

Correlation between Serum Uric Acid and Microalbuminuria in Type 2 Diabetes Mellitus-An Observational Study at a Tertiary Care Hospital

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Conflict of interest: Nil

Abstract

Background: Diabetes mellitus (DM) represents a complex metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. Diabetic nephropathy, a prevalent complication, frequently progresses to end-stage renal disease (ESRD). Notably, a substantial proportion of individuals with type 1 diabetes mellitus (T1DM) and a notable subset of those with type 2 diabetes mellitus (T2DM), ranging from 5% to 15%, advance to ESRD. Elevated serum uric acid (SUA) levels, associated with hyperuricemia, have been implicated in the pathogenesis of diabetic nephropathy due to their potential to induce endothelial dysfunction, provoke inflammation, and promote oxidative stress. This study aims to elucidate the relationship between SUA levels and microalbuminuria in patients diagnosed with T2DM.

Objective: The primary objective is to investigate the correlation between serum uric acid levels and microalbuminuria in patients with type 2 diabetes mellitus, thereby assessing SUA's potential utility as a biomarker for renal dysfunction and microvascular complications.

Methodology: An observational, descriptive, cross-sectional, comparative single centric study was conducted at R.G. Kar Medical College & Hospital, involving patients admitted to the General Medicine department with T2DM. Serum uric acid concentrations and urinary albumin-to-creatinine ratios (ACR) were quantified. Statistical analyses were employed to evaluate the association between SUA levels and the presence of microalbuminuria.

Conclusion: This investigation underscores a significant association between elevated serum uric acid levels and microalbuminuria among individuals with T2DM. The findings suggest that SUA may serve as a promising biomarker for the early detection of renal dysfunction and microvascular complications in diabetic cohorts. Further research is warranted to elucidate the underlying pathophysiological mechanisms and validate SUA's prognostic value in predicting microalbuminuria. Such insights could potentially inform targeted therapeutic strategies and enhance clinical outcomes for patients with diabetes mellitus.

Keywords: T1DM- Type 1 Diabetes Mellitus, T2DM- Type 2 Diabetes Mellitus, UA – Uric Acid, CKD – Chronic Kidney Diseases, CVD – Cardiovascular Disease, ROS - Reactive Oxygen Species, GFR – Glomerulus Filtration Rate, TG – Triglyceride, GLUT2 - Glucose Transporter 2.

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Introduction

Diabetes mellitus (DM) is a chronic illness associated with high blood glucose levels, or hyperglycemia, occurring from deficiencies in insulin secretion, action, or both. In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus, followed by China (20.8 million) and the United States (17.7 million) in second and third place, respectively [1]. The projection of diabetes is more concerning,

indicating a significant rise of global prevalence to 10.2% by the year 2030 and 10.9% by 2045[2]. Diabetes leads to a wide spectrum of systemic complications, among which diabetic nephropathy, often leading to End Stage Renal Disease (ESRD) is a major health concern. The primary risk factors for the development of diabetes include changes in dietary habits, changing lifestyles, overweight, and ethnic disparities. However, extrinsic variables

such as dyslipidemia, risk factors for cardiovascular diseases, obesity, and metabolic syndrome- which are either linked with or unrelated to diabetes- play a major role in the pathogenesis of diabetic nephropathy in individuals with type 2 diabetes[3-4].

Microalbuminuria serves as the paramount indicator of kidney complications associated with diabetes, manifesting in its early stages and serving as a predictive marker for the progression of complications. Patients with diabetes should be screened annually for microalbuminuria and elevated blood creatinine levels, according to the American Diabetes Association (ADA) [5]. The first stage of diabetic kidney disease that can be clinically identified is microalbuminuria (typically ranging from 30 to 300 mg/day of urinary albumin secretion), which provides a window of opportunity for therapies that may be able to stop the disease's development into nephropathy characterized by microalbuminuria [6]. Remarkably, nearly 30% of patients diagnosed with type 2 diabetes may still have excessively high urine albumin levels [7]. Therefore finding people who have microalbuminuria is essential for identifying the risk of complications followed by diabetes.

The final product of human purine metabolism is uric acid (UA), of which about one-third is processed in the intestines and the other two-thirds excreted by the kidneys [8]. According to some epidemiological research, hyperuricemia may be linked to a decline in kidney function and may be a risk factor for renal decline in individuals with diabetes mellitus. Hyperuricemia is defined as serum UA levels that are greater than >6 mg/dl (360 μ mol/l) for women, >7 mg/dl (416 μ mol/l) for males, and, irrespective of gender, in the range of $\geq$ 6.5 mg/dl (387 μ mol/l) to >8.3 mg/dl (494 μ mol/l) [9].

