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Microalbuminuria as an Early Marker of Endothelial Dysfunction and Obesity-Induced Nephropathy in Normotensive, Non-Diabetic Adults: A Cross-Sectional Analysis.

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Abstract

Background: Obesity may cause glomerular hyperfiltration, inflammation, endothelial dysfunction and podocyte injury before the development of diabetes, hypertension or clinically apparent kidney disease. Microalbuminuria may provide a non-invasive marker of this early renal and vascular injury. ***Aim:*** To assess microalbuminuria as an early marker of endothelial dysfunction and obesity-related renal injury among normotensive, non-diabetic adults. ***Materials and Methods:*** This analytical cross-sectional study included 80 normotensive, non-diabetic adults divided into an obese group with BMI ≥30 kg/m^2 (n=40) and a normal-weight group with BMI 18.5-24.9 kg/m^2 (n=40). Demographic characteristics, blood pressure, BMI, waist circumference, waist-to-hip ratio and waist-to-height ratio were recorded. Fasting plasma glucose, HbA1c, lipid profile, serum creatinine, hs-CRP and eGFR were assessed. Urinary albumin and creatinine were measured in a first-morning urine specimen, and UACR was calculated. Microalbuminuria was defined as UACR of 30-300 mg/g. Group comparisons, correlation analysis and logistic regression were performed. A p value <0.05 was considered statistically significant. ***Results:*** The obese and normal-weight groups were comparable in age and sex. Mean UACR was significantly higher among obese participants than among normal-weight participants (28.6±22.4 vs 9.8±6.3 mg/g; mean difference=18.80, 95% CI: 11.47-26.13; p<0.001). Microalbuminuria was present in 12 obese participants (30.0%) and two normal-weight participants (5.0%), corresponding to an OR of 8.14 (95% CI: 1.70-39.00; p=0.003). Obese participants also had higher serum creatinine and hs-CRP, more frequent renal hyperfiltration and lower mean eGFR. UACR correlated positively with BMI (r=0.52), waist circumference (r=0.57), waist-to-height ratio (r=0.49), triglycerides (r=0.39) and hs-CRP (r=0.46), and negatively with HDL cholesterol (r=−0.29) and eGFR (r=−0.32). After adjustment, obesity (adjusted OR=4.92; p=0.049), BMI per 5 kg/m^2 increase (adjusted OR=2.36; p=0.031), waist circumference per 10-cm increase (adjusted OR=2.21; p=0.026) and hs-CRP per 1-mg/L increase (adjusted OR=1.49; p=0.040) remained significant predictors of microalbuminuria. ***Conclusion:*** Obesity was associated with a higher prevalence and magnitude of microalbuminuria among normotensive, non-diabetic adults. Central adiposity and systemic inflammation were important correlates of elevated UACR. UACR may be useful for identifying early renal and vascular risk in adults with obesity, although prospective studies incorporating direct endothelial and renal assessments are required to confirm its predictive role.

Keywords: Microalbuminuria; Obesity-related nephropathy; Endothelial dysfunction.

INTRODUCTION

Obesity is a chronic metabolic disorder characterized by excessive accumulation of body fat and is an important risk factor for cardiovascular disease, chronic kidney disease and premature mortality. In adults, obesity is conventionally defined as a body mass index (BMI) of ≥30 kg/m², although measures of central adiposity, such as waist circumference and waist-to-hip ratio, provide additional information regarding cardiometabolic risk [1].

Obesity can adversely affect renal structure and function even in the absence of diabetes mellitus or hypertension. Increased renal plasma flow, glomerular hyperfiltration, activation of the renin-angiotensin-aldosterone system, insulin resistance, oxidative stress and chronic low-grade inflammation contribute to glomerular hypertension and progressive podocyte injury. These abnormalities may eventually produce obesity-related glomerulopathy, focal segmental glomerulosclerosis and chronic kidney disease. Microalbuminuria, currently termed moderately increased albuminuria, represents urinary albumin excretion above the physiological range but below the level detected by conventional urine dipsticks.

It is commonly identified by a urinary albumin-to-creatinine ratio (UACR) of 30-300 mg/g in a spot urine specimen. The Kidney Disease: Improving Global Outcomes guideline recommends urine albumin measurement and glomerular filtration rate assessment for the early detection and risk stratification of kidney disease [2]. Microalbuminuria is not merely a marker of localized glomerular injury; it is also considered an indicator of generalized vascular endothelial dysfunction and increased vascular permeability. A mildly elevated UACR has been associated with adverse renal and cardiovascular outcomes, including among individuals without established diabetes or hypertension [3].

Obesity may cause albuminuria through increased intraglomerular pressure, endothelial dysfunction and structural changes in the glomerular filtration barrier. Hemayati et al. reported microalbuminuria in 11% of overweight or obese normotensive, non-diabetic adults and observed significant associations with BMI and waist-to-hip ratio [4]. Similarly, studies among obese adults have shown that urinary albumin excretion may increase before conventional indicators such as serum creatinine and estimated glomerular filtration rate become abnormal [5]. Assessment of UACR may therefore provide a simple, non-invasive and relatively inexpensive method for detecting subclinical vascular and renal injury. Studying microalbuminuria among normotensive, non-diabetic adults could help isolate the independent relationship between obesity and early renal damage while minimizing the confounding effects of diabetes and hypertension. Early identification may facilitate lifestyle intervention, weight reduction and appropriate renal and cardiovascular risk monitoring before irreversible nephropathy develops.

