identifying independent predictors of hyperuricemia among T2DM patients. The analysis revealed that a BMI ≥ 27 kg/m² was significantly associated with increased odds of hyperuricemia (Adjusted OR: 1.84; 95% CI: 1.21–2.81; $p = 0.004$). Similarly, insulin resistance (HOMA-IR ≥ 5) was found to be a strong and significant predictor (Adjusted OR: 2.03; 95% CI: 1.33–3.10; $p = 0.001$). Elevated triglyceride levels (TG ≥ 150 mg/dL) also showed a significant association (Adjusted OR: 1.77; 95% CI: 1.15–2.71; $p = 0.009$). In contrast, male sex did not emerge as a significant predictor (Adjusted OR: 1.23; 95% CI: 0.82–1.85; $p = 0.31$).

Table 9. Subgroup Analysis: Glycemic Control and Metabolic Disturbances

HbA1c Group	Dyslipidemia (%)	Hyperuricemia (%)
Good (<7.0%)	62.4	25.3
Poor (≥7.0%)	81.9	42.8

Table 9 demonstrates the impact of glycemic control, as measured by HbA1c, on the prevalence of metabolic disturbances in T2DM patients. Individuals with poor glycemic control (HbA1c ≥ 7.0%) showed a significantly higher prevalence of both dyslipidemia (81.9%) and hyperuricemia (42.8%), compared to those with good glycemic control (HbA1c < 7.0%), who had lower rates of dyslipidemia (62.4%) and hyperuricemia (25.3%).

Discussion

This hospital-based case-control study involving 298 type 2 diabetes mellitus (T2DM) patients and 310 age- and sex-matched healthy controls provides comprehensive insights into the metabolic alterations associated with T2DM, focusing particularly on insulin resistance, dyslipidemia, hyperuricemia, and their inter-relationships. The demographic parameters including age and gender distribution were not statistically different between the groups, indicating appropriate matching and reducing potential confounding. However, clinical parameters like BMI and waist circumference were significantly higher among T2DM patients compared to controls ($p < 0.001$), confirming the strong association between obesity—particularly central obesity—and T2DM. Numerous studies have consistently demonstrated that increased adiposity, particularly visceral fat, plays a key role in the pathogenesis of insulin resistance and T2DM by promoting chronic inflammation, lipotoxicity, and adipokine dysregulation [20,21]. Hypertension was also significantly more prevalent in the diabetic cohort (54.3% vs. 17.4%, $p < 0.001$), consistent with existing evidence linking insulin resistance and endothelial dysfunction to elevated blood pressure in diabetics [22] . The coexistence of obesity, hypertension, and T2DM underscores the clustering of cardiometabolic risk factors typical of metabolic syndrome [22].

T2DM patients demonstrated significantly elevated fasting glucose, HbA1c, fasting insulin levels, and HOMA-IR indices compared to controls (all $p < 0.001$). The mean HbA1c in T2DM patients was 8.2 ± 1.5%, well above the diagnostic threshold, indicating poor glycemic control. Elevated HOMA-IR (7.6 ± 3.1 vs. 2.4 ± 0.8) is a clear marker of insulin resistance, which is central to the pathogenesis of T2DM and its complications [23]. Insulin resistance contributes to impaired glucose uptake, hepatic gluconeogenesis, and dyslipidemia. Our findings align with those of previous studies that show insulin resistance is highly prevalent in T2DM and is associated with both lipid and uric acid abnormalities [24] .

As shown in Table 4, patients with T2DM exhibited significantly elevated levels of total cholesterol, triglycerides (TG), LDL-C, and reduced HDL-C compared to controls. This pattern of atherogenic dyslipidemia, characterized by high TG, small dense LDL, and low HDL-C, is a hallmark of T2DM and insulin resistance [25] . The mean TG level in T2DM patients was 183.2 ± 53.5 mg/dL compared to 124.8 ± 41.2 mg/dL in controls. Similarly, LDL-C was elevated (122.8 ± 28.9 vs. 96.2 ± 22.7 mg/dL), while HDL-C was lower (38.7 ± 8.5 vs. 48.5 ± 10.2 mg/dL). These alterations significantly increase the risk of cardiovascular disease (CVD) in diabetics [25]. Indeed, dyslipidemia in T2DM is not just a comorbidity but a driving factor for macrovascular complications [26].

Uric acid levels were significantly higher in T2DM patients (7.2 ± 1.4 mg/dL) than in controls (5.6 ± 1.2 mg/dL), with hyperuricemia prevalence at 38.9% vs. 13.5%, respectively (Table 5). Hyperuricemia has been increasingly recognized as a marker of metabolic dysfunction and a potential contributor to insulin resistance and endothelial dysfunction . Insulin resistance may impair renal uric acid excretion, leading to elevated serum urate levels [27] . In turn, hyperuricemia may exacerbate oxidative stress and inflammation, further worsening insulin sensitivity [27]. Our study supports this bidirectional relationship by demonstrating significantly higher uric acid levels and hyperuricemia prevalence in T2DM.