The series of harmful consequences triggered by hyperuricemia include endothelial dysfunction and nitric oxide inhibition- mechanisms that are closely linked to the development of insulin resistance and as a result, the onset of diabetes mellitus[10-11].

This study aims to shed light on that relationship, a relationship that has significant therapeutic implications that have not yet been fully explained. Investigating the relationship between serum UA and microalbuminuria in patients with type 2 diabetes is crucial due to its clinical significance and possible effects on preventative measures and public health regulations.

In summary, this thesis endeavours to bridge the existing knowledge gap regarding the correlation between SUA and microalbuminuria in patients with T2DM, within the dynamic clinical milieu of Kolkata. By employing strict protocols and

thorough data analysis, our goal is to uncover new information about the biology of diabetic nephropathy and open the door to customised therapies that lessen the severity of renal problems in this susceptible group of patients. Patients with diabetes who are having their hyperuricemia investigated and managed early may not develop overt nephropathy.

Materials and Methodology

Study Design: This research employs an observational descriptive comparative cross-sectional single-centric study design. Through this design, we can gather data on demographic variables, clinical parameters, and relevant laboratory investigations to analyze the prevalence and associated factors of diabetic nephropathy in our patient cohort.

Study Setting: The study will be conducted within the Department of General Medicine and Nephrology at R.G. Kar Medical College & Hospital, situated in Kolkata, India.

Study Population: Our target population comprises patients admitted indoors in the General Medicine department during the designated study period. The eligibility of a subject concerning laboratory criteria were assessed according to the result.

Inclusion Criteria

1. Patients aged 16-65 years.
2. Either gender.
3. Diagnosed with type 2 diabetic nephropathy.
4. Presence of microalbuminuria (albumin to creatinine ratio (ACR) between 30-300 mg/dl).
5. Patients must provide informed consent and be willing to participate in the study.

Exclusion Criteria

1. Having experienced a myocardial infarction within the past three months.
2. Having impaired kidney function (serum creatinine level over 1.5 mg/dL or macroalbuminuria with an albumin to creatinine ratio exceeding 300 mg/dL).
3. Having severe valvular heart disease or heart failure.
4. Having hypothyroidism.
5. Having a malignancy.
6. Having alcoholism.
7. Having gout or other inflammatory diseases.
8. Having a urinary tract infection.
9. Pregnant or menstruating.
10. Having a history of fever or severe trauma within the last seven days.
11. Using corticosteroids or cytotoxic drugs
12. Do not provide consent for the study.

Sample Size Calculation:

We have calculated the sample size using the following formula:

$$N = (Z)^2 \times p \times (1-p) / L^2$$

Where,

N= Sample size required

Z= Standard normal deviation corresponding to the desired confidence level (95%)

p= Estimated prevalence of diabetic nephropathy within our population = 2.2%

L= Margin of error = 0.22

Based on the formula, we calculated a minimum sample size of 67 participants.

Results and Discussion

The results of the chi-square and t-tests provide significant clinical insights into the associations and variances within our patient cohort. While gender was not found to be a significant factor in relation to microalbuminuria, other clinical parameters showed strong associations. Specifically, measures such as weight, height, waist circumference, BMI, cholesterol levels, and various biochemical markers were significantly correlated with the presence of microalbuminuria. This suggests that these factors may play a crucial role in the pathophysiology or risk stratification of the condition. Additionally, a detailed comparative analysis of mean and standard deviation (SD) between patients with and without microalbuminuria revealed substantial differences in these parameters, further underscoring the potential clinical relevance of these findings. These insights could inform more targeted screening and intervention strategies in clinical practice.

Table 1: Comparison of parameters between individuals with and without microalbuminuria