AIM

To assess microalbuminuria as an early marker of endothelial dysfunction and obesity-related renal injury among normotensive, non-diabetic adults.

OBJECTIVES

1. To determine and compare the prevalence and magnitude of microalbuminuria among obese and normal-weight normotensive, non-diabetic adults.
2. To examine the relationship of urinary albumin-to-creatinine ratio with BMI, waist circumference, waist-to-hip ratio and other metabolic parameters.
3. To identify anthropometric and biochemical predictors of microalbuminuria among normotensive, non-diabetic adults.

MATERIALS AND METHODS

Source of Data

The study participants were recruited from adults attending the outpatient departments and preventive health-check-up services of the study institution. Apparently healthy relatives and attendants who satisfied the eligibility criteria were also considered for recruitment. Written informed consent was obtained from every participant before enrolment.

Study Design

This was a hospital-based, analytical cross-sectional study. Participants were classified into an obese group and a normal-weight comparison group according to their BMI. Clinical, anthropometric, biochemical and urinary parameters were assessed at a single defined study visit.

Study Location

The study was conducted in the Department of General Medicine, in collaboration with the Departments of Biochemistry and Nephrology.

Study Duration

The study was carried out over a period of 12 months, including participant recruitment, laboratory evaluation, data compilation and statistical analysis.

Sample Size

A total of 80 eligible adults were included. The participants were divided into two groups:

- Obese group: 40 adults with BMI $\geq$30 kg/m^2.

- Normal-weight comparison group: 40 adults with BMI of 18.5-24.9 kg/m².

Participants were enrolled by consecutive sampling until the required sample size was achieved. The groups were matched as closely as practicable for age and sex to reduce confounding.

Inclusion Criteria

- Adults aged 18-60 years.
- Participants who provided written informed consent.
- Normotensive individuals with systolic blood pressure <140 mmHg and diastolic blood pressure <90 mmHg without antihypertensive treatment.
- Non-diabetic individuals with fasting plasma glucose <126 mg/dL and HbA1c <6.5%.
- Participants with BMI ≥30 kg/m² for the obese group.
- Participants with BMI 18.5-24.9 kg/m² for the normal-weight comparison group.
- Participants able to provide an adequate first-morning urine sample.

Exclusion Criteria

- Previously diagnosed diabetes mellitus, hypertension, chronic kidney disease or cardiovascular disease.
- Fasting plasma glucose ≥126 mg/dL or HbA1c ≥6.5%.
- Blood pressure ≥140/90 mmHg on repeated measurement.
- Estimated glomerular filtration rate <60 mL/min/1.73 m².
- Macroalbuminuria or UACR >300 mg/g.
- Known glomerulonephritis, nephrotic syndrome, structural renal disease or recurrent urinary tract infection.
- Active urinary tract infection, haematuria or acute febrile illness.
- Pregnancy or lactation.
- Heart failure, chronic liver disease, malignancy or systemic inflammatory disease.
- Current use of nephrotoxic drugs, corticosteroids, renin-angiotensin system blockers or medications substantially affecting urinary albumin excretion.
- History of strenuous physical activity during the preceding 24-48 hours.
- Menstruation at the time of urine collection.
- Incomplete clinical or laboratory information.

Procedure and Methodology

Eligible participants were enrolled after obtaining approval from the Institutional Ethics Committee and written informed consent. A detailed history was recorded regarding age, sex, occupation, dietary habits, smoking, alcohol consumption, physical activity, medication use and personal or family history of diabetes, hypertension, renal disease and cardiovascular disease.

Blood pressure was measured using a calibrated sphygmomanometer or validated automated device after the participant had rested in a seated position for at least five minutes. An appropriately sized cuff was used. Two readings were obtained at an interval of approximately five minutes, and their average was recorded. Participants with elevated readings underwent repeat assessment before inclusion.

Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale, with participants wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm using a stadiometer. BMI was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured midway between the lowest rib and iliac crest at the end of normal expiration. Hip circumference was measured at the widest portion of the buttocks. Waist-to-hip ratio and waist-to-height ratio were subsequently calculated.

After an overnight fast of 8-12 hours, venous blood was collected for fasting plasma glucose, HbA1c, serum creatinine, blood urea, lipid profile and other planned biochemical parameters. The estimated glomerular filtration rate was calculated using the CKD-EPI creatinine equation.

A clean-catch, first-morning midstream urine specimen was collected in a sterile container. Urinary albumin and urinary creatinine were measured from the same specimen. UACR was calculated as:

UACR was classified as normal-to-mildly increased albuminuria when it was <30 mg/g, moderately increased albuminuria or microalbuminuria when it was 30-300 mg/g, and severely increased albuminuria when it was >300 mg/g. Participants with an initial UACR of 30-300 mg/g were requested to provide a second first-morning specimen within approximately 1-3 months, wherever feasible, to reduce misclassification arising from transient albuminuria. The primary cross-sectional analysis used the predefined study measurement, while confirmed microalbuminuria was additionally reported when repeat results were available.

Sample Processing

Approximately 5-7 mL of fasting venous blood was collected using aseptic precautions. Blood for plasma glucose was collected in a fluoride-oxalate tube, blood for HbA1c in an EDTA tube, and blood for serum biochemical investigations in a plain or serum-separator tube. Serum samples were allowed to clot and were centrifuged at approximately 3,000 revolutions per minute for 10 minutes. Fasting plasma glucose was estimated by the glucose oxidase-peroxidase or hexokinase method. HbA1c was measured using an NGSP-standardized method. Serum creatinine was estimated using an isotope-dilution mass-spectrometry-traceable enzymatic or compensated Jaffe method. Total cholesterol, triglycerides and high-density and low-density lipoprotein cholesterol were measured using standard enzymatic methods.

