

Correlation between Dyslipidemia and Urine Albumin Excretion in Non Diabetic Obese Subjects: A Cross-sectional Study

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ABSTRACT

Introduction: Obesity is a global health concern, and recent research has shown that it can cause kidney problems, especially in non diabetics. Obesity-related dyslipidemia, characterised by aberrant lipid profiles, may contribute to kidney damage.

Aim: The purpose of the present cross-sectional study was to shed light on the complex interaction between dyslipidemia and renal health by examining the relationship between dyslipidemia and urine albumin excretion in non diabetic obese adults.

Materials and Methods: The cross-sectional investigation was conducted in the Outpatient Obesity Clinic of the Endocrinology Department at a Tertiary Hospital in Kerala, India. A total of 144 non diabetic obese individuals between the ages of 18 and 65 years. All subjects provided informed consent, and ethical approval was obtained. The primary inclusion criteria were a Body Mass Index (BMI) of less than 25.0, the absence of Type 2 Diabetes Mellitus (T2DM), and the absence of proteinuria. Urine and blood samples were collected for laboratory testing, and statistical analysis was conducted using International Business Machines (IBM) Statistical Package for Social Sciences (SPSS) Statistics 20.0.

Results: Dyslipidemia was found to be common, with low High-Density Lipoproteins (HDL) levels in 41.4% of participants, high Low-Density Lipoproteins (LDL) levels in 49.6%, high Triglycerides (TG) levels in 29.7%, and high Total Cholesterol (TC) levels in 43%. The study revealed that obese individuals with dyslipidemia excrete albumin at a significantly higher rate than obese individuals without dyslipidemia. Furthermore, a weak correlation between TG levels and the Urine Albumin Creatinine Ratio (UACR) was discovered. However, UACR did not significantly correlate with metabolic risk markers such as Fasting Plasma Glucose (FPG), serum insulin, blood pressure, HDL, LDL, TC, and systolic/Diastolic Blood Pressure (DBP).

Conclusion: The results of the present study demonstrate that dyslipidemia significantly contributes to kidney damage in non diabetic obese individuals. The presence of dyslipidemia in obese then could serve as an early warning sign of potential renal problems. Further research is needed to fully understand the complex interactions between obesity, dyslipidemia, and renal health. These insights could be crucial for developing effective preventative and intervention methods in this context.

Keywords: Albuminuria, Cholesterol, Metabolic risk factors, Obesity-related nephropathy, Renal dysfunction

INTRODUCTION

Chronic Kidney Disease (CKD) is a growing concern, with emerging evidence suggesting that even modest weight gain, falling within the normal range of BMI, can elevate CKD risk [1]. Obesity is linked to several mechanisms that aggravate renal problems and make people more vulnerable to CKD. These causes include inflammation brought on by obesity, endothelial dysfunction, altered renal haemodynamics, hyperlipidaemia, oxidative stress, podocyte stress, and an imbalance in adipokines. Even in the absence of hypertension or hyperglycaemia, endothelial dysfunction- a crucial early event in obesity- is still present, highlighting the significance of obesity as a risk factor for endothelial dysfunction [2]. In obesity, adipose tissue secretes adipokines and inflammatory cytokines, accompanied by elevated levels of Free Fatty Acids (FFA), increased LDL, reduced HDL, activation of the Renin-Angiotensin System (RAS), and Sympathetic Nervous System (SNS) activation, all of which contribute to endothelial dysfunction through direct or indirect effects on vascular endothelium-derived Nitric Oxide (NO) [3,4].

The substantial link between obesity and microvascular dysfunction in numerous tissues is further shown by the relationship between weight loss and better endothelial function [5]. One specific complication of obesity is known as Obesity-related Glomerulopathy (ORG) [6]. Renal lipotoxicity is caused by lipid dysmetabolism and abnormalities in lipid profiles, which also promote glomerulosclerosis and directly affect ORG [7]. Renal hyperfiltration, alterations in renal haemodynamics brought on by obesity, and slow-onset proteinuria

with microalbuminuria as the initial clinical manifestation are the hallmarks of ORG [8].

Maintaining insulin sensitivity, promoting fatty acid oxidation, lowering plasma Triglycerides (TG), and enhancing glucose metabolism are all made possible by the adipocyte hormone adiponectin, which is also implicated in lipid and glucose metabolism. Endothelial dysfunction and associated co-morbidities are caused by decreased adiponectin levels in obesity [9].