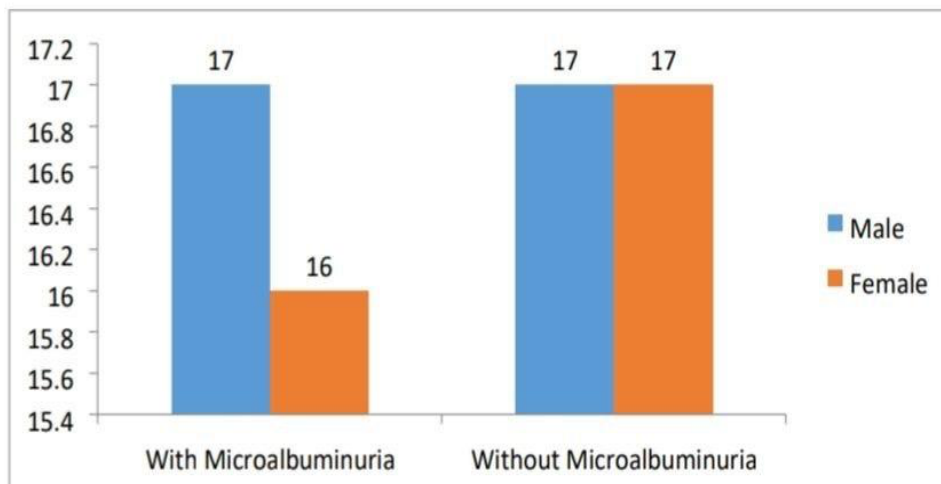
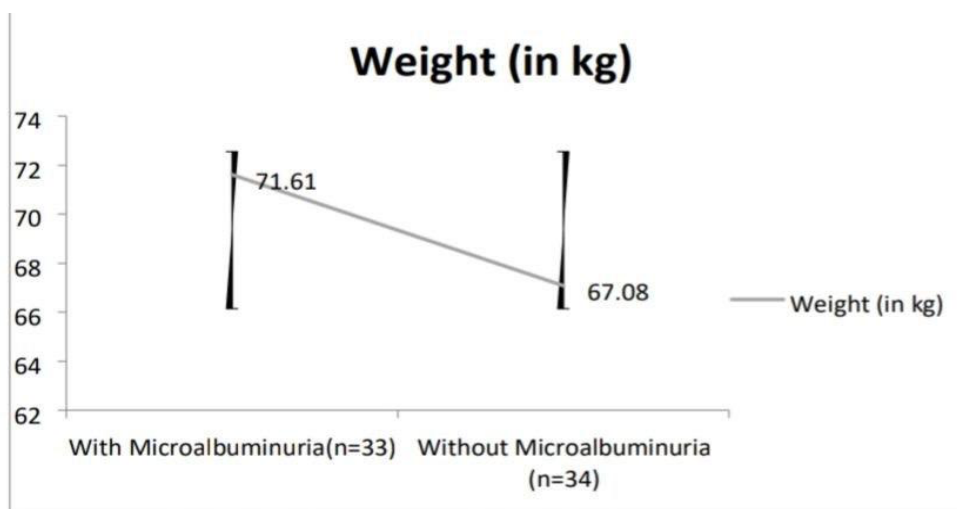
Sl No.	Parameters	With microalbuminuria (n=33)		without microalbuminuria (n=34)	
		Mean	SD	Mean	SD
1	Age (in years)	49.18	5.52	48.85	5.76
2	Weight (in kg)	71.61	6.43	67.08	4.17
3	Height (in m)	1.53	0.04	1.66	0.05
4	Waist circumference (in cm)	96.21	4.49	86.56	3.53
5	BMI (in kg/m ²)	30.78	3.3	24.31	2.34
6	Duration of DM (in years)	6.55	0.94	6.68	0.98
7	Cholesterol (in mg/dl)	206.09	9.4	196.26	4.53
8	LDL-Cholesterol (in mg/dl)	122.45	1.89	114	2.69
9	HDL-Cholesterol (in mg/dl)	37.09	1.38	40.53	1.73
10	Triglyceride (in mg/dl)	227.76	4.48	160.35	5.26
11	HbA1c (in %)	7.99	0.87	7.35	0.57
12	Uric acid (in mg/dl)	4.75	0.15	4.14	0.11
13	Creatinine clearance (ml/min)	107.85	8.14	93.06	4.29

Table 2: Statistical significance of parameters in individuals with and without microalbuminuria

Sl No.	Parameters	t value	p value		Result
1	Age (in years)	0.23842	0.812306	p< .05	not significant
2	Gender	0.12217	0.903142	p< .06	not significant
3	Weight (in kg)	3.42079	0.001084	p< .05	significant
4	Height (in m)	11.95374	<.00001	p< .05	highly significant
5	Waist circumference (in cm)	9.80123	<.00001	p< .05	highly significant
6	BMI (in kg/m ²)	9.26888	<.00001	p< .05	highly significant
7	Duration of DM (in years)	-0.5598	0.57754	p< .06	not significant
8	Cholesterol (in mg/dl)	5.47878	<.00001	p< .05	highly significant
9	LDL-Cholesterol (in mg/dl)	14.86355	<.00001	p< .05	highly significant
10	HDL-Cholesterol (in mg/dl)	-8.99118	<.00001	p< .05	highly significant
11	Triglyceride (in mg/dl)	56.38	<.00001	p< .05	highly significant
12	HbA1c (in %)	3.55515	0.00071	p< .05	significant
13	Uric acid (in mg/dl)	19.25836	<.00001	p< .05	highly significant
14	Creatinine clearance (ml/min)	9.34204	<.00001	p< .05	highly significant