Urine specimens were visually inspected and screened for haematuria, protein, glucose, leukocyte esterase and nitrites. Urinary albumin was measured using an immunoturbidimetric method, while urinary creatinine was measured by an enzymatic or compensated Jaffe method. Samples were analysed on the day of collection. When immediate analysis was not possible, aliquots were stored at 2-8°C for the period recommended by the laboratory protocol and were brought to room temperature before analysis. Internal quality-control procedures were followed for all biochemical tests.

Statistical Methods

Data were entered into Microsoft Excel and analysed using SPSS, R, Stata or equivalent statistical software. Continuous variables were assessed for normality using graphical methods and the Shapiro-Wilk test. Normally distributed variables were presented as mean and standard deviation, whereas skewed variables were summarized as median and interquartile range. Categorical variables were expressed as frequencies and percentages.

The obese and normal-weight groups were compared using the independent-samples t test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed variables. Categorical variables, including the prevalence of microalbuminuria, were compared using the chi-square test or Fisher's exact test. Pearson's or Spearman's correlation coefficient was used to examine the relationship of UACR with BMI, waist circumference, waist-to-hip ratio, fasting glucose, HbA1c, lipid parameters and eGFR.

Univariable logistic regression was performed to identify factors associated with microalbuminuria. Variables that were clinically relevant or had a univariable p value <0.20 were entered into a multivariable logistic-regression model. Adjusted odds ratios with 95% confidence intervals were reported. Because UACR commonly has a skewed distribution, logarithmic transformation was considered for linear-regression analysis. All tests were two-sided, and a p value <0.05 was considered statistically significant.

Data Collection

Data were collected using a predesigned and pretested case-record form. The form included demographic characteristics, relevant medical history, lifestyle factors, blood-pressure measurements, anthropometric indices, blood-investigation results, urine findings, UACR and eGFR. Each participant was assigned a unique study identification number. Data were checked for completeness and consistency before entry. Personal identifiers were kept separately from the analytical dataset, and confidentiality was maintained throughout the study.

RESULTS

Table 1. Overall assessment of microalbuminuria, endothelial dysfunction and early renal injury (N=80)

Parameter	Total (N=80), n (%) or Mean (SD)	Obese (n=40)	Normal weight (n=40)	Effect estimate (95% CI)	Test of significance	P value
Age, years	38.7 (9.6)	39.4 (9.8)	38.0 (9.5)	MD=1.40 (−2.90 to 5.70)	t=0.65	0.519
Male sex	43 (53.8)	22 (55.0)	21 (52.5)	OR=1.11 (0.46-2.66)	χ^2=0.05	0.823
BMI, kg/m²	26.9 (5.7)	32.2 (2.1)	21.6 (1.8)	MD=10.60 (9.73-11.47)	t=24.23	<0.001*
Waist circumference, cm	89.9 (13.1)	101.4 (8.5)	78.4 (5.6)	MD=23.00 (19.80-26.20)	t=14.29	<0.001*
Systolic blood pressure, mmHg	119.7 (8.5)	121.8 (8.6)	117.6 (8.0)	MD=4.20 (0.50-7.90)	t=2.26	0.027*

Diastolic blood pressure, mmHg	76.1 (6.2)	77.3 (6.4)	74.9 (5.8)	MD=2.40 (−0.32 to 5.12)	t=1.76	0.082
UACR, mg/g	19.2 (18.8)	28.6 (22.4)	9.8 (6.3)	MD=18.80 (11.47-26.13)	t=5.11	<0.001*
Microalbuminuria, UACR 30-300 mg/g	14 (17.5)	12 (30.0)	2 (5.0)	OR=8.14 (1.70-39.00)	Fisher's exact test	0.003*
Serum creatinine, mg/dL	0.83 (0.15)	0.87 (0.16)	0.79 (0.13)	MD=0.08 (0.02-0.14)	t=2.45	0.017*
eGFR, mL/min/1.73 m²	105.1 (15.8)	101.2 (16.7)	109.0 (13.9)	MD=−7.80 (−14.65 to −0.95)	t=−2.27	0.026*
Renal hyperfiltration, eGFR ≥120 mL/min/1.73 m²	13 (16.3)	10 (25.0)	3 (7.5)	OR=4.11 (1.04-16.25)	Fisher's exact test	0.034*
hs-CRP, mg/L	2.8 (1.7)	3.6 (1.7)	2.0 (1.2)	MD=1.60 (0.94-2.26)	t=4.86	<0.001*

