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The Relationship Between High Body Mass Index and Microalbuminuria: A Cross Sectional Study

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ABSTRACT

Background: Obesity is a major health concern that contributes to comorbid conditions such as hypertension, diabetes, and cardiovascular disease, and it can accelerate the progression of end-stage renal disease. This study aimed to evaluate the relationship between body mass index (BMI) and waist-to-hip ratio (WHR) with microalbuminuria, a marker of renal microvascular damage.

Methods: A cross-sectional study was conducted on consecutive normotensive, non-diabetic adults. Participants were divided into two groups: a control group of 50 adults with a BMI of 18–24.9 kg/m² and an overweight/obese group of 100 adults with a BMI \geq 25 kg/m². Microalbuminuria was defined as a urinary albumin-to-creatinine ratio (UACR) exceeding 30 mg/g of creatinine.

Results: Microalbuminuria prevalence was significantly linked to higher WHR, particularly in males ($p < 0.0001$) and to a lesser extent in females ($p = 0.0224$). BMI showed significant correlations with urinary albumin ($p = 0.0084$) and creatinine ($p < 0.0001$), and was associated with elevated total cholesterol and triglycerides ($p < 0.001$).

Conclusion: Individuals with higher BMI and WHR are more likely to exhibit elevated UACR, indicating a greater risk of renal microvascular dysfunction.

Key words: Obesity; Microalbuminuria; Body mass index; Waist-to-hip ratio



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INTRODUCTION

Obesity and overweight are characterized by excessive fat accumulation that poses significant health risks, including hypertension, diabetes mellitus, cardiovascular disease, and chronic kidney disease (CKD) [1, 2].

Body mass index (BMI), calculated as weight in kilograms divided by the square of height in meters, is the most commonly used measure to classify individuals as overweight (BMI \geq 25) or obese (BMI \geq 30) [3]. Waist-to-hip ratio (WHR) complements BMI by indicating central adiposity, a key marker of metabolic risk [4].

According to the World Health Organization (WHO), over 1.9 billion adults globally are overweight, and more than 650 million are obese. Projections suggest that by 2030, more than 57% of the global population will fall into these categories [5]. In Iraq, the 2015 Noncommunicable Diseases Risk Factors Survey (STEPS) revealed that 33.5% of adults were obese, with notably higher rates among women. This public health concern is exacerbated by ongoing systemic limitations in Iraq's healthcare infrastructure, which has struggled since 2003 due to the absence of public insurance, limited treatment options, and a reliance on centralized governance, despite increased government spending [6, 7].

Metabolic syndrome, a cluster of conditions including atherosclerotic dyslipidemia, elevated blood pressure, insulin resistance, and central obesity, is closely associated with type 2 diabetes and cardiovascular disease. Between 1998 and 2009, various organizations, including the WHO, proposed multiple definitions and diagnostic criteria for this syndrome [8].

While BMI and WHR are widely used to assess obesity, it is increasingly recognized that obesity is a heterogeneous condition. Not all obese individuals develop metabolic complications; however, central obesity is particularly linked to insulin resistance, dyslipidemia, and hypertension hallmarks of metabolic syndrome [9, 10].

Obesity driven metabolic dysfunction significantly contributes to chronic kidney disease (CKD) by promoting oxidative stress, systemic inflammation, and adipokine imbalance [11, 12]. Elevated BMI and WHR have been associated with proteinuria and reduced glomerular filtration rate (GFR), indicating progressive renal damage [13, 14]. Nevertheless, the pathophysiological link between obesity and CKD remains complex, and a subset of obese individuals termed "metabolically healthy obese" may not exhibit overt renal impairment [15, 16].

Microalbuminuria, defined as a urinary albumin-to-

creatinine ratio (UACR) of 30–300 mg/g, is a sensitive early indicator of renal microvascular injury and a predictor of both CKD and cardiovascular events [17]. As routine dipstick tests often fail to detect this subtle proteinuria, targeted screening is essential [18]. Although the relationship between obesity and CKD is well documented, limited research has explored the specific association between BMI, WHR, and microalbuminuria in normotensive, non-diabetic adults, particularly within the Iraqi population, where obesity rates are rising [19].

