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Original Research

Comprehensive Metabolic Profiling in Type 2 Diabetes Mellitus: Insights from Comparative Evaluation with Healthy Controls

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a multifactorial metabolic disorder characterized by insulin resistance and chronic hyperglycemia. It is often associated with obesity, dyslipidemia, hypertension, and altered renal biomarkers such as uric acid and creatinine.

Objective: To compare the body mass index (BMI), lipid profile, blood pressure, serum uric acid, and serum creatinine levels between healthy individuals and patients with Type 2 Diabetes Mellitus.

Methods: This cross-sectional study included two groups — 100 diagnosed cases of T2DM and 100 age- and sex-matched healthy controls. Anthropometric measurements, fasting lipid profile, blood pressure, uric acid, and creatinine levels were recorded and analyzed using standard protocols.

Results: Diabetic subjects showed significantly higher BMI, total cholesterol, triglycerides, LDL-C, VLDL-C, systolic and diastolic blood pressure, uric acid, and creatinine levels compared to healthy controls ($p < 0.05$). HDL-C levels were significantly lower among diabetics.

Conclusion: The findings highlight the clustering of metabolic risk factors such as obesity, dyslipidemia, hypertension, and renal dysfunction in diabetic patients, underscoring the importance of regular metabolic monitoring to prevent complications.

Keywords: Type 2 Diabetes Mellitus, Body Mass Index, Lipid Profile, Uric Acid, Creatinine, Blood Pressure, Metabolic Syndrome

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) represents one of the most prevalent and challenging non-communicable diseases globally. It is characterized by chronic hyperglycemia resulting from insulin resistance and/or inadequate insulin secretion. The increasing prevalence of T2DM has reached epidemic proportions, particularly in developing nations such as India, where rapid urbanization, sedentary lifestyles, and dietary transitions have significantly contributed to its rising burden. According to the International Diabetes Federation (IDF), over 537 million adults were living with diabetes in 2021.[1]

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T2DM is not merely a disorder of glucose metabolism but a complex metabolic syndrome that encompasses multiple interrelated abnormalities including central obesity, dyslipidemia, hypertension, endothelial dysfunction, and subclinical inflammation. These metabolic derangements together predispose individuals to macrovascular and microvascular complications such as coronary artery disease, stroke, nephropathy, and retinopathy, making diabetes one of the leading causes of morbidity and premature mortality worldwide.

Among the various anthropometric and biochemical indicators, Body Mass Index (BMI) serves as a simple yet powerful tool to evaluate obesity—a primary risk factor for insulin resistance. Excess adiposity, particularly visceral fat accumulation, alters adipokine secretion and promotes systemic inflammation, leading to impaired insulin signaling and β-cell dysfunction. This chronic inflammatory state in adipose tissue is mediated by dysregulated adipokines (e.g., increased resistin, TNF-α, IL-6), which interfere with insulin signaling

pathways.[2] The lipid profile, comprising total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), provides critical insight into the atherogenic lipid abnormalities commonly observed in diabetics. Diabetic dyslipidemia typically manifests as elevated triglycerides, low HDL-C, and an increased proportion of small, dense LDL particles, collectively contributing to accelerated atherosclerosis and heightened cardiovascular risk.[3,4] Similarly, blood pressure plays a pivotal role in the metabolic spectrum of diabetes. Hypertension frequently coexists with T2DM and further exacerbates vascular damage through mechanisms involving endothelial dysfunction, oxidative stress, and activation of the renin-angiotensin-aldosterone system.

In addition, renal parameters such as serum uric acid and creatinine serve as important biochemical markers reflecting metabolic and renal health. Elevated uric acid levels are associated with insulin resistance, oxidative stress, and inflammation, while increased serum creatinine indicates early impairment of renal function, a common long-term complication of uncontrolled diabetes.[5]

Comparing these parameters between healthy individuals and patients with T2DM offers valuable insights into the extent of metabolic disturbances associated with the disease. Such comparative analyses not only help in understanding the pathophysiological interplay among obesity, dyslipidemia, hypertension, and renal dysfunction but also assist in identifying individuals at high risk for developing complications. Early recognition and comprehensive metabolic evaluation, therefore, form the cornerstone of effective prevention and management strategies in T2DM.

MATERIALS AND METHODS

Study Design and Participants

The present study was a hospital-based, cross-sectional comparative study conducted over a period of six months in the Department of Physiology in collaboration with the Department of Medicine. The primary objective was to compare BMI, lipid profile, blood pressure, serum uric acid, and serum creatinine levels between healthy individuals and patients with T2DM.

