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Evaluation of Metabolic Parameters in Diabetic Patients With and Without Nephropathy

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Abstract:

Background: Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from impaired insulin secretion, insulin action, or both. The interplay between glucose metabolism and lipid homeostasis plays a critical role in the onset and progression of diabetic complications, particularly diabetic nephropathy. **Aim:** This study aimed to compare the glucose and lipid profiles in diabetic patients with nephropathy to those without nephropathy. **Materials and Methods:** A cross-sectional pilot study was conducted at a tertiary care hospital, involving 200 adult patients diagnosed with Type 2 Diabetes Mellitus (T2DM) for at least five years. Participants, aged between 30 and 70 years, were divided into two groups: those with diabetic nephropathy and those without. Informed consent was obtained from all participants. Blood samples were collected and analyzed using standardized biochemical methods, including the DPEC-GOD/POD technique, ClinRep full kit, LDN IRMA reagent, and CHOD/POD method. **Result:** Significant differences were observed between the two groups in terms of fasting blood sugar (FBS) ($t = 2.692$, $df = 198$, $p < 0.05$), HbA1c ($t = 5.279$, $df = 198$, $p < 0.05$), serum insulin ($t = 6.123$, $df = 198$, $p < 0.05$), and HOMA-IR ($t = 37.767$, $df = 198$, $p < 0.05$). However, no significant differences were found in age ($t = 0.779$, $df = 198$, $p > 0.05$) or diastolic blood pressure ($t = 1.063$, $df = 198$, $p > 0.05$). Regarding lipid profile, total cholesterol (TC) ($t = 3.665$, $df = 198$, $p < 0.05$), low-density lipoprotein (LDL) ($t = 3.079$, $df = 198$, $p < 0.05$), and high-density lipoprotein (HDL) ($t = 9.225$, $df = 198$, $p < 0.05$) showed significant differences. Triglycerides (TAG) ($t = 0.216$, $df = 198$, $p > 0.05$) and very low-density lipoprotein (VLDL) ($t = 0.501$, $df =$

198, $p > 0.05$) did not differ significantly between the groups. **Conclusion:** The findings emphasize the critical role of altered glucose and lipid metabolism in the development of diabetic nephropathy. Monitoring these parameters may aid in early detection and better clinical management of affected individuals. Nevertheless, larger longitudinal studies and research into genetic susceptibility are essential for developing more effective therapeutic strategies.

Key words: Diabetes mellitus, cholesterol, triacylglycerols, fasting blood sugar, end stage renal disease, low density lipoprotein, diabetic nephropathy.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by hyperglycemia resulting from impaired insulin secretion, insulin action, or both. It affects millions globally and is a major cause of morbidity and mortality [1,2]. One of the most serious complications of diabetes is diabetic nephropathy (DN), a progressive kidney disease and the leading cause of end-stage renal disease (ESRD) [3–5].

The interplay between glucose metabolism and lipid homeostasis plays a crucial role in the onset and progression of diabetic complications, including nephropathy. Persistent hyperglycemia leads to oxidative stress, inflammation, and endothelial dysfunction, which collectively damage renal structures [6]. Concurrently, dyslipidemia—marked by elevated triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C)—exacerbates these effects [7–15,18–20].

In patients with diabetic nephropathy, lipid abnormalities tend to be more severe compared to those without nephropathy. Elevated very low-density lipoprotein cholesterol (VLDL-C) and small dense LDL particles are associated with glomerular injury, proteinuria, and inflammation, while high TG and low HDL-C levels promote lipid accumulation in renal cells, oxidative stress, and podocyte dysfunction [18–20].

Assessing differences in glucose and lipid profiles between diabetic patients with and without nephropathy is essential for early detection and prevention. Markers such as fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) reflect glycemic control and are closely linked to microvascular complications, including DN [9–17]. Chronic hyperglycemia also contributes to the formation of advanced glycation end products (AGEs), which drive structural changes in the kidney, such as glomerular basement membrane thickening and mesangial expansion [11–13]. This study aims to compare glucose and lipid profiles in diabetic patients with and without nephropathy to uncover metabolic patterns associated with DN. The findings could inform early interventions to optimize glucose and lipid control, potentially delaying or preventing the progression of diabetic nephropathy.