This study aims to assess the relationship between BMI and WHR and the presence of microalbuminuria, measured by UACR, in normotensive, non-diabetic Iraqi adults. By identifying early markers of kidney injury linked to obesity, this work seeks to inform screening strategies and highlight the emerging burden of obesity related renal disease in Iraq.

MATERIAL AND METHODS

This descriptive cross-sectional study enrolled 150 volunteers (87 males and 63 females) aged 18 to 80 years and was conducted between August and November 2023 at the outpatient clinic of Kirkuk Teaching Hospital in Kirkuk, Iraq. Among these, 100 participants had a BMI of 25 kg/m² or higher and were classified as overweight or obese, while 50 participants with BMI between 18 and 24.9 kg/m² served as controls. Volunteers were recruited consecutively during routine clinic visits and were screened based on specific inclusion and exclusion criteria. Eligible participants were required to be normotensive and non-diabetic adults aged between 18 and 80 years, with BMI either in the overweight and obese range (\geq 25 kg/m²) or within the normal range (18–24.9 kg/m²). Participants were excluded if they had a history of diabetes mellitus, hypertension, hyperlipidemia, smoking, personal or family history of cardiovascular disease, any renal or liver conditions, pregnancy, urinary tract infections, or if female participants were in the menstrual phase of their cycle.

After obtaining informed verbal consent, participants completed a standardized questionnaire designed for individuals with high BMI, including personal and medical history. Anthropometric measurements were taken: standing height was measured with a stadiometer, and weight was measured using a balanced scale to calculate BMI (weight in kg divided by height in m²). Waist and hip circumferences were measured using a non-stretchable measuring tape, and WHR was calculated as the ratio of waist circumference (cm) to hip circumference (cm), measured at the level of the bony prominence of the pelvis. Individuals were classified as obese if their

BMI was 30 kg/m² or more and overweight if their BMI was between 25 and 29.9 kg/m².

Blood pressure was measured using a mercury manometer, and participants with readings above 140/90 mm Hg were excluded. For biochemical analysis, five milliliters of spot urine were collected twice over a three-month period in sterile containers to determine urinary albumin and creatinine concentrations. Additionally, fasting blood samples were drawn after a 10- to 12-hour overnight fast to assess serum creatinine, fasting blood glucose, blood lipids, random blood glucose, and HbA1c levels. All biochemical analyses were performed in a central laboratory using the Smart 150 fully automated biochemistry analyzer. Urinary albumin-to-creatinine ratio (UACR) was calculated as mg of albumin per g of creatinine, and microalbuminuria was defined as UACR levels between 30 and 300 mg/g in at least two samples. The extent of cardiac disease was determined based on participants' prior medical histories.

All participants were assured of the confidentiality of their data and informed that the information would be used solely for research purposes. Ethical approval was granted by the Board of Health in the Arab World, and the study adhered to the principles of the Declaration of Helsinki.

Data analysis was conducted using SPSS software (version 25; IBM, USA). The Shapiro-Wilk test revealed that the quantitative data were not normally distributed and were thus presented as medians and ranges. Comparisons between two groups were made using the Mann-Whitney U test, while comparisons among three groups were performed using the Kruskal-Wallis test. Categorical variables were analyzed using the chi-square test and presented as frequencies and percentages. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 150 participants (87 males and 63 females) aged 18 to 80 years were included in this descriptive cross-sectional study, conducted from August to November 2023 at the Kirkuk Teaching Hospital outpatient clinic. Of these, 100 participants had BMI values of ≥ 25 kg/m² and were classified as overweight or obese, while 50 participants with BMI between 18 and 24.9 kg/m² served as the control group. The overall average age was 39.84 years, and the mean BMI was 30.31 kg/m². Table 1 presents the distribution of participants by age and gender, along with the prevalence of microalbuminuria (UACR >30 mg/g). The largest representation for both genders was in the 25–39 age group, particularly for males. The small-

est representation was in the 65–80 age group. The analysis showed no significant difference in microalbuminuria prevalence across age groups ($p = 0.800$).