Correlation analysis in Table 6 reveals several significant associations among metabolic variables within the T2DM group: Fasting glucose positively correlated with triglycerides ($r = 0.41$) and uric acid ($r = 0.33$), suggesting a link between poor glycemic control and both lipid and purine metabolism disturbances. HbA1c correlated with LDL-C ($r = 0.27$), underscoring the long-term impact of glycemic control on lipid abnormalities. Fasting insulin and HOMA-IR both showed significant correlations with TG and LDL-C, confirming the role of insulin resistance in driving dyslipidemia. Uric acid was significantly associated with both TG ($r = 0.35$) and HOMA-IR ($r = 0.37$), supporting the hypothesis that hyperuricemia and insulin resistance are interlinked. These findings are in line with previous research showing that insulin resistance contributes to both hypertriglyceridemia and reduced renal uric acid excretion [27].

Table 7 shows that over one-third (34.8%) of T2DM patients had both dyslipidemia and hyperuricemia, while 29.2% had dyslipidemia alone and 7.4% had isolated hyperuricemia. Only 28.6% were free of these metabolic derangements. These results reflect the high burden of multiple metabolic abnormalities in T2DM. This clustering is consistent with the concept of "diabesity"—where obesity, insulin resistance, dyslipidemia, and hyperuricemia frequently co-occur, creating a vicious cycle that accelerates cardiovascular and renal complications [28]. The high proportion of patients with both dyslipidemia and hyperuricemia emphasizes the need for integrated metabolic screening and management in T2DM.

The multivariate logistic regression analysis (Table 8) identified three significant independent predictors of hyperuricemia in T2DM patients: BMI ≥ 27 kg/m² (Adjusted OR = 1.84; p = 0.004), HOMA-IR ≥ 5 (Adjusted OR = 2.03; p = 0.001), Triglycerides ≥ 150 mg/dL (Adjusted OR = 1.77; p = 0.009). These findings are supported by literature indicating that both obesity and insulin resistance are strong predictors of reduced uric acid clearance [29]. Hypertriglyceridemia may also contribute to hepatic overproduction of uric acid due to increased de novo purine synthesis . Interestingly, male sex did not emerge as a significant predictor, suggesting that metabolic factors may have a stronger influence on hyperuricemia than gender in the diabetic population. These results support targeted interventions addressing insulin resistance, dyslipidemia, and weight control to reduce the burden of hyperuricemia in T2DM. Subgroup analysis (Table 9) showed that patients with poor glycemic control (HbA1c ≥ 7.0%) had markedly higher prevalence of both dyslipidemia (81.9%) and hyperuricemia (42.8%), compared to those with good glycemic control (62.4% and 25.3%, respectively). This underscores the role of chronic hyperglycemia in exacerbating metabolic derangements. Poor glycemic control promotes hepatic VLDL overproduction, impairs lipoprotein lipase activity, and leads to accumulation of small dense LDL and TG [30] . Additionally, hyperglycemia may worsen oxidative stress and renal tubular dysfunction, impairing uric acid excretion [31]. These findings reinforce the clinical value of achieving optimal HbA1c targets not only for glycemic management but also for mitigating the risk of lipid and uric acid disturbances. Further studies on large sample size are required in different ethnic populations for its prognostic and diagnostic role at timely intervention management of T2DM and its complications.

Conclusion

This case-control study highlights the profound metabolic disturbances in patients with type 2 diabetes mellitus (T2DM), particularly focusing on insulin resistance, dyslipidemia, and hyperuricemia. Despite similar demographic profiles between T2DM patients and healthy controls, the diabetic cohort demonstrated significantly higher BMI, waist circumference, and hypertension prevalence—hallmarks of metabolic syndrome. Glycemic parameters, insulin levels, and HOMA-IR were markedly elevated in T2DM patients, underscoring the central role of insulin resistance in disease pathophysiology. T2DM patients exhibited a characteristic pattern of atherogenic dyslipidemia, including elevated triglycerides, LDL-C, and reduced HDL-C, significantly increasing cardiovascular risk. Uric acid levels and the prevalence of hyperuricemia were also notably higher in diabetics, reinforcing its association with insulin resistance and metabolic dysfunction. Correlation analysis revealed significant interrelations among fasting glucose, insulin resistance, lipid profile, and uric acid levels—further emphasizing the interconnected nature of these abnormalities.

The coexistence of dyslipidemia and hyperuricemia in over one-third of the diabetic patients suggests a high burden of combined metabolic derangements. Multivariate analysis identified BMI ≥ 27 kg/m², HOMA-IR ≥ 5, and triglycerides ≥ 150 mg/dL as significant independent predictors of hyperuricemia. Subgroup analysis revealed that poor glycemic control (HbA1c ≥ 7%) was strongly associated with higher rates of both dyslipidemia and hyperuricemia. Overall, these findings underscore the importance of comprehensive metabolic assessment and control in T2DM management. Targeting glycemic control, insulin resistance, and lipid abnormalities may be crucial in mitigating the cardiometabolic burden and reducing long-term complications in this high-risk population.

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