fortune Journals

Research Article

ARCHIVES OF NEPHROLOGY AND UROLOGY

ISSN: 2644-2833

Association of Adiponectin with Renal Involvement in Sedentary Obese Individuals

Abdur Rahaman[1*], Omar faroque[2], Kabir Hossain[3], Ferdous Jahan[3], Nourin sultana[1], Bedar Uddin[4], Muhammad Nazrul Islam[5]

Abstract

Background: The global prevalence of chronic kidney disease (CKD) among obese individuals is on the rise, accompanied by a growing economic burden associated with its treatment. Therefore, early detection of renal dysfunction is crucial to prevent CKD progression. Sedentary obesity is a significant risk factor for early renal impairment, often marked by microalbuminuria. Adiponectin, an anti-inflammatory adipokine, serves a protective role in mitigating metabolic and renal disorders. Adiponectin is typically reduced in obese individuals. The study was aimed to evaluate the association of serum adiponectin and renal involvement in sedentary obese individuals without diabetes, hypertension & hypothyroidism.

Methods: This observational cross-sectional study was carried out in the Department of Nephrology at BSMMU, Dhaka. The study period ranged from February 2023 to August 2024. The study included 67 sedentary obese individuals, selected according to specific inclusion and exclusion criteria. A purposive sampling approach was employed, and data were gathered using a pre-tested questionnaire, which incorporated patient history and clinical examination. Serum adiponectin levels were measured using the DRG Adiponectin ELISA Kit, which operates on the sandwich principle. Renal involvement was evaluated by determining the urinary albumin-to-creatinine ratio (uACR) and estimating the glomerular filtration rate (eGFR) using the MDRD equation. Correlation and ROC curve analyses were conducted to investigate the association between adiponectin levels and microalbuminuria.

Results: The study included participants with an average age of 35.88 ± 8.34 years, predominantly male (55.2%), with a significant portion being students (34.3%). Most participants exhibited obesity, with a mean BMI of 34.84 ± 3.47 kg/m², and more than 95% had central obesity. Laboratory results indicated normal glycemic control, with a mean fasting blood sugar of 4.93 ± 0.31 mmol/L and HbA1c of 5.06 ± 0.27%. However, 86.6% of participants had low HDL levels, and a significant number had elevated triglycerides and LDL levels. At enrollment, 8 participants (11.9%) had elevated urinary ACR. After excluding cases of transient microalbuminuria, 7 participants (10.4%) were confirmed to have microalbuminuria, resulting in an overall renal involvement rate of 10.4%. Participants with microalbuminuria had significantly lower serum adiponectin levels (0.93 ± 0.35 µg/ml, $p<0.001$). A weak inverse correlation was found between BMI and serum adiponectin ($r = -0.295$, $p = 0.013$). Adiponectin was found to be a strong predictor of microalbuminuria with a cut-off value of ≤1.0 µg/ml, exhibiting good sensitivity and specificity.

Affiliation:

[1]Resident of Nephrology (Phase-B), Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

[2]Associate professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

[3]Assistant Professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

[4]Medical officer, 250 Bedded General Hospital, Jashore, Bangladesh

[5]Professor, Department of Nephrology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh

***Corresponding author:**
Abdur Rahaman, Resident of Nephrology (Phase-B), Department of Nephrology, Bangabandhu Sheikh Mujib Medical University, (BSMMU), Dhaka, Bangladesh.

Citation: Abdur Rahaman, Omar faroque, Kabir Hossain, Ferdous Jahan, Nourin sultana, Bedar Uddin, Muhammad Nazrul Islam.Association of Adiponectin with Renal Involvement in Sedentary Obese Individuals. Archives of Nephrology and Urology. 8 (2025): 18-27.

Received: December 16, 2024
Accepted: January 06, 2025
Published: January 29, 2025

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

Conclusion: Reduced adiponectin levels were significantly associated with microalbuminuria, underscoring its potential as a biomarker for early renal involvement in sedentary obese individuals.