Table 2 summarizes the distribution of BMI across age categories. Among participants with BMI ≥ 25 kg/m², 15.98% were overweight (BMI 25–30 kg/m²), 17.32% were obese (BMI 31–35 kg/m²), and 33.31% were severely obese (BMI >35 kg/m²). Notably, the 40–49 age group exhibited a noticeable rise in obesity prevalence, particularly in the BMI >35 kg/m² category. Despite this trend, no statistically significant association was observed between age group and BMI distribution ($p = 0.296$).

Table 3 highlights microalbuminuria prevalence in relation to waist-to-hip ratio (WHR). A significant association was found between WHR and UACR levels, with a stronger correlation in males ($p < 0.0001$) than in females ($p = 0.0224$). Men with WHR >1 cm and women with WHR >0.85 cm demonstrated the highest frequencies of microalbuminuria.

Table 4 illustrates the relationship between BMI categories and renal function markers (urinary albumin, urinary creatinine) and lipid profiles (total cholesterol, triglycerides). Both total cholesterol and triglyceride levels increased with BMI, with the highest levels in participants with BMI >35 kg/m². This relationship was statistically significant ($p < 0.0001$ for total cholesterol and $p = 0.0007$ for triglycerides). Similarly, urinary albumin and urinary creatinine levels showed significant increases with BMI ($p = 0.0084$ and $p = 0.0001$, respectively), indicating a strong correlation between BMI and renal dysfunction. UACR levels demonstrated a clear upward trend across BMI categories, as depicted in Figure 1. The asterisks (***) indicate that the differences in UACR levels between BMI categories were statistically highly significant ($p < 0.001$). This trend suggests that higher BMI is associated with increased microalbuminuria.

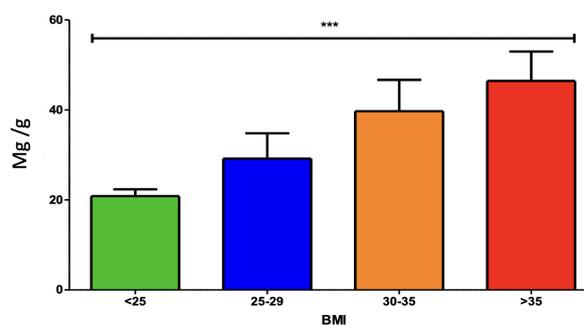


Figure 1. The distribution of microalbuminuria across BMI categories.

Overall, these results suggest that elevated BMI and WHR are

Table 1. Distribution of Microalbuminuria Across Age Groups and Gender

| Age Group (years) | UACR ≤30 mg/g, Male | | UACR ≤30 mg/g, Female | | UACR >30 mg/g, Male | | UACR >30 mg/g, Female | | P-value |
|-------------------|---------------------|--------------|-----------------------|--------------|---------------------|--------------|-----------------------|--------------|---------|
| | No | % | No | % | No | % | No | % | |
| 18–24 | 2 | 1.33 | 2 | 1.33 | 2 | 1.33 | 3 | 2.00 | 0.800 |
| 25–39 | 29 | 19.33 | 14 | 9.33 | 18 | 12.00 | 13 | 8.66 | |
| 40–49 | 15 | 10.00 | 6 | 4.00 | 6 | 4.00 | 9 | 6.00 | |
| 50–64 | 12 | 8.00 | 5 | 3.33 | 1 | 0.66 | 10 | 6.66 | |
| 65–80 | 0 | 0.00 | 1 | 0.66 | 2 | 1.33 | 0 | 0.00 | |
| Total | 58 | 38.66 | 28 | 18.65 | 29 | 19.32 | 35 | 23.32 | |

UACR = Urinary Albumin-to-Creatinine Ratio; No = number of participants.