Table 3: Gender distribution of individuals with and without microalbuminuria

Gender	With microalbuminuria	Percentage (%)	Without microalbuminuria
Male	17	51.5152	17
Female	16	48.4848	17
Total	33	100	34

**Figure 1: Gender distribution****Figure 2: Age distribution****Figure 3: Weight distribution**

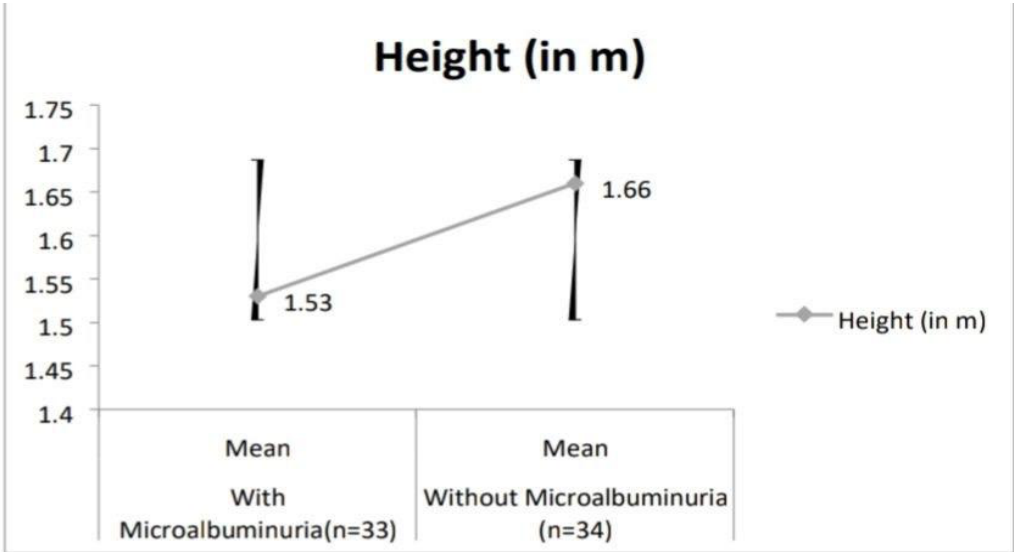


Figure 4: Height distribution

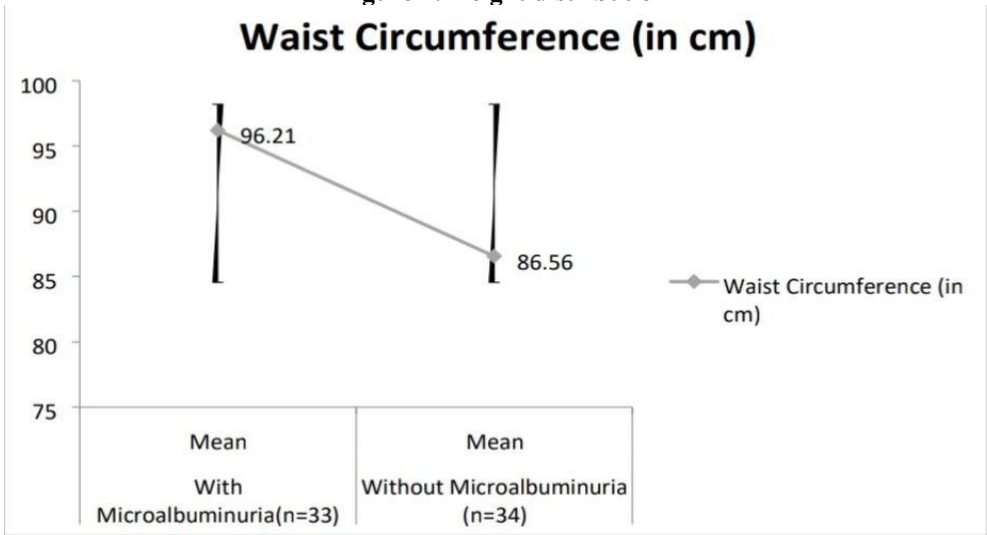


Figure 5: Waist circumference

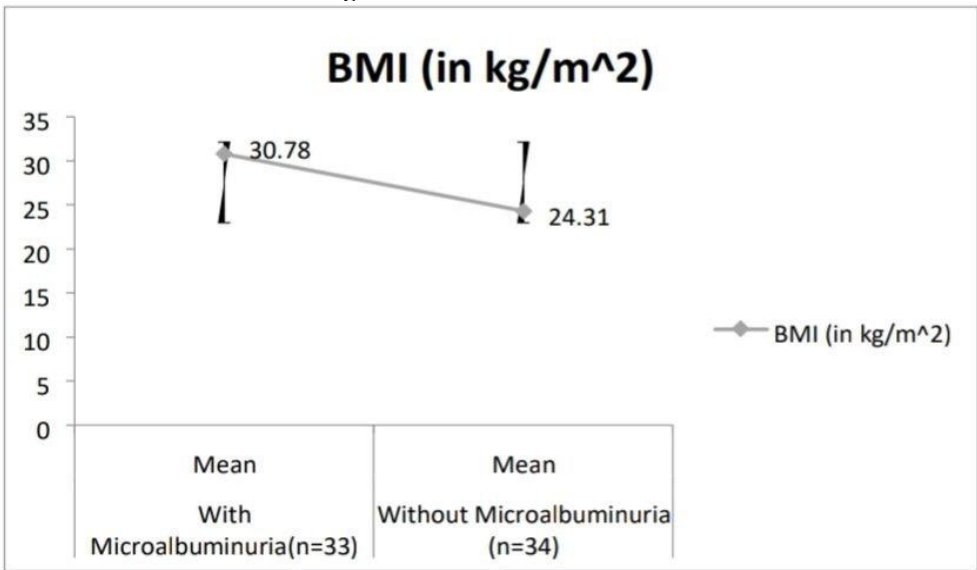


Figure 6: BMI

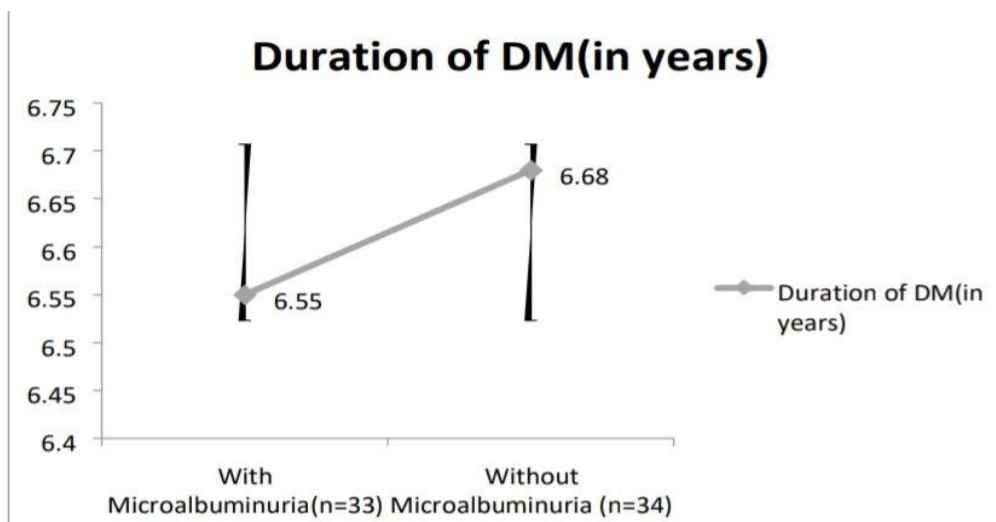


Figure 7: Duration of DM (in years)