A total of 200 subjects were enrolled and divided into two groups. **Group I (Control)** included 100 healthy individuals without any history of diabetes, hypertension, renal disease, or other major systemic illness. These participants were selected from hospital staff and attendants after thorough screening and confirmation of normal fasting blood glucose levels.

Group II (T2DM) consisted of 100 known cases of Type 2 Diabetes Mellitus with a minimum duration of one year, attending the outpatient department for regular follow-up. The diagnosis of diabetes was confirmed based on the American Diabetes Association (ADA) criteria, *i.e.*, fasting blood sugar (FBS) ≥126 mg/dL and/or glycated hemoglobin (HbA1c) ≥6.5%.[6] All participants were aged between 30 and 65 years and were matched for age and sex distribution to minimize confounding effects.

Exclusion Criteria

Subjects with Type 1 Diabetes Mellitus, chronic kidney disease, known liver disorders, thyroid dysfunction, or any acute infection or malignancy were excluded from the study. In addition, individuals on lipid-lowering, uric acid–lowering, or nephrotoxic drugs were not included to avoid biochemical interference.

Data collection and measurements

After obtaining informed consent from all participants, detailed clinical and demographic data were recorded, including age, sex, duration of diabetes, medical history, and lifestyle factors. Anthropometric measurements were performed following standardized procedures. Body weight was measured to the nearest 0.1 kg using a calibrated digital scale, and height was recorded to the nearest 0.1 cm using a stadiometer.

Blood pressure was measured using a standard mercury sphygmomanometer in the sitting position after 10 minutes of rest. Two consecutive readings were taken five minutes apart, and the average of the two was considered for analysis. Systolic and diastolic blood pressures were recorded to the nearest 2 mmHg.

Venous blood samples were collected from all subjects after an overnight fast of at least 10–12 hours. Serum was separated by centrifugation and analyzed for biochemical parameters. The lipid profile included total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C). These parameters were estimated using standard enzymatic colorimetric methods on an automated analyzer, following manufacturer protocols. Serum uric acid and creatinine levels were measured using the uricase–peroxidase and Jaffe's enzymatic colorimetric methods, respectively. All laboratory analyses were performed in the same biochemistry laboratory using quality-controlled reagents to ensure accuracy and reproducibility.

Statistical Analysis

All data were entered into Microsoft Excel and analyzed using the Statistical Package for the Social Sciences (SPSS) software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). The comparison of mean values between the two groups was performed using the unpaired Student's t-test. Categorical variables were expressed as frequencies and percentages and analyzed using the chi-square test wherever applicable. A *p-value* of less than 0.05 was considered statistically significant for all comparisons.

Prior to statistical analysis, the normality of data distribution was verified using the Kolmogorov–Smirnov test. Appropriate statistical tests were applied accordingly to maintain the validity of results. The findings were presented in tabular and graphical formats to facilitate clear interpretation and comparison between diabetic and non-diabetic groups.

RESULTS

A total of 200 participants were included in the study, comprising 100 healthy controls (Group I) and 100 patients with T2DM (Group II). The mean age and sex distribution between the two groups were comparable, eliminating demographic bias. The comparison of anthropometric, hemodynamic, and biochemical parameters between healthy and diabetic subjects is summarized in Table 1.

Table 1: Comparison of BMI, Blood Pressure, Lipid Profile, Uric Acid, and Creatinine between Healthy Controls and Type 2 Diabetic Patients.

Parameter	Healthy Controls (Mean ± SD)	T2DM Patients (Mean ± SD)	*p*-Value
BMI (kg/m²)	23.1 ± 2.5	27.8 ± 3.2	<0.001
Systolic BP (mmHg)	118 ± 10	136 ± 12	<0.001
Diastolic BP (mmHg)	76 ± 8	88 ± 10	<0.001
Total Cholesterol (mg/dL)	168 ± 22	210 ± 30	<0.001
Triglycerides (mg/dL)	118 ± 28	185 ± 36	<0.001
HDL-C (mg/dL)	51 ± 7	38 ± 6	<0.001
LDL-C (mg/dL)	94 ± 20	132 ± 25	<0.001
VLDL-C (mg/dL)	23 ± 6	37 ± 7	<0.001
Uric Acid (mg/dL)	4.6 ± 0.9	6.1 ± 1.1	<0.001
Creatinine (mg/dL)	0.8 ± 0.2	1.1 ± 0.3	<0.001

All values are expressed as Mean ± Standard Deviation (SD); $p < 0.05$ considered statistically significant.