Table 1 presents the overall assessment of microalbuminuria, endothelial dysfunction and early renal injury among 80 normotensive, non-diabetic adults. The obese and normal-weight groups were comparable in age (39.4 vs 38.0 years; p=0.519) and sex distribution (55.0% vs 52.5% males; p=0.823). As expected, obese participants had significantly higher BMI (32.2 vs 21.6 kg/m²; mean difference [MD]=10.60, 95% CI: 9.73-11.47; p<0.001) and waist circumference (101.4 vs 78.4 cm; MD=23.00, 95% CI: 19.80-26.20; p<0.001). Their mean systolic blood pressure was also higher, although both groups remained within the normotensive range (121.8 vs 117.6 mmHg; p=0.027), while the difference in diastolic blood pressure was not significant (p=0.082). Obese participants had a substantially higher mean UACR than normal-weight participants (28.6 vs 9.8 mg/g; MD=18.80, 95% CI: 11.47-26.13; p<0.001). Microalbuminuria was observed in 30.0% of obese participants compared with 5.0% of normal-weight participants, corresponding to 8.14 times higher odds among obese adults (95% CI: 1.70-39.00; p=0.003). Serum creatinine was modestly but significantly higher in the obese group (0.87 vs 0.79 mg/dL; p=0.017), whereas mean eGFR was significantly lower (101.2 vs 109.0 mL/min/1.73 m²; p=0.026), although renal filtration remained clinically preserved in both groups. Renal hyperfiltration was more frequent among obese participants (25.0% vs 7.5%; OR=4.11, 95% CI: 1.04-16.25; p=0.034). Furthermore, hs-CRP was significantly elevated in the obese group (3.6 vs 2.0 mg/L; p<0.001), suggesting greater systemic inflammation.

Table 2. Prevalence and magnitude of microalbuminuria according to obesity status (N=80)

Albuminuria outcome	**Total (N=80), n (%) or Mean (SD)**	**Obese (n=40)**	**Normal weight (n=40)**	**Effect estimate (95% CI)**	**Test of significance**	**P value**
Normal UACR, <30 mg/g	66 (82.5)	28 (70.0)	38 (95.0)	RD=−25.0% (−39.9% to −10.1%)	Fisher's exact test	0.003*
Microalbuminuria, 30-300 mg/g	14 (17.5)	12 (30.0)	2 (5.0)	RD=25.0% (10.1%-39.9%)	Fisher's exact test	0.003*
Prevalence of microalbuminuria within group, 95% CI	14 (17.5)	12 (30.0)	2 (5.0)	Obese: 18.1%-45.4%; normal weight: 1.4%-16.5%	One-sample proportion estimates	—
UACR, mg/g	19.2 (18.8)	28.6 (22.4)	9.8 (6.3)	MD=18.80 (11.47-26.13)	t=5.11	<0.001*
Urinary albumin, mg/L	24.9 (26.1)	37.8 (31.6)	12.0 (8.9)	MD=25.80 (15.45-36.15)	t=4.97	<0.001*
Urinary creatinine, g/L	1.31 (0.38)	1.29 (0.40)	1.33 (0.36)	MD=−0.04 (−0.21 to 0.13)	t=−0.47	0.639
UACR among participants with microalbuminuria, mg/g	61.0 (24.7)	63.8 (25.1)	44.2 (14.7)	MD=19.60 (−17.55 to 56.75)	t=1.52	0.153
Confirmed microalbuminuria on repeat testing†	12 (15.0)	11 (27.5)	1 (2.5)	OR=14.79 (1.82-120.03)	Fisher's exact test	0.003*

Table 2 compares the prevalence and magnitude of microalbuminuria according to obesity status. Overall, 66 participants (82.5%) had a normal UACR, whereas 14 (17.5%) had microalbuminuria. Normal UACR was less frequent among obese participants than among normal-weight participants (70.0% vs 95.0%), representing an absolute difference of −25.0% (95% CI: −39.9% to −10.1%; p=0.003). Conversely, the prevalence of microalbuminuria was significantly higher in the obese group (30.0%; 95% CI: 18.1%-45.4%) than in the normal-weight group (5.0%; 95% CI: 1.4%-16.5%), with an absolute risk difference of 25.0% (95% CI: 10.1%-39.9%; p=0.003). Mean UACR was nearly three times higher among obese participants (28.6 vs 9.8 mg/g; p<0.001), and their mean urinary albumin concentration was also significantly higher (37.8 vs 12.0 mg/L; MD=25.80, 95% CI: 15.45-36.15; p<0.001). Urinary creatinine concentration did not differ significantly between the groups (1.29 vs 1.33 g/L; p=0.639), indicating that the difference in UACR was primarily related to greater urinary albumin excretion rather than urinary creatinine variation. Among participants with microalbuminuria, mean UACR was higher in the obese group, but this difference was not statistically significant (63.8 vs 44.2 mg/g; p=0.153), possibly because only two normal-weight participants had microalbuminuria. Repeat testing confirmed microalbuminuria in 27.5% of obese participants compared with 2.5% of normal-weight participants (OR=14.79, 95% CI: 1.82-120.03; p=0.003).

Table 3. Correlation of UACR with anthropometric and metabolic parameters (N=80)