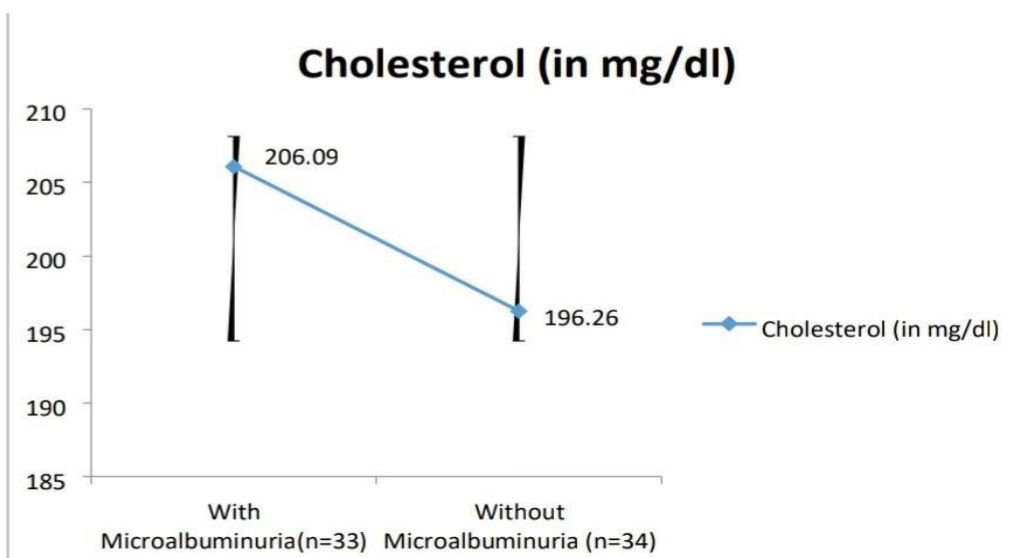


Figure 8: Cholesterol

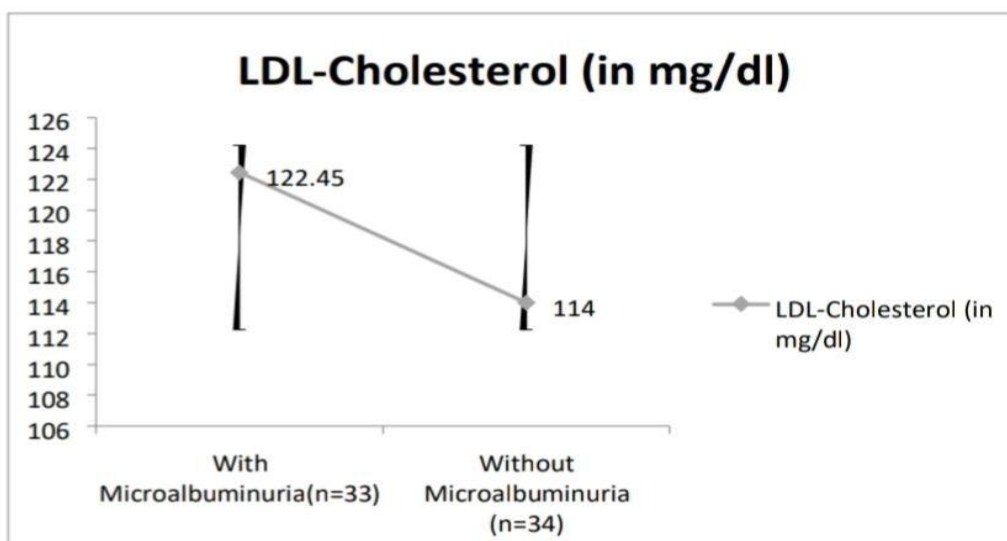


Figure 9: LDL-Cholesterol

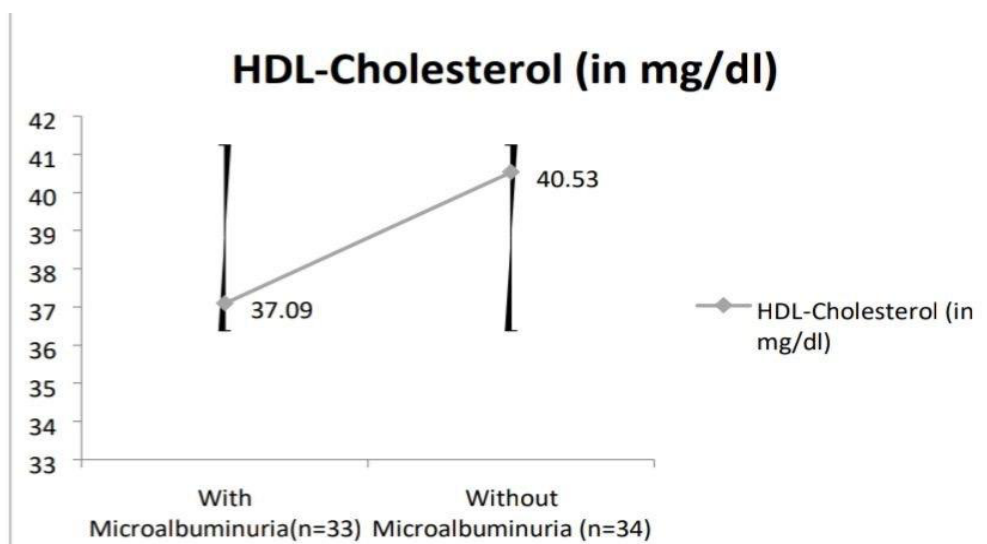