In obese people, lower adiponectin levels are linked to higher TG and fatty liver [10]. Obesity-induced dyslipidemia may independently cause renal damage through the increase of Sterol-regulatory Element-binding Proteins (SREBP-1 and 2) in glomerular cells, resulting in lipotoxicity [11]. Increased FFA intake also contributes to tubulointerstitial inflammation, fibrosis, and glomerular podocyte damage in proximal tubular epithelial cells due to glomerular lipid accumulation and the production of foam cells [12,13]. Studies have shown that glomerular lipid deposition and foam cell production in dyslipidemia are related to the onset of Focal Segmental Glomerulosclerosis (FSGS) [14]. Additionally, it has been demonstrated that lipid-lowering medications slow the onset of ORG and proteinuria [13]. Obesity-related development of CKD is a complex interplay of numerous factors affecting renal pathophysiology rather than a single isolated event. Obesity is a "multi-hit" condition that starts and/or hastens kidney damage [15].

Therefore, the goal of the present study was to shed light on the complex pathways relating obesity to renal problems by examining the relationship between dyslipidemia and urine albumin excretion in

non diabetic obese individuals. Understanding these mechanisms is essential for successful preventive and therapeutic measures for CKD in the obese population.

MATERIALS AND METHODS

The cross-sectional investigation was conducted in the Outpatient Obesity Clinic of the Endocrinology Department at a tertiary hospital in Kerala, India. All participants provided informed consent, and ethical clearance was granted by the Ethics Committee [IEC number:- Order No. B6-155/2019/MCTCR(28)].

Sample size calculation: The minimum sample size required, considering the variables of UACR and BMI, was calculated to be 140 based on a 95% confidence level and 80% power.

Inclusion criteria: Participants with a BMI of less than 25.0 were selected from both sexes within the age range of 18 to 65 years. The following criteria were used for inclusion:

- No presence of red blood cells, pus cells, or proteinuria detected during routine urine dipstick screening for Type 2 Diabetes Mellitus (T2DM).
- No recent history of vigorous exercise within 24 hours of the test.
- HbA1c (haemoglobin A1c) less than 6.5%.
- Serum creatinine level of 1.4 mg% or less.

Exclusion criteria: The following groups of subjects were not included in the study:

- Age less than 18 or greater than 65 years.
- HbA1c \geq 6.5%.
- Clinical suspicion of urinary tract infection.
- History of hypertension, chronic liver disease, CKD, or known cardiovascular disease.
- Presence of fever or any other recent or ongoing illnesses.
- Taking medications for diabetes, obesity, or high blood pressure.
- Taking corticosteroids, Non Steroidal Anti-inflammatory Drug (NSAIDs), or other nephrotoxic medications.
- Pregnancy or use of contraceptives.
- Participants menstruating at the time of the study.
- Extended upright posture prior to testing.

Study Procedure

- Spot urine samples were collected in sterile containers for the purpose of determining the UACR, and the ratio of albumin to creatinine was calculated.
- Venous blood samples were aseptically taken while the subject was seated. FPG samples were obtained using fluoride-containing vacutainers. Plasma was then transferred to labelled vials after centrifugation at 3000 g for 15 minutes.
- Vacutainers with Ethylenediaminetetraacetic Acid (EDTA) were used to collect samples for HbA1c testing.
- Blood was drawn into vacutainers devoid of anticoagulant to obtain samples for additional biochemical markers.
- The Beckman Coulter Olympus AU2700 was used to measure serum creatinine and FPG, while the Bio-Rad D-10 was used to measure HbA1c.
- The UACR was expressed as mg/g.

UACR calculation:

- Urine albumin (mg/dL)=UACR in mg/g/Urine creatinine (g/dL) was used to calculate UACR.
- The Beckman Coulter Olympus AU2700 was used to calculate the value.

Urine sample considerations: Urine samples tainted with blood were declared unacceptable for UACR computation, as blood

might artificially raise albumin levels. Patients were instructed to avoid strenuous exercise 24 hours prior to the test.

Laboratory analysis:

- Urine albumin was estimated using an immunoturbidimetric technique.
- Urine creatinine was estimated using Jaffe's kinetic technique.

STATISTICAL ANALYSIS

For statistical analysis in this study, IBM SPSS Statistics 20.0 for Windows was used. Continuous variables were presented as mean \pm standard deviation. The Mann-Whitney U test was used to compare parameters with non normal distribution, while the Pearson's correlation coefficient was calculated for variables with normal distribution. Multiple regression analysis was used to find determinants of UACR. Log transformation was used for UACR due to its non normal distribution and significant unpredictability. The study maintained an 80% statistical power, and p-values less than 0.05 were considered significant. These techniques allowed for a thorough examination of the data and informed judgements.