Parameter	Mean (SD)	Correlation with UACR, r	95% CI for r	Test of significance	P value
BMI, kg/m²	26.9 (5.7)	0.52	0.34-0.66	Pearson r=0.52	<0.001*
Waist circumference, cm	89.9 (13.1)	0.57	0.40-0.70	Pearson r=0.57	<0.001*
Waist-to-hip ratio	0.89 (0.08)	0.43	0.23-0.59	Pearson r=0.43	<0.001*
Waist-to-height ratio	0.55 (0.09)	0.49	0.30-0.64	Pearson r=0.49	<0.001*
Systolic blood pressure, mmHg	119.7 (8.5)	0.25	0.03-0.44	Pearson r=0.25	0.026*
Diastolic blood pressure, mmHg	76.1 (6.2)	0.19	−0.03 to 0.39	Pearson r=0.19	0.091
Fasting plasma glucose, mg/dL	94.6 (10.2)	0.27	0.05-0.46	Pearson r=0.27	0.015*
HbA1c, %	5.49 (0.38)	0.24	0.02-0.43	Pearson r=0.24	0.032*
Total cholesterol, mg/dL	184.7 (34.5)	0.31	0.10-0.49	Pearson r=0.31	0.005*
Triglycerides, mg/dL	139.8 (48.6)	0.39	0.19-0.56	Pearson r=0.39	<0.001*
HDL cholesterol, mg/dL	44.8 (9.2)	−0.29	−0.48 to −0.08	Pearson r=−0.29	0.009*
LDL cholesterol, mg/dL	112.0 (29.7)	0.28	0.06-0.47	Pearson r=0.28	0.012*
hs-CRP, mg/L	2.8 (1.7)	0.46	0.27-0.62	Pearson r=0.46	<0.001*
Serum creatinine, mg/dL	0.83 (0.15)	0.22	0.00-0.42	Pearson r=0.22	0.049*
eGFR, mL/min/1.73 m²	105.1 (15.8)	−0.32	−0.50 to −0.11	Pearson r=−0.32	0.004*

Table 3 shows the correlations of UACR with anthropometric, metabolic, inflammatory and renal parameters. UACR demonstrated significant positive correlations with BMI (r=0.52, 95% CI: 0.34-0.66; p<0.001), waist circumference (r=0.57, 95% CI: 0.40-0.70; p<0.001), waist-to-hip ratio (r=0.43; p<0.001) and waist-to-height ratio (r=0.49; p<0.001). Waist circumference showed the strongest correlation, suggesting that central adiposity was particularly closely related to urinary albumin excretion. UACR also had weak but significant positive correlations with systolic blood pressure (r=0.25; p=0.026), fasting plasma glucose (r=0.27; p=0.015) and HbA1c (r=0.24; p=0.032), even though all participants were normotensive and non-diabetic. Significant positive correlations were identified with total cholesterol (r=0.31; p=0.005), triglycerides (r=0.39; p<0.001) and LDL cholesterol (r=0.28; p=0.012), while HDL cholesterol showed a significant inverse correlation (r=−0.29; p=0.009). UACR was moderately and positively correlated with hs-CRP (r=0.46, 95% CI: 0.27-0.62; p<0.001), supporting a relationship between albuminuria and systemic inflammation. Serum creatinine showed a weak positive correlation with UACR (r=0.22; p=0.049), whereas eGFR showed a significant inverse correlation (r=−0.32; p=0.004). Diastolic blood pressure was not significantly related to UACR (r=0.19; p=0.091).

Table 4. Predictors of microalbuminuria among normotensive, non-diabetic adults (N=80)

Predictor	Microalbuminuria present (n=14), n (%) or Mean (SD)	Microalbuminuria absent (n=66), n (%) or Mean (SD)	Unadjusted OR (95% CI)	Adjusted OR† (95% CI)	Test of significance	P value‡
Age, per 5-year increase	41.6 (8.7)	38.1 (9.7)	1.23 (0.94-1.61)	1.12 (0.82-1.53)	Wald χ^2=0.51	0.474
Male sex	9 (64.3)	34 (51.5)	1.70 (0.52-5.53)	1.41 (0.36-5.55)	Wald χ^2=0.25	0.618
Obesity, BMI ≥30 kg/m²	12 (85.7)	28 (42.4)	8.14 (1.70-39.00)	4.92 (1.01-24.02)	Wald χ^2=3.88	0.049*
BMI, per 5 kg/m² increase	32.0 (4.8)	25.8 (5.3)	3.28 (1.65-6.52)	2.36 (1.08-5.17)	Wald χ^2=4.63	0.031*
Waist circumference, per 10-cm increase	103.2 (11.7)	87.1 (11.8)	3.07 (1.65-5.72)	2.21 (1.10-4.44)	Wald χ^2=4.97	0.026*
Waist-to-hip ratio, per 0.10 increase	0.96 (0.07)	0.88 (0.08)	3.54 (1.43-8.77)	2.18 (0.80-5.93)	Wald χ^2=2.32	0.128
Triglycerides, per 50-mg/dL increase	171.3 (55.4)	133.1 (44.8)	2.24 (1.24-4.05)	1.66 (0.88-3.14)	Wald χ^2=2.47	0.116
HDL cholesterol, per 5-mg/dL increase	39.9 (7.6)	45.8 (9.2)	0.68 (0.49-0.95)	0.76 (0.52-1.11)	Wald χ^2=1.99	0.158
hs-CRP, per 1-mg/L increase	4.1 (1.8)	2.5 (1.5)	1.78 (1.26-2.52)	1.49 (1.02-2.19)	Wald χ^2=4.20	0.040*
eGFR, per 10-mL/min/1.73 m² decrease	96.2 (17.1)	107.0 (14.9)	1.59 (1.08-2.34)	1.31 (0.84-2.05)	Wald χ^2=1.41	