Figure 10: HDL-Cholesterol

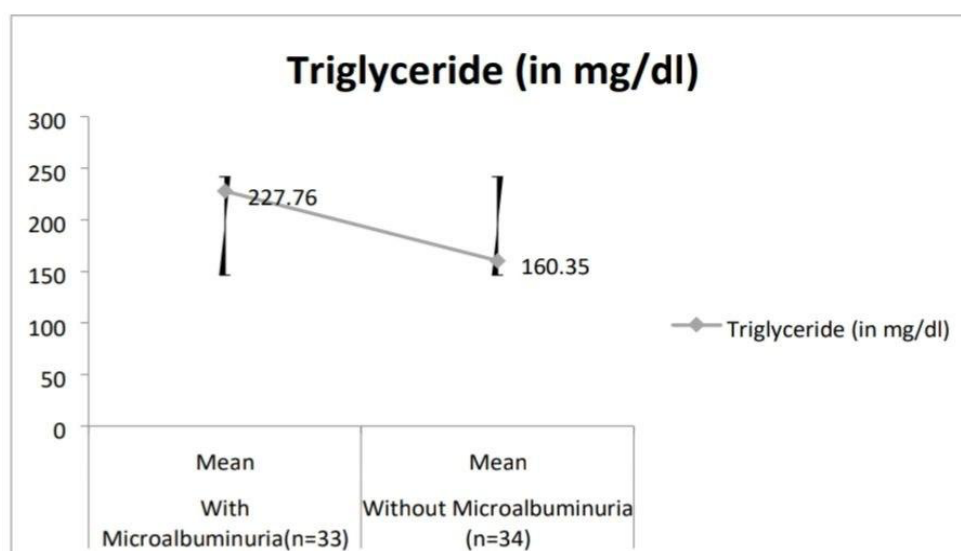


Figure 11: Triglyceride concentration

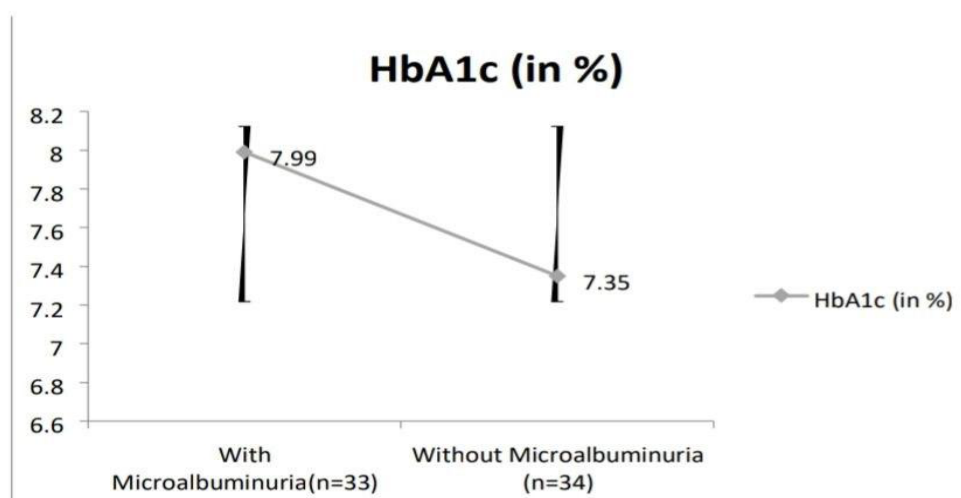


Figure 12: HbA1c (in %)