Keywords: Chronic Kidney Disease; Urinary Albumin-to-Creatinine Ratio; Glomerular Filtration Rate

Introduction

Sedentary obesity, defined by a combination of excessive body fat and insufficient physical activity, plays a substantial role in the global prevalence of metabolic and cardiovascular disorders. Adiponectin, an adipokine produced by adipose tissue, is essential for regulating metabolic processes such as glucose balance, lipid metabolism, and anti-inflammatory mechanisms. Unlike most adipokines, adiponectin levels are notably lower in individuals with obesity, especially those with a sedentary lifestyle. This decline is linked to a heightened risk of insulin resistance, dyslipidemia, and chronic kidney disease (CKD). Growing evidence indicates that reduced adiponectin levels could be associated with early kidney involvement, manifesting as microalbuminuria.

Obesity triggers chronic low-grade systemic inflammation, which in turn drives systemic metabolic dysfunction and is closely associated with the development of renal disease [1]. Albuminuria is viewed as a consequence of widespread endothelial dysfunction, which is intensified by an inflammatory state. Therefore, it is reasonable to propose that the persistent inflammation commonly observed in people affected by obesity contributes to the advancement of microalbuminuria [2].

Obesity is recognized as a potential risk factor for microalbuminuria, particularly in individuals with hypertension and diabetes. It could also contribute to proteinuria in individuals with CKD. Some researchers propose that obesity can negatively impact kidney health even in those without a prior diagnosis of hypertension, diabetes, or existing renal conditions [3].

In individuals with obesity, there is an elevation in the levels of various inflammatory markers and cytokines, including C-reactive protein (CRP), tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and macrophage migration inhibitory factor (MIF). Conversely, the levels of adiponectin, a protein hormone known for its anti-inflammatory properties, are reduced [4]. Adiponectin is a peptide hormone produced by adipocytes, known for its anti-inflammatory, anti-diabetic, and anti-atherogenic effects [5]. Reduced serum adiponectin levels have been linked to obesity, insulin resistance, coronary heart disease, and metabolic syndrome in the in the general population [6].

Hypoadiponectinemia plays a significant role in the onset of a persistent, low-grade systemic inflammatory condition. Its proinflammatory properties suggest that hypoadiponectinemia could be a key factor driving the systemic and vascular inflammation commonly associated with obesity and related disorders, such as renal damage [7].

Elevated albumin excretion rates (AER), commonly seen in individuals with obesity, are associated with an increased risk of cardiovascular morbidity and mortality, as well as the early onset of renal dysfunction. Overall, the prevalence of microalbuminuria in the general population rises with total and central obesity, mostly impacting nondiabetic and non-hypertensive people [8]. In adolescents with moderate obesity, the prevalence of proteinuria is reported to be 2.4%. In those with severe obesity, 3% exhibit proteinuria, 14% show microalbuminuria, and 3% have a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m² [9].

Despite the established association between obesity and albuminuria, this condition frequently remains undiagnosed, as it typically presents without clinical symptoms and is not routinely screened for, particularly in cases of low-grade albuminuria. Some evidence suggests that in cases of obesity, AER increases due to hemodynamic effects, which include heightened renal blood flow and glomerular pressure. This results in glomerular hyperfiltration and increased permeability to albumin. While abnormalities in renal hemodynamics are likely a significant factor, it is also plausible that inflammatory and metabolic mechanisms play a role. Adipose tissue is responsible for the production of many key components of the renin-angiotensin-aldosterone system (RAAS). Obese individuals typically exhibit elevated levels of angiotensin-converting enzyme activity, plasma renin activity, angiotensinogen, and circulating angiotensin II. Additionally, non-hemodynamic factors such as hyperinsulinemia, oxidative stress, and inflammation, along with changes in renal hemodynamics, can contribute to or worsen renal damage [10].

Proteinuria may result from these effects, which may also cause focal glomerulosclerosis, glomerular hypertrophy, and hyperfiltration. Visceral fat may play a role in the production of various renin-angiotensin system (RAS) proteins and other hemodynamic factors, including elevated intra-abdominal pressure or cardiopulmonary dysfunction. Additionally, it can disrupt renal blood flow by exerting compressive effects on the renal hilum and/or renal parenchyma. Moreover, perirenal fat seems to exert lipotoxic effects on the kidney by increasing glomerular hydrostatic pressure and activating the renin-angiotensin-aldosterone system (RAAS), thereby accelerating renal damage [11].