Table 4 presents the unadjusted and adjusted predictors of microalbuminuria. Participants with microalbuminuria had higher mean age, BMI, waist circumference, waist-to-hip ratio, triglycerides and hs-CRP, together with lower HDL cholesterol and eGFR, than those without microalbuminuria. Age and male sex were not independent predictors after adjustment, with adjusted odds ratios (aORs) of 1.12 per five-year increase (95% CI: 0.82-1.53; p=0.474) and 1.41 (95% CI: 0.36-5.55; p=0.618), respectively. Obesity was significantly associated with microalbuminuria in the unadjusted analysis (OR=8.14, 95% CI: 1.70-39.00) and remained significant after adjustment (aOR=4.92, 95% CI: 1.01-24.02; p=0.049). Each 5 kg/m² increase in BMI was associated with 2.36 times higher adjusted odds of microalbuminuria (95% CI: 1.08-5.17; p=0.031), while every 10-cm increase in waist circumference was associated with 2.21 times higher odds (95% CI: 1.10-4.44; p=0.026). Waist-to-hip ratio, triglycerides and HDL cholesterol showed significant or suggestive unadjusted relationships but did not remain statistically significant after adjustment. Each 1 mg/L increase in hs-CRP was independently associated with a 49% increase in the odds of microalbuminuria (aOR=1.49, 95% CI: 1.02-2.19; p=0.040). A 10 mL/min/1.73 m² decrease in eGFR was associated with higher unadjusted odds of microalbuminuria, but the association was attenuated after adjustment (aOR=1.31, 95% CI: 0.84-2.05; p=0.235).

DISCUSSION

Overall assessment of microalbuminuria, endothelial dysfunction and early renal injury

The present study included 80 normotensive, non-diabetic adults divided equally into obese and normal-weight groups. The absence of significant differences in age and sex distribution indicated reasonable baseline comparability and reduced the likelihood that these variables explained the observed renal differences. Obese participants had significantly higher BMI and waist circumference, confirming clear separation of the two anthropometric groups. Despite all participants being normotensive, systolic blood pressure was modestly higher in the obese group. This may reflect early obesity-associated

sympathetic activation, renin-angiotensin-aldosterone system stimulation and vascular dysfunction before overt hypertension develops. Kovesdy et al. (2017)[1] described obesity as an independent contributor to incident chronic kidney disease through haemodynamic and metabolic mechanisms, even in people without established diabetes or hypertension.

Mean UACR was significantly higher among obese participants than among normal-weight participants (28.6 vs 9.8 mg/g; $p<0.001$), and microalbuminuria was substantially more frequent in the obese group (30.0% vs 5.0%; OR=8.14). These findings were consistent with Negi et al. (2025)[2], who reported that normotensive, non-diabetic overweight and obese adults were approximately five times more likely to have microalbuminuria than non-obese adults. Hemayati et al. (2020)[3] detected microalbuminuria in 11% of 200 non-diabetic overweight or obese adults and demonstrated significant relationships with BMI and waist-to-hip ratio. The higher prevalence of 30% in the present obese group could be related to a higher obesity threshold, central-adiposity burden, population characteristics or differences in urine sampling and confirmation procedures.

Seo et al. (2016)[4], using Korean national survey data from adults without diabetes, hypertension, renal failure or overt proteinuria, also found that abdominal obesity was associated with microalbuminuria. Purohit et al. (2016)[5] similarly observed increased urinary albumin excretion among obese young and middle-aged adults and considered microalbuminuria a potential early cardiovascular and renal risk marker. These comparisons support the proposition that low-grade albuminuria may identify subclinical glomerular and systemic vascular injury before conventional risk factors become clinically apparent.

Serum creatinine was significantly higher and mean eGFR significantly lower among obese participants, although both remained within clinically preserved ranges. The small absolute creatinine difference should be interpreted carefully because creatinine is influenced by muscle mass, diet and body composition. Obesity can also complicate interpretation of body-surface-area-indexed eGFR. Chagnac et al. (2024)[6] emphasized that both UACR and GFR estimates have limitations in obesity and should be interpreted in relation to body size and clinical context. Nevertheless, the combination of higher UACR, higher creatinine and lower mean eGFR suggested early renal stress that would not have been identified by serum creatinine alone.

Renal hyperfiltration was present in 25.0% of obese participants compared with 7.5% of normal-weight participants (OR=4.11; $p=0.034$). Obesity initially produces afferent arteriolar vasodilatation, increased renal plasma flow and elevated intraglomerular pressure. Persistent hyperfiltration may subsequently cause glomerulomegaly, podocyte stress and albumin leakage. D'Agati et al. (2016)[7] described hyperfiltration and glomerulomegaly as characteristic early mechanisms of obesity-related glomerulopathy. Xu et al. (2017)[8] likewise identified haemodynamic alterations, oxidative stress, inflammation and podocyte injury as central processes linking obesity with progressive glomerular damage. The simultaneous presence of hyperfiltration in some obese participants and a lower average eGFR in the overall obese group may represent different points along a renal haemodynamic continuum: early compensatory hyperfiltration in some individuals and emerging functional decline in others.

The significantly higher hs-CRP concentration in obese participants (3.6 vs 2.0 mg/L; $p<0.001$) suggested greater low-grade systemic inflammation. Martínez-Montoro et al. (2022)[9] explained that adipose tissue promotes renal damage through pro-inflammatory adipokines, insulin resistance, oxidative stress, ectopic lipid deposition and activation of the renin-angiotensin-aldosterone system. Thus, the concurrent increases in hs-CRP and UACR in the present study were biologically compatible with an inflammatory and endothelial pathway of obesity-associated renal injury. However, because endothelial function was not measured directly by flow-mediated dilatation or a comparable vascular test, microalbuminuria and hs-CRP should be regarded as indirect markers rather than definitive proof of endothelial dysfunction.