Table 2. Distribution of BMI Across Age Groups

| Age Group (years) | BMI 18–24.9 kg/m ² | | BMI 25–30 kg/m ² | | BMI 31–35 kg/m ² | | BMI >35 kg/m ² | | P-value |
|-------------------|-------------------------------|--------------|-----------------------------|--------------|-----------------------------|--------------|---------------------------|--------------|---------|
| | No | % | No | % | No | % | No | % | |
| 18–24 | 0 | 0.00 | 2 | 1.33 | 4 | 2.66 | 4 | 2.66 | 0.296 |
| 25–39 | 30 | 20.00 | 10 | 6.66 | 9 | 6.00 | 22 | 14.66 | |
| 40–49 | 10 | 6.66 | 7 | 4.66 | 5 | 3.33 | 14 | 9.33 | |
| 50–64 | 10 | 6.66 | 5 | 3.33 | 6 | 4.00 | 8 | 5.33 | |
| 65–80 | 0 | 0.00 | 0 | 0.00 | 2 | 1.33 | 2 | 1.33 | |
| Total | 50 | 33.32 | 24 | 15.98 | 26 | 17.32 | 50 | 33.31 | |

BMI = Body Mass Index; No = number of participants.

Table 3. UACR and Statistical Comparison Across Waist-Hip Ratio Categories

| W/H Ratio Category | Females | | Males | |
|--|------------------|---------------|------------------|-------------------|
| | UACR (Mean ± SD) | P-value | UACR (Mean ± SD) | P-value |
| Low (<0.95 cm males, <0.80 cm females) | 23.4 ± 14.0 | | 24.8 ± 35.8 | |
| Moderate (0.95–1 cm males, 0.80–0.85 cm females) | 35.1 ± 30.0 | 0.0224 | 29.3 ± 31.8 | <0.0001 |
| High (>1 cm males, >0.85 cm females) | 50.4 ± 41.8 | | 33.3 ± 12.7 | |

UACR = Urinary Albumin-to-Creatinine Ratio; SD = Standard Deviation; W/H = Waist-Hip Ratio.

Table 4. Relation between BMI with Urinary Creatinine, Urinary Albumin, and Lipid Profiles (Total Cholesterol and Triglycerides)

| Parameter | 18–24.9 kg/m ² | 25–30 kg/m ² | 31–35 kg/m ² | >35 kg/m ² | P-value |
|----------------------------|---------------------------|-------------------------|-------------------------|-----------------------|-------------------|
| | Mean ± SD | Mean ± SD | Mean ± SD | Mean ± SD | |
| Total Cholesterol (mg/dl) | 121.5 ± 32.52 | 186.9 ± 40.52 | 182.4 ± 42.86 | 186.8 ± 43.90 | <0.0001 |
| Triglyceride (mg/dl) | 129.9 ± 37.16 | 161.0 ± 67.27 | 173.8 ± 76.09 | 178.6 ± 72.17 | 0.0007 |
| Urinary Albumin (mg/dl) | 3.5 ± 1.67 | 5.0 ± 10.09 | 7.2 ± 11.06 | 4.9 ± 2.78 | 0.0084 |
| Urinary Creatinine (mg/dl) | 168.5 ± 54.00 | 139.8 ± 68.99 | 130.1 ± 37.74 | 129.2 ± 37.55 | 0.0001 |
| UACR (mg/gm) | 20.87 ± 10.77 | 29.19 ± 28.21 | 39.72 ± 35.56 | 46.47 ± 44.70 | <0.0001 |

UACR = Urinary Albumin-to-Creatinine Ratio; SD = Standard Deviation; BMI = Body Mass Index.

significantly associated with higher UACR levels and dyslipidemia, particularly in males. However, the lack of statistical significance between age groups and BMI suggests that while obesity prevalence rises with age, it is not evenly distributed.

DISCUSSION

Obesity has emerged as a significant public health challenge, intricately linked to various comorbid conditions such as diabetes mellitus, hypertension, cardiovascular diseases, and renal dysfunction [20, 21].