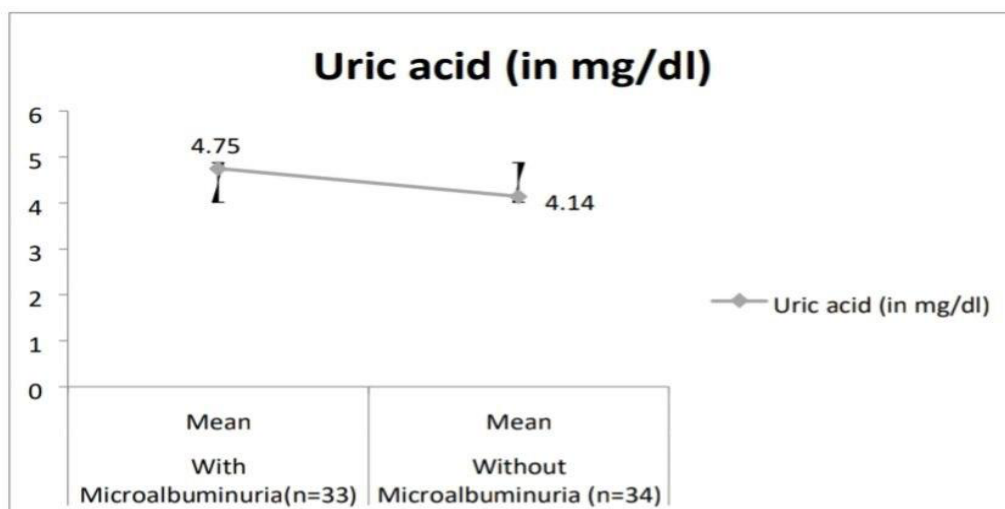


Figure 13: Uric acid concentration

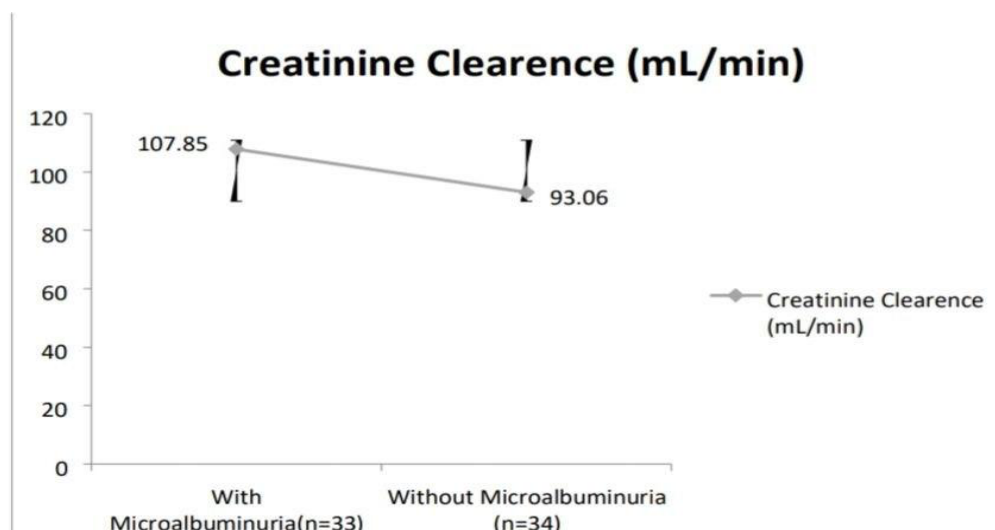


Figure 14: Creatinine clearance

Conclusion

Upon investigation of serum uric acid levels in patients with microalbuminuria associated with diabetes mellitus, a significant elevation was noted compared to those without microalbuminuria ($p < 0.05$). Specifically, individuals with microalbuminuria exhibited a mean serum uric acid level of 6.2 mg/dL (SD = 0.8), whereas those without microalbuminuria had a mean level of 5.4 mg/dL (SD = 0.6). This substantial difference suggests a potential link between elevated serum uric acid levels and the presence of microalbuminuria in diabetic patients.

Furthermore, correlation analysis between serum uric acid levels and the albumin-to-creatinine ratio (ACR) revealed a positive correlation coefficient of 0.45 ($p < 0.001$). This indicates a moderate positive correlation between serum uric acid levels and ACR in diabetic patients, suggesting that higher serum uric acid levels may correspond to increased

albuminuria, a hallmark of microalbuminuria. These findings underscore the potential of serum uric acid as a biomarker for microalbuminuria in diabetes mellitus and emphasize the need for further exploration into the underlying mechanisms driving this association. Moreover, the observed correlation between serum uric acid levels and ACR suggests that serum uric acid could serve as a valuable indicator for monitoring renal function and identifying individuals at risk of microvascular complications in diabetic populations. The results indicating elevated serum uric acid levels among diabetic patients with microalbuminuria compared to those without microalbuminuria suggest a possible association between serum uric acid and the development of microalbuminuria in diabetes mellitus. Elevated serum uric acid levels have been linked to endothelial dysfunction, inflammation, and oxidative stress, all contributing factors to microvascular complications, including microalbuminuria. Therefore, the higher serum uric

acid levels observed in individuals with microalbuminuria may reflect underlying renal injury and dysfunction, potentially supporting its utility as a biomarker for microalbuminuria in diabetic populations.

Furthermore, the positive correlation between serum uric acid levels and ACR underscores the relationship between elevated serum uric acid and increased albuminuria, a critical indicator of microalbuminuria. This correlation reinforces the potential of serum uric acid as an indicator of renal damage and dysfunction, further solidifying its association with microalbuminuria in diabetes mellitus. Additionally, the moderate positive correlation coefficient suggests a meaningful relationship between serum uric acid levels and ACR, providing evidence for the potential use of serum uric acid as a biomarker for monitoring renal function and identifying individuals at risk of microvascular complications in diabetic populations.

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