Anthropometric and Hemodynamic Findings

The mean BMI among Type 2 diabetic patients (27.8 ± 3.2 kg/m²) was significantly higher than that of healthy controls (23.1 ± 2.5 kg/m²), indicating a strong association between obesity and diabetes ($p < 0.001$). Both systolic and diastolic blood pressures were markedly elevated in the diabetic group (136 ± 12 mmHg and 88 ± 10 mmHg, respectively) compared to controls (118 ± 10 mmHg and 76 ± 8 mmHg), suggesting coexistence of hypertension in a large proportion of diabetic individuals.

Lipid Profile Findings

The lipid profile revealed significant dyslipidemia in diabetic patients. The mean total cholesterol, triglycerides, LDL-C, and VLDL-C levels were substantially higher in diabetics (210 ± 30 mg/dL, 185 ± 36 mg/dL, 132 ± 25 mg/dL, and 37 ± 7 mg/dL, respectively) than in healthy individuals (168 ± 22 mg/dL, 118 ± 28 mg/dL, 94 ± 20 mg/dL, and 23 ± 6 mg/dL). Conversely, HDL-C levels were significantly lower in diabetics (38 ± 6 mg/dL) compared to controls (51 ± 7 mg/dL). All these differences were statistically significant ($p < 0.001$).

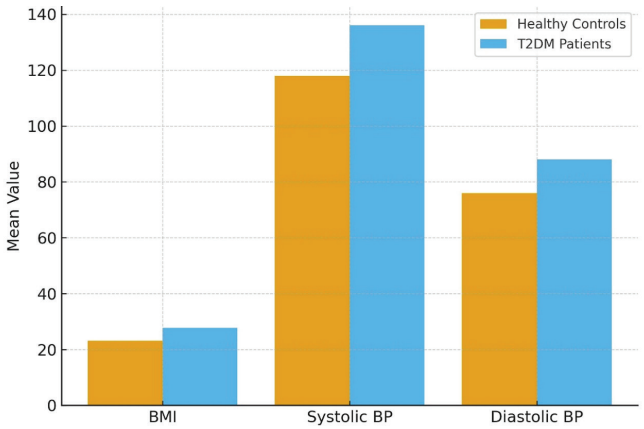

Figure 1: Comparison of BMI and Blood Pressure between Healthy and Diabetic Groups.

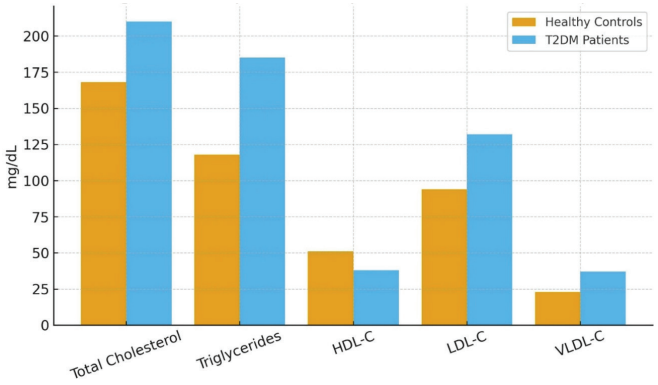

Figure 2: Comparison of Lipid Profile Components.

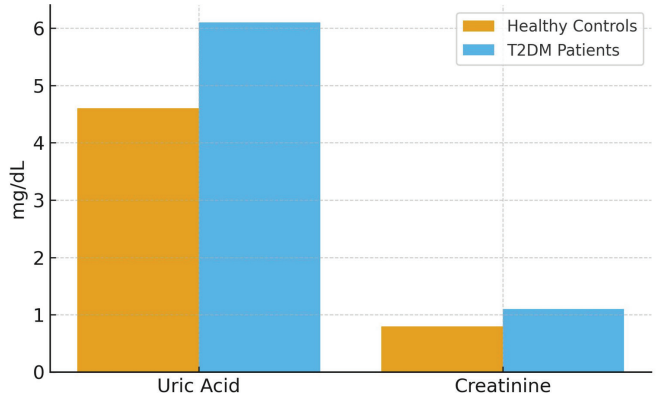

Figure 3: Comparison of Renal Parameters.

This pattern — elevated triglycerides, LDL-C, and VLDL-C with reduced HDL-C — represents the classical "diabetic dyslipidemia" that predisposes to premature atherosclerosis and cardiovascular events.