Materials & methods:

A cross-sectional study was conducted at a tertiary care hospital after getting Ethics clearance from the Institutional Ethics Committee. Patients were recruited from outpatient and inpatient

departments of endocrinology and nephrology. 200 diabetic patients were enrolled, equally divided into two groups: Group 1: 100 patients with diabetes and nephropathy (defined by albuminuria and/or reduced eGFR). Group 2: 100 patients with diabetes without nephropathy (normal renal function and absence of albuminuria).

Inclusion Criteria: Adults aged 30–70 years. Diagnosed with Type 2 Diabetes Mellitus for at least five years. Availability of informed consent.

Exclusion Criteria: Pregnant or lactating women, patients with chronic illnesses other than nephropathy, and use of vitamin supplements within three months prior to the study.

Using a disposable syringe and cannula in a sterile environment, 5ml of each individual's fasting venous blood was extracted into flat containers in both groups. After being separated from blood by centrifugation at 3000 rpm for 20 minutes, serum samples were aliquoted and stored at 20 ° C. Avantor laboratories' DPEC – GOD/POD technique measured plasma glucose. The manual's instructions generated the reagents. The ClinRep full kit was used on the BioRad Diamant and Variant to measure HbA1C. 4.5-6.1% is normal. Serum insulin levels were measured with an LDN IRMA reagent. Supplier instructions were followed. With sensitivity of 0.5 IU/mL, inter- and intra-assay CVs were 4.3% and 3.4%, respectively. According to Muniyappa et al. (2008), HOMA-IR was calculated. In the case of lipid profile; to measure serum TC, CHOD/POD procedure was used. Glycerol Phosphate Oxidase and Peroxidase (Liquid stable) assessed serum TAGs. All reagents were purchased from Avantor Performance Materials India Limited, Dehradun, Uttarakhand, India, and the estimation followed the kit manual. Supplier instructions were followed.

Statistical analysis:

The study used Microsoft Excel to analyze data, representing categorical variables as frequencies and percentages, and continuous variables as mean ± SD. The t test was used to compare diabetic patients with and without nephropathy. Statistical significance was with a p-value of less than 0.05.

Results:

Table 1: Glucose profile in the study populations.

Variable	**Diabetes with nephropathy (n=100)**	**Diabetes without nephropathy (n=100)**	**P Value**
Fasting Blood Sugar (FBS) (mg/dL)	133.9 ± 56	117.7 ± 22	=0.0077 T=2.692 Df = 198
	196.8±73.3	169.8±22.6	=0.3691

Post prandial blood sugar (PPBS) (mg/dL)			T= 0.900 Df = 198
Glycosylated hemoglobin (HbA1C) (gm%)	8.1 ± 2.6	6.4 ± 1.9	=0.001 T= 5.279 Df= 198
Insulin (µU/mL)	22.2 ± 5.9	15.3 ± 9.6	=0.0001 T= 6.123 Df = 198
Homeostasis metabolic assessment- insulin resistance (HOMA-IR)	24.9 ± 2.3	6.8 ± 3.9	= 0.0001 T = 37.767 Df = 198

In the present study (Table 1), glucose profile details of the present study participants are given. We observed significant differences when compared between the two groups with regards to FBS (t=2.692, df=198, P <0.05), HbA1c (t=5.279, df=198, P <0.05), serum insulin (t=6.123, df=198, P <0.05), and HOMA-IR (t=37.767, df=198, P <0.05). On the other hand, we did not observe any significant difference when compared between the two groups with regards to age (t=0.779, df=198, P >0.05) and diastolic BP (t=1.063, df=198, P >0.05).

Table 2: Laboratory details of lipid profile in the study population.

Variable	**Diabetes with nephropathy (n=100)**	**Diabetes without nephropathy (n=100)**	**P Value**
Total Cholesterol (TC) (mg/dL)	175.8 ± 39.2	158.6 ±25.8	= 0.003 T = 3.665 Df = 198
Triacylglycerols (TAG) (mg/dl)	178.5 ± 54.3	177 ± 43.2	= 0.829 T = 0.216 Df = 198
Low density lipoprotein (mg/dL)	162.2± 64.7	139.9 ± 32.5	= 0.0024 T = 3.079 Df = 198
High Density Lipoproteins (HDL) (mg/dl)	23.2 ± 9.8	35.6 ± 9.2	= 0.0001 T = 9.225 Df = 198
Very Low Density Lipoproteins (VLDL) (mg/dl)	35.7 ± 10.9	35.4 ± 8.7	= 0.616 T = 0.501 Df = 198

In the present study (Table 2), lipid profile details of the present study participants are given. We observed significant differences when compared between the two groups with regards to TC (t=3.665, df=198, P <0.05), LDL (t=3.079, df=198, P <0.05), and HDL (t=9.225, df=198, P <0.05).