One of the earliest and most subtle manifestations of kidney injury is microalbuminuria, reflected by an elevated urinary albumin-to-creatinine ratio (UACR) [22]. Early detection of microalbuminuria is crucial because it allows for timely interventions that can slow the progression of chronic kidney disease (CKD) [23]. However, the relationship between obesity and microalbuminuria remains complex and multifactorial, influenced by factors such as fat distribution, metabolic activity, and inflammatory responses [24].

The present study found a significant association between high BMI and increased UACR, indicating early renal microvascular dysfunction in overweight and obese individuals compared to those with normal BMI. A similarly strong association was observed with waist-to-hip ratio (WHR), especially in males. These findings align with those reported by Wang Y et al. [25], who identified a positive correlation between BMI and microalbuminuria, supporting the hypothesis that obesity contributes to renal microvascular injury. However, our results differ from Nguyen et al. [26], who found no significant association between BMI and microalbuminuria. This discrepancy may stem from differences in study populations, with Nguyen's study focusing on patients with established hypertension and diabetes, whereas our study targeted normotensive, non-diabetic adults.

Comparing with other studies, Zimmermann et al. [27] and Memarian et al. [28] demonstrated a stronger correlation between waist circumference or WHR and CKD progression, reinforcing the notion that central obesity may have a more pronounced effect on renal health than general obesity as measured by BMI. These findings are comparable to ours, which emphasized the role of WHR alongside BMI. However, some studies, such as those by Braga et al. [29], failed to find such correlations, likely due to methodological differences, such as relying on raw albumin concentration rather than UACR, confounders like antihypertensive use, and limited statistical power. In contrast, our study controlled for these variables and used UACR, minimizing biases.

Several limitations of this study should be acknowledged. First, the cross-sectional design precludes any causal inference between high BMI, WHR, and microalbuminuria. Second, the sample size was relatively small and not evenly distributed across age groups, which may limit the generalizability of the results. Third, key factors such as dietary habits, physical activity, socioeconomic status, and hormonal influences were not assessed, all of which could modulate the relationship between obesity and microalbuminuria. Fourth, while the study focused on normotensive, non-diabetic adults, it did not control for other potential confounders such as smoking history or underlying subclinical diseases.

Given these considerations, future research should prioritize longitudinal, multicenter studies with larger and more diverse populations to validate and expand upon these findings. Incorporating assessments of dietary intake, physical activity levels, and comprehensive metabolic profiles—including insulin resistance and inflammatory markers—will help clarify the mechanisms underlying the observed associations.

Furthermore, future studies should explore the utility of alternative obesity indices, such as visceral fat measurement via imaging, alongside genetic and environmental factors influencing renal health. Ultimately, this will provide a more nuanced understanding of how obesity contributes to early kidney damage and inform targeted strategies for prevention and intervention.

CONCLUSION

This study demonstrates a clear association between elevated BMI and increased UACR, highlighting early signs of kidney stress linked to obesity. The correlation between W/H ratio and UACR further underscores the role of fat distribution in renal health. Although gender differences in UACR were not significant across age groups, the rising trend of obesity with age points to the importance of early intervention, especially in younger populations. These findings support the urgent need for targeted public health strategies in Iraq and beyond to manage obesity and reduce the risk of kidney and metabolic diseases.

ETHICAL DECLARATIONS

• Ethics Approval and Consent to Participate

All subjects gave informed consent for inclusion before participating in the study. The study was approved by the Ethics Committee of the Scientific Council of the Board of

Health in the Arab World (Internal Medicine, Document no. 1752, dated 16–9–2024).

• Consent for Publication

Non.

• Availability of Data and Material

The datasets are available from the corresponding author upon reasonable request.

• Competing Interests

The authors declare that there is no conflict of interest.

• Funding

Self funded.

• Use of Generative Artificial Intelligence

The authors declare that ChatGPT was used only to improve clarity and grammar during final editing, not for content generation, analysis, or interpretation.

• Authors' Contributions

All authors contributed significantly, directly, and intellectually to the work and consented to its publication.

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