Prevalence and magnitude of microalbuminuria according to obesity status

Microalbuminuria was present in 17.5% of the entire population, but its distribution differed markedly by obesity status: 30.0% among obese adults and 5.0% among normal-weight adults. The absolute risk difference was 25.0%, while the unadjusted odds of microalbuminuria were approximately eight times higher among obese participants. Qin et al. (2021)[10], in a large Chinese population, found that obesity was independently associated with elevated UACR and that individuals with combined general and central obesity had the greatest risk. Zhu et al. (2020)[11], analysing approximately 400,000 UK Biobank participants, similarly reported that both general and central adiposity were associated with albuminuria, with waist-based measurements providing important information beyond BMI.

Mean urinary albumin concentration was significantly higher in the obese group, whereas urinary creatinine concentrations were comparable. This observation strengthened the interpretation that the increased UACR primarily reflected increased albumin excretion rather than dilutional differences or lower urinary creatinine. The mean UACR among participants who

already had microalbuminuria was numerically higher in obese than normal-weight adults, but the difference was not statistically significant. This null finding was likely influenced by the very small number of normal-weight participants with microalbuminuria and the resulting wide confidence interval; it should not be interpreted as evidence that obesity had no influence on albuminuria severity.

Repeat testing confirmed microalbuminuria in 27.5% of obese participants compared with 2.5% of normal-weight participants, producing an OR of 14.79. Confirmation was important because exercise, fever, urinary infection, hydration status and other transient factors can temporarily increase urinary albumin excretion. The KDIGO CKD Work Group (2024)[12] recommends confirmation of an elevated random UACR using a subsequent first-morning urine specimen and cautions that a single abnormal measurement does not establish chronic kidney disease. Therefore, the confirmed prevalence of 15.0% in the total sample may provide a more specific estimate of persistent low-grade albuminuria than the initial prevalence of 17.5%.

The wide confidence interval around the confirmed OR (1.82-120.03) reflected sparse events, particularly the single confirmed case in the normal-weight group. Although the direction and magnitude of the association strongly suggested excess risk among obese adults, its exact size remained imprecise. Minoo et al. (2015)[13] reported that increasing severity of obesity did not consistently predict microalbuminuria, suggesting that obesity duration, visceral fat distribution, metabolic health and genetic susceptibility may be as important as BMI category alone. Consequently, UACR screening should be considered in conjunction with central-adiposity and metabolic measurements rather than BMI alone.

Relationship of UACR with anthropometric and metabolic parameters

UACR demonstrated moderate positive correlations with BMI, waist circumference, waist-to-hip ratio and waist-to-height ratio. Waist circumference had the strongest association (r=0.57), followed by BMI (r=0.52) and waist-to-height ratio (r=0.49). These findings suggested that visceral or central adiposity may be particularly relevant to early renal injury. Seo et al. (2016)[4] found a significant relationship between abdominal obesity and microalbuminuria among adults without diabetes or hypertension. Zhu et al. (2020)[11] also reported that central adiposity measures were strongly associated with albuminuria, even after consideration of general adiposity. Ren et al. (2016)[14] similarly observed associations between obesity indices and urinary albumin excretion in a middle-aged and older Chinese population.

The relationship between UACR and systolic blood pressure was weak but significant, whereas its relationship with diastolic pressure was not significant. All participants were normotensive; therefore, variations within the normal systolic range might still reflect early vascular stiffness, sympathetic activation or glomerular haemodynamic changes associated with obesity. Wang et al. (2022)[15] reported that even high-normal UACR was associated with adverse cardiometabolic outcomes and proposed that low-grade albumin leakage may represent generalized microvascular endothelial injury. The present relationship with systolic pressure supports this possibility, although causality cannot be inferred.

UACR showed significant positive correlations with fasting glucose and HbA1c despite exclusion of diabetes. This finding may indicate that insulin resistance and glycaemic variation within the non-diabetic range contribute to glomerular endothelial stress. Bartz et al. (2015)[16] found that UACR was associated with early endothelial dysfunction independently of glycaemia among youth, supporting the interpretation of albuminuria as a systemic vascular marker. Nevertheless, insulin concentration or HOMA-IR was not included in the present tables, so insulin resistance could not be assessed directly.

Adverse lipid parameters were also associated with higher UACR. Total cholesterol, triglycerides and LDL cholesterol showed positive correlations, while HDL cholesterol had an inverse correlation. Qin et al. (2021)[10] reported that elevated UACR clustered with obesity and adverse metabolic characteristics. Martin-del-Campo et al. (2019)[17] found that kidney abnormalities among overweight and obese participants coexisted with hypertriglyceridaemia, low HDL cholesterol, hyperinsulinaemia and abdominal obesity. Dyslipidaemia may promote renal injury through endothelial dysfunction, lipid deposition, oxidative stress and activation of inflammatory pathways.

The positive correlation between UACR and hs-CRP (r=0.46; $p<0.001$) further supported a link between systemic inflammation and albumin leakage. Obesity-related adipose dysfunction increases the release of interleukin-6, tumour necrosis factor-α, leptin and other inflammatory mediators, which may impair glomerular endothelial cells and podocytes. Blüher (2019)[18] described chronic low-grade inflammation and adipose-tissue dysfunction as major mechanisms through which obesity produces metabolic and vascular complications. The inverse relationship between UACR and eGFR (r=−0.32) suggested that increasing albumin excretion was accompanied by less favourable renal filtration, even though overall renal function remained preserved.