On the other hand, we did not observe any significant difference when compared between the two groups with regards to TAG (t=0.216, df=198, P >0.05) and VLDL (t=0.501, df=198, P >0.05).

Discussion

Diabetes mellitus (DM) is a multifaceted metabolic disorder marked by chronic hyperglycemia and is closely associated with a spectrum of microvascular and macrovascular complications, including diabetic nephropathy. Evaluating key biomarkers—such as fasting blood glucose (FBS), glycated hemoglobin (HbA1c), and the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR)—provides critical insights into the metabolic disturbances underlying diabetes, particularly in the context of nephropathy.

FBS serves as a fundamental parameter for diagnosing and monitoring diabetes. In patients without nephropathy, elevated FBS levels typically result from insulin resistance and impaired glucose uptake. However, in those with diabetic nephropathy, glucose dysregulation is often more pronounced due to diminished renal gluconeogenesis and altered glucose clearance. Evidence suggests a positive correlation between FBS levels and the severity of nephropathy, highlighting the kidney's essential role in glucose homeostasis [9,10].

HbA1c reflects average blood glucose over the preceding 2–3 months, making it a robust indicator of long-term glycemic control. Studies report elevated HbA1c levels in patients with diabetic nephropathy, attributed to sustained hyperglycemia and heightened oxidative stress [11]. Moreover, advanced glycation end-products (AGEs), which are strongly associated with elevated HbA1c, contribute to renal structural damage. It is important to note that HbA1c interpretation may be affected in nephropathy due to anemia and altered erythrocyte lifespan—common in advanced renal disease—which can skew results [12].

HOMA-IR, calculated from fasting glucose and insulin concentrations, offers a measure of insulin resistance—an underlying driver of both diabetes progression and nephropathy. Research indicates significantly elevated HOMA-IR values in individuals with diabetic nephropathy, reflecting the central role of systemic insulin resistance in renal impairment [13,14]. Increased HOMA-IR is also linked with inflammation, oxidative stress, and dyslipidemia, all of which contribute to worsening kidney damage.

The combined assessment of FBS, HbA1c, and HOMA-IR underscores the complexity of diabetic pathophysiology and its complications. Early and routine evaluation of these biomarkers is vital for identifying patients at elevated risk for nephropathy. Interventions that improve glycemic control and enhance insulin sensitivity may play a key role in preventing or slowing nephropathy progression.

Recent research has also highlighted the potential of novel therapies, such as sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs), in improving glycemic outcomes while providing renal protective effects [15–17]. Continued studies are necessary to clarify the molecular mechanisms linking these metabolic markers to diabetic nephropathy and to refine targeted treatment strategies.

Dyslipidemia remains a defining feature of diabetes, characterized by elevated triglycerides, reduced high-density lipoprotein (HDL) cholesterol, and increased low-density lipoprotein (LDL) cholesterol. In diabetic nephropathy, these lipid disturbances are often more severe and include an increase in small dense LDL particles and lipoprotein(a), both of which are implicated in atherogenesis and glomerular injury. Studies [18–20] have shown that such lipid abnormalities further accelerate renal function decline and heighten cardiovascular risk.

Additionally, patients with diabetic nephropathy tend to exhibit significantly lower serum Vitamin D levels compared to those without nephropathy, likely due to impaired renal hydroxylation of Vitamin D. Deficiencies in Vitamin D, Vitamin B12, and folic acid are more prevalent and severe in this population due to altered renal metabolism and clearance. These deficiencies, in conjunction with worsening dyslipidemia, contribute to the rapid progression of both cardiovascular and renal complications. Proactive management—including routine screening and timely supplementation—may help improve metabolic and renal outcomes. However, further longitudinal and interventional studies are needed to elucidate the precise roles of these parameters in the progression of diabetes-related complications.

Conclusion:

We conclude that the interaction between glucose and lipid metabolism is essential to the onset and advancement of diabetic nephropathy. Looking at these parameters gives us important information about how this condition starts, which makes it easier to find it early and improves management plans for groups that are at risk. However, it has several drawbacks, including the need for longitudinal studies and further investigation into genetic predisposition in status determination. Future research with varied populations is needed to address these gaps and develop more effective population-specific therapies.

Conflict of interest:

The present study authors do not possess any conflict of interest among themselves.

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