Renal Biochemical Parameters

Serum uric acid and creatinine levels were both significantly elevated in the diabetic group (6.1 ± 1.1 mg/dL and 1.1 ± 0.3 mg/dL, respectively) compared to controls (4.6 ± 0.9 mg/dL and 0.8 ± 0.2 mg/dL). These findings reflect early renal involvement and alter purine metabolism commonly associated with insulin resistance and oxidative stress in diabetes.

The study demonstrated statistically significant differences in all evaluated parameters between healthy controls and diabetic patients. The findings clearly indicate that individuals with Type 2 Diabetes Mellitus exhibit higher BMI, elevated blood pressure, dyslipidemia (high TC, TG, LDL-C, VLDL-C, and low HDL-C), and increased renal biomarkers (uric acid and creatinine). These alterations collectively underscore the multifactorial metabolic disturbances inherent to diabetes, emphasizing the need for comprehensive metabolic assessment and early intervention.

DISCUSSION

The present study demonstrated a clear and consistent metabolic distinction between healthy individuals and patients with T2DM. The findings reaffirm the multidimensional nature of diabetes as not merely a disorder of carbohydrate metabolism but a complex interplay involving obesity, dyslipidemia, hypertension, and renal dysfunction — together constituting a constellation of metabolic abnormalities that substantially elevate cardiovascular and renal risk.[7,8]

The significantly higher BMI among diabetic participants underscores the pivotal role of obesity in the pathogenesis of T2DM.[9,10] Adipose tissue, particularly visceral fat, acts as an active endocrine organ that secretes various adipokines, including leptin, adiponectin, resistin, and inflammatory cytokines such as TNF-α and IL-6.[11,12] These bioactive molecules contribute to the development of insulin resistance by impairing insulin receptor signaling pathways, thereby promoting chronic hyperglycemia.[13] In addition, obesity-induced low-grade inflammation and altered lipid metabolism further perpetuate β-cell dysfunction, completing a vicious metabolic cycle that sustains diabetes progression.[14]

A characteristic feature of T2DM observed in this study was diabetic dyslipidemia, marked by elevated triglycerides, total cholesterol, LDL-C, VLDL-C, and reduced HDL-C.[15,16] This lipid triad — high TG, high LDL-C, and low HDL-C — is widely recognized as a hallmark of insulin-resistant states.[17] Mechanistically, insulin resistance leads to unrestrained lipolysis in adipose tissue, resulting in excess free fatty acid flux to the liver.[18] The hepatic overproduction of VLDL and reduced lipoprotein lipase activity culminate in hypertriglyceridemia and formation of small dense LDL particles.[19] These small dense LDL particles are more prone to oxidation and have a greater atherogenic potential.[20] The low HDL-C levels further impair reverse cholesterol transport, exacerbating endothelial injury and atherogenesis.[21]

The observed elevation in blood pressure among diabetic subjects corroborates the well-established coexistence of hypertension and T2DM.[22] Multiple mechanisms contribute to this phenomenon, including hyperinsulinemia-induced sodium retention, increased sympathetic activity, endothelial dysfunction, and arterial stiffness due to non-enzymatic glycation.[23] Reduced nitric oxide (NO) bioavailability in diabetes further increases peripheral vascular resistance.[24] The convergence of hyperglycemia and hypertension markedly accelerates target organ damage, affecting the heart, kidneys, and retina.[25]

In the present study, serum uric acid levels were significantly higher among diabetics compared to healthy controls. Elevated uric acid reflects oxidative stress, increased purine metabolism, and reduced renal clearance. Hyperuricemia contributes to insulin resistance and endothelial dysfunction by decreasing nitric oxide synthesis.[26]

Similarly, elevated serum creatinine levels among T2DM patients in this study suggest early renal involvement, possibly due to glomerular hyperfiltration and microvascular injury. Chronic hyperglycemia induces non-enzymatic glycation of basement membrane proteins, leading to structural and functional changes in the glomerulus.[25] The results of this study are in close agreement with earlier findings by Choudhary et al. and Sharma et al., who reported similar patterns of dyslipidemia, hypertension, and renal dysfunction among T2DM patients.[15,16]

Collectively, the present results highlight the interconnected pathophysiological mechanisms underlying the metabolic disturbances in T2DM. Chronic hyperglycemia, insulin resistance, and oxidative stress form the central axis around which these abnormalities revolve, ultimately leading to vascular inflammation, endothelial dysfunction, lipid peroxidation, and renal injury.[24,26]

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