RESULTS

The initial characteristics of the study group are shown in [Table/Fig-1]. The average BMI of the individuals was 36.78 kg/m², with an average age of 31.94 years. The average waist circumference (112.61 cm), Systolic Blood Pressure (SBP) (125.35 mmHg), and DBP (78.15 mmHg) were recorded. HbA1c averaged 5.64%, and the FPG level was 98.80 mg/dL. The mean lipid profile values were as follows: TC 195.92 mg/dL, LDL 129.68 mg/dL, HDL 44.38 mg/dL, and TG 134.03 mg/dL. The serum creatinine level was 0.8982 mg/dL, and the UACR had a mean value of 8.49. Pearson's correlations between several variables and UACR are shown in [Table/Fig-2]. It shows a positive association between BMI and UACR ($r=0.23$, $p=0.006$), indicating that a higher BMI is linked to a higher UACR. There was also a strong positive connection between TG and UACR ($r=0.258$, $p=0.003$). A larger waist circumference was associated with a greater UACR, as indicated by a strong positive correlation between waist circumference and UACR ($r=0.302$, $p<0.001$). Other factors such as FPG, HDL, LDL, TC, HbA1c, SBP, and DBP showed weaker or no significant correlations with UACR.

| Characteristics | n | Mean | SD |
|--------------------------------|-----|--------|--------|
| Age (years) | 144 | 31.94 | 11.9 |
| BMI (kg/m ²) | 144 | 36.78 | 5.15 |
| WC (cm) | 144 | 112.61 | 10.89 |
| SBP (mmHg) | 144 | 125.35 | 12.35 |
| DBP (mmHg) | 144 | 78.15 | 6.64 |
| FPG (mg/dL) | 131 | 98.80 | 14.75 |
| HbA1c (%) | 144 | 5.64 | 0.4476 |
| Total Cholesterol (TC) (mg/dL) | 129 | 195.92 | 41.6 |
| LDL (mg/dL) | 129 | 129.68 | 31.99 |
| HDL (mg/dL) | 128 | 44.38 | 14.85 |
| TG (mmol/L) | 128 | 134.03 | 62.11 |
| UACR (mg/g) | 144 | 8.49 | 9.42 |
| Serum creatinine (mg/dL) | 143 | .8982 | 0.1187 |

[Table/Fig-1]: Baseline characteristics of study groups.

**BMI: Body mass index; WC: Waist circumference; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; FPG: Fasting plasma glucose; HbA1c: Haemoglobin A1c (Glycated Haemoglobin); TC: Total cholesterol; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TG: Triglycerides; UACR: Urine albumin creatinine ratio

| Factors | n | UACR | |
|--------------------------|-----|---------------------|---------|
| | | Pearson correlation | p-value |
| BMI (kg/m ²) | 144 | 0.23 | 0.006 |
| FPG (mg/dL) | 131 | 0.157 | 0.073 |

| | | | |
|-------------|-----|--------|--------|
| Insulin | 111 | 0.105* | 0.274 |
| HDL (mg/dL) | 128 | 0.014* | 0.871 |
| TG (mg/dL) | 128 | 0.258* | 0.003 |
| LDL (mg/dL) | 129 | 0.123 | 0.165 |
| TC | 129 | 0.106 | 0.231 |
| WC (cm) | 144 | 0.302 | <0.001 |
| HBA1c (%) | 144 | 0.159 | 0.056 |
| SBP (mmHg) | 144 | 0.067 | 0.426 |
| DBP (mmHg) | 144 | 0.090 | 0.286 |

[Table/Fig-2]: Correlation of UACR with key factors in the study population.

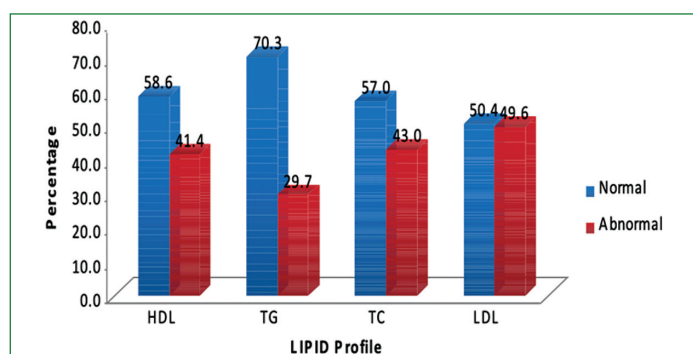
In [Table/Fig-3], the UACR between individuals with normal and disturbed lipid profiles was compared. The Mann-Whitney U test revealed that individuals with abnormal lipid profiles had significantly higher UACR (mean=9.70, SD=10.64) compared to those with normal lipid profiles (mean=5.97, SD=6.55) (p=0.011). In [Table/Fig-4], the Mann-Whitney U test indicated no statistically significant difference in UACR between people with normal TG levels (mean=8.08, SD=8.74) and those with aberrant TG levels (mean=9.92, SD=11.771) (p=0.388). [Table/Fig-5] presents a graph depicting the percentage distribution of lipid profile parameters in the research population. [Table/Fig-6] shows a scatterplot graph illustrating the association between UACR and TG levels.