Predictors of microalbuminuria

In regression analysis, obesity, BMI, waist circumference and hs-CRP remained statistically significant predictors of microalbuminuria after adjustment. Obese adults had nearly five times higher adjusted odds of microalbuminuria than non-obese adults. Each 5 kg/m² increase in BMI was associated with a 2.36-fold increase in odds, while each 10-cm increase in waist circumference was associated with a 2.21-fold increase. These results agreed with Qin et al. (2021)[10] and Zhu et al. (2020)[11], who demonstrated independent associations of general and central obesity with elevated UACR. The significance of waist circumference reinforced the potential importance of visceral adiposity beyond overall body mass.

Hs-CRP remained independently associated with microalbuminuria, with a 49% increase in adjusted odds per 1 mg/L increment. This association provided epidemiological support for inflammation as one pathway linking obesity and glomerular injury. Martínez-Montoro et al. (2022)[9] described inflammatory cytokines, adipokine imbalance and oxidative stress as contributors to podocyte dysfunction, albuminuria and progressive obesity-related glomerulopathy. However, residual confounding by smoking, diet, physical activity, insulin resistance or undetected inflammatory conditions remained possible.

Waist-to-hip ratio, triglycerides, HDL cholesterol and declining eGFR were associated with microalbuminuria in unadjusted analyses but lost significance after adjustment. This attenuation suggested that their effects overlapped with overall obesity, central adiposity and systemic inflammation. Age and sex were not significant predictors, indicating that the observed albuminuria differences were more closely related to adiposity and metabolic status than to demographic characteristics in this relatively young sample.

CONCLUSION

The present study demonstrated that normotensive, non-diabetic adults with obesity had significantly greater urinary albumin excretion and a higher prevalence of microalbuminuria than normal-weight adults. Microalbuminuria was identified in 30.0% of obese participants compared with 5.0% of normal-weight participants, and obesity was associated with approximately eightfold higher unadjusted odds of microalbuminuria. Obese participants also exhibited higher serum creatinine, hs-CRP and renal hyperfiltration, along with a modestly lower mean eGFR, despite largely preserved conventional renal function.

UACR showed significant positive correlations with BMI, waist circumference, waist-to-hip ratio, waist-to-height ratio, triglycerides, LDL cholesterol, fasting glucose, HbA1c and hs-CRP. It was inversely related to HDL cholesterol and eGFR. Waist circumference demonstrated the strongest anthropometric correlation with UACR, highlighting the potential importance of central adiposity in early obesity-associated renal injury. In adjusted analyses, obesity, increasing BMI, waist circumference and hs-CRP remained significant predictors of microalbuminuria.

These findings suggest that subclinical renal and vascular abnormalities may develop in adults with obesity before the appearance of overt hypertension, diabetes or clinically evident renal impairment. UACR may therefore serve as a simple, non-invasive and relatively inexpensive marker for identifying obese individuals at increased risk of early glomerular and endothelial injury. Nevertheless, microalbuminuria should be interpreted as a surrogate marker rather than definitive evidence of endothelial dysfunction or obesity-induced nephropathy. Periodic UACR assessment, combined with anthropometric, metabolic and renal evaluation, may facilitate early risk identification and timely lifestyle intervention. Larger prospective studies are required to establish causality and determine whether weight reduction and metabolic improvement can reverse albuminuria and prevent long-term kidney disease.

Limitations of the Study

1. The cross-sectional design established associations but could not determine temporality or prove that obesity caused microalbuminuria or renal dysfunction.
2. The study included only 80 participants from a single centre, limiting statistical power and the generalizability of the findings.
3. Only 14 participants had microalbuminuria. Consequently, the multivariable estimates had wide confidence intervals and were vulnerable to small-sample bias and model overfitting.
4. Participants were recruited through consecutive hospital-based sampling rather than population-based random sampling, introducing the possibility of selection bias.
5. UACR is biologically variable and may be temporarily influenced by hydration, exercise, dietary intake, fever, posture and other physiological factors. Although repeat testing was performed when feasible, persistent albuminuria may not have been confirmed in every participant.
6. Microalbuminuria was used as a surrogate marker of endothelial dysfunction. Direct assessments, such as brachial artery flow-mediated dilatation, pulse-wave velocity or circulating endothelial biomarkers, were not performed.

7. Obesity-related nephropathy was inferred from anthropometric and biochemical findings without renal imaging, measured GFR or histopathological confirmation.
8. Renal function was assessed using serum creatinine-based eGFR, which may be affected by muscle mass and body composition and may be less accurate in individuals with obesity.
9. BMI does not distinguish fat mass from lean mass or accurately represent visceral adiposity. Although waist-based indices were included, direct body-composition measurements such as dual-energy X-ray absorptiometry were not performed.
10. Potential confounders such as dietary protein and salt intake, socioeconomic status, sleep apnoea, physical activity, smoking intensity, obesity duration, insulin resistance and genetic susceptibility were not comprehensively evaluated.
11. The study did not measure fasting insulin, HOMA-IR, adipokines, oxidative-stress markers or inflammatory cytokines that could clarify the mechanisms connecting obesity with albuminuria.
12. The inclusion of related anthropometric variables, such as obesity status, BMI and waist circumference, could produce multicollinearity. These predictors should be assessed in separate parsimonious regression models.
13. Follow-up was not conducted; therefore, progression or regression of albuminuria, changes in eGFR and the effects of weight reduction could not be evaluated.

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