| Groups | Classification | n | UACR | | p-value |
|--------------|----------------|----|------|-------|---------|
| | | | Mean | SD | |
| Dyslipidemia | Normal | 38 | 5.97 | 6.55 | 0.011 |
| | Abnormal | 90 | 9.70 | 10.64 | |

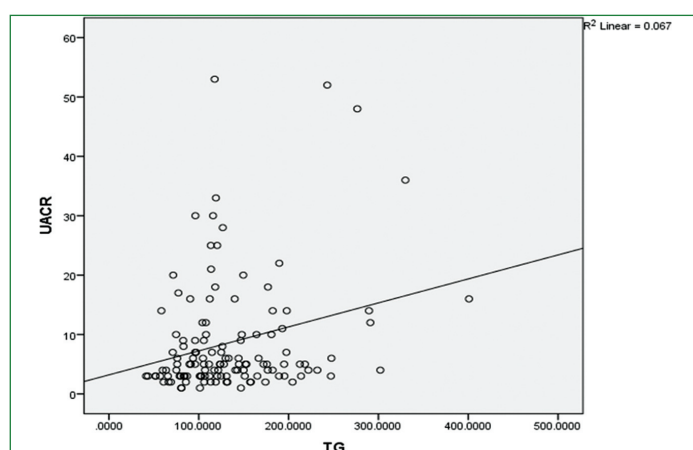
[Table/Fig-3]: Comparison of UACR in dyslipidemia (Mann Whitney U Test).

| Factor | Classification | n | UACR | | p-value |
|--------|----------------------|----|------|--------|---------|
| | | | Mean | SD | |
| TG | Normal <150 mgm/dL | 90 | 8.08 | 8.74 | 0.388 |
| | Abnormal >150 mgm/dL | 38 | 9.92 | 11.771 | |

[Table/Fig-4]: Comparison of UACR with TG (Mann Whitney U Test).



[Table/Fig-5]: Graph showing the percentage distribution of lipid profile.



[Table/Fig-6]: Scatter diagram showing the correlation between TG and UACR.

Therefore, the findings suggest that higher UACR is associated with higher BMI, waist circumference, and dyslipidemia, particularly elevated TG levels. These results highlight the importance of controlling obesity and dyslipidemia in non diabetic obese individuals to reduce the risk of renal impairment. However, further research is needed to examine the underlying causes and potential therapies.

DISCUSSION

The present cross-sectional study examined 144 non diabetic obese individuals between the ages of 18 and 65 years who were enrolled in an obesity clinic at a tertiary hospital in Kerala. The aim was to better understand the complex association between UACR and dyslipidemia and determine whether dyslipidemia significantly affects kidney health. The results confirm the growing concern about the harmful effects of obesity on renal health, which is supported by previous research [1,16,17]. Obesity has been associated with an increased risk of CKD globally. Understanding the complex relationship between obesity and renal problems, including dyslipidemia, is crucial [1]. Dyslipidemia, characterised by abnormal lipid profiles, has been linked to kidney injury, particularly in the context of obesity [6,7]. Previous studies have demonstrated the relationship between obesity and hepatic triglyceride synthesis, leading to adverse changes in lipid profiles and hyperlipidemia [18]. These findings are consistent with the present investigation, which showed a significant proportion of participants with high TC, high TG, high LDL, low HDL, and high triglyceride levels [Table/Fig-1].

Furthermore, the present study reveals that obese individuals with dyslipidemia had significantly higher albumin excretion compared to those without dyslipidemia [Table/Fig-3]. However, it is noteworthy that the mean albumin excretion remained within the reference range for normalcy. Among the lipid profile characteristics examined, the TG profile showed a notable but weak association with UACR [Table/Fig-2]. Interestingly, these findings align with a study suggesting that visceral fat, plasma insulin, triglyceride, and low HDL cholesterol concentrations in obese individuals may not be closely correlated with urine albumin excretion, indicating a later onset of abnormal albuminuria during insulin resistance syndrome [19]. These results are consistent with other research highlighting the role of lipids in the development of kidney disease, suggesting that dyslipidemia contributes to renal damage, including glomerulosclerosis [6,7]. Dyslipidemia-induced glomerular lipid deposition and foam cell production have been linked to FSGS [7]. Moreover, there is clinical evidence indicating that controlling dyslipidemia can reduce kidney risks. For instance, LDL-apheresis has been shown to improve steroid-resistant FSGS by reducing proteinuria and increasing serum albumin levels [20]. This underscores the therapeutic potential of addressing lipid abnormalities in obese individuals to preserve renal function. The present study supports previous research by demonstrating that dyslipidemia is a contributing factor to renal damage and that dyslipidemic obese individuals have significantly higher albumin excretion compared to those without dyslipidemia [21]. However, it is important to note that the mean albumin excretion remained within the reference range for normality. Interestingly, among the evaluated lipid profile markers, only TG showed a significant albeit weak association with UACR [Table/Fig-2] [21].

These results align with a study suggesting that visceral fat, plasma insulin, triglyceride, and low HDL cholesterol concentrations in obese individuals may not be closely correlated with urine albumin excretion, indicating a later onset of abnormal albuminuria during insulin resistance syndrome [19]. Apart from Waist Circumference

(WC), Chandie Shaw PK et al., (2007) found that markers for metabolic syndrome, such as insulin resistance, C-reactive protein, lipids, and high blood pressure, could not independently predict higher urine albumin excretion [22]. This is consistent with the findings of the present study, which also showed no conclusive links between metabolic risk markers such as FPG, serum insulin, blood pressure, and UACR. [Table/Fig-2] only demonstrates a very weak correlation between TG and UACR. However, the value of our study lies in elucidating the nuanced interaction between dyslipidemia and UACR in non diabetic obesity, paving the way for further research and new therapeutic approaches. The study emphasises the need of comprehensive strategies to address dyslipidemia in the context of obesity and its potential impact on renal health. Therefore, this study contributes to the existing body of research that demonstrates a connection between obesity, dyslipidemia, and kidney health. In non diabetic obese individuals, dyslipidemia emerges as a significant contributor to kidney injury. Our findings underscore the importance of managing lipid abnormalities as a potential treatment avenue to protect renal function in this vulnerable population, although further study is needed to elucidate the exact mechanisms involved.

It is crucial to understand the broader implications of the present study findings. Obesity is a global epidemic with significant health consequences, including kidney problems [16]. The present study sheds light on the complex interactions between obesity, dyslipidemia, and renal health. The intricate nature of these connections is highlighted by the limited association between UACR and specific markers, particularly BMI and TG [Table/Fig-2]. Additionally, these findings underscore the importance of targeting dyslipidemia as a treatment approach to reduce renal risks in obese individuals [20]. Managing lipid abnormalities may help protect renal function, especially in those at a higher risk of ORG [6].

Limitation(s)

It is important to acknowledge some limitations of the study. Firstly, the cross-sectional design prevents drawing conclusions about cause and effect, and the relatively small sample size may limit the applicability of the present study findings. Secondly, the study's focus on non diabetic obese individuals means that the results may not necessarily apply to other demographics or those with diabetes. Furthermore, factors such as dietary practices, genetic predispositions, and lifestyle choices were not thoroughly examined.

CONCLUSION(S)

The present study clarifies the intricate connections between dyslipidemia and kidney health in non diabetic obese individuals. The present study findings demonstrate the significance of dyslipidemia as a major risk factor for renal damage in this susceptible population. Dyslipidemia, characterised by abnormal lipid profiles, has been linked to ORG and FSGS, two renal problems. The relationship between obesity and kidney injury supports the "multi-hit hypothesis" of CKD, which suggests that CKD development is not a single isolated event but rather the result of multiple factors influencing renal pathophysiology. While shedding light on these intricate relationships, the present study also underscores the need for further investigation to fully understand how non diabetic obesity impacts renal function. A comprehensive strategy is necessary to address the various health effects, including kidney health, of the worldwide obesity

epidemic. Recent advancements in obesity research are gradually uncovering the complex web of connections between obesity, dyslipidemia, and kidney health. Future studies should continue to explore these relationships to discover new therapeutic approaches for preserving renal function in obese individuals. By drawing this conclusion, the present study emphasises the significance of recognising dyslipidemia as a major contributor to kidney injury in non diabetic obesity.

These results highlight the importance of incorporating routine microalbuminuria testing into the clinical assessment and management of obesity. Identifying individuals at risk of renal problems is crucial. Therefore, conducting adequate research to study the complex relationships between obesity, dyslipidemia, and renal health is advised. By overcoming the challenges posed by the worldwide obesity epidemic, clinicians can develop effective preventive and intervention plans.

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