There have only been a few studies carried out in worldwide, which demonstrate the role of serum adiponectin as predictor of renal involvement in sedentary obese

individuals. With this background in mind, this study is intended to evaluate the independent association of adiponectin with renal involvement as well as investigating the role of adiponectin as predictor of renal involvement in sedentary obese individuals without diabetes, hypertension & hypothyroidism.

Methodology & Materials

A cross-sectional observational research was undertaken at the Department of Nephrology, BSMMU, Dhaka, approximately for 18 months. A total of 67 sedentary obese individuals were taken from the general population, teachers & resident doctor of BSMMU & from Obesity clinic of Endocrinology department, BSMMU. The sampling method was purposive sampling. Those who fulfilled the selection criteria (inclusion and exclusion criteria) were selected and evaluated for this study.

Inclusion Criteria:

This study included individuals with a BMI of 30 kg/m² or higher.

Exclusion Criteria:

Disorder affecting renal function (e.g. diabetes, hypertension), secondary causes of obesity (e.g. cushing's syndrome, hypothyroidism), conditions affecting adipocytokines e.g. psychiatric disorders, pregnancy, acute cardiovascular events or with history of abdominal surgery, cancer, stroke, & all individuals takings steroid, OCP, statins & beta blocker, antidepressants (amitryptyline, mirtazepine), pregabaline, gabapentine & sodium valproate & age < 18 years were not considered for enrollment in the study.

Study procedure:

Before enrollment, the objectives, purpose, and procedure of the study were thoroughly explained to the participants. The potential benefits and risks (if any) of the study were clearly outlined, with particular emphasis on how the participants would benefit from their involvement. Informed written consent was obtained in the prescribed format from them when they voluntarily consented to participate. After enrollment, a detailed medical and socioeconomic history was documented in a preformatted data sheet. A full physical examination was performed, and the results were stated.

All study participants had their body mass, height, and waist (measured at the midpoint between the lowest rib and the iliac crest) and hip (measured at the widest point over the greater trochanters) circumferences measured to calculate obesity indices as follows:

Body mass index: BMI = weight/height (kg/m2).

Waist-to-hip ratio: WHR = waist circumference (cm)/hip circumference (cm).

Blood pressure was measured using a sphygmomanometer in sitting posture after taking 5 minutes of rest, reducing their anxiety by explaining and reassuring that the sensation of the cuff tightening on their arm is safe.

With all aseptic precautions, 5 ml of venous blood were collected from the ante-cubital vein of the patient in a vacutainer tube and were brought to Kidney Research Laboratory (Department of Nephrology), Department of Laboratory Medicine and Department of Immunology and Microbiology for required tests to be done. Thereafter, the samples were Centrifuged @ 2200-2500 rpm for 15 min, and the obtained serum were kept frozen at -20°C. Different Hematological, biochemical and hormonal test (uACR, serum creatinine, S.TSH, Lipid profile, HbA1C) were done by fully automated SIEMENS (Dimension EXL with LM) machine through spectrophotometry method in Laboratory Medicine Department, BSMMU among the study population.

Serum adiponectin level was measured by using The DRG Adiponectin ELISA Kit based on the sandwich principle. Urinary ACR was measured by ELISA method. An estimated glomerular filtration rate (eGFR) was calculated for all serum creatinine values by using the Modification of Diet in Renal Disease (MDRD) formula.

Operational Definitions:

BMI:

Body mass index (BMI) is a straightforward and commonly used method for estimating body fat. It is calculated by dividing a person's weight by the square of their height, usually expressed in metric units: Weight in kg / Height in m² [12].

eGFR:

Glomerular filtration rate (GFR) is an indicator of kidney function. This test measures the creatinine level in the blood and uses the result in a formula to calculate a value known as the estimated GFR (eGFR), which reflects the efficiency of kidney function [13].

Microalbuminuria:

Microalbuminuria is characterized by an abnormal rise in albumin excretion, ranging from 30–300 mg of albumin per gram of creatinine in a spot urine sample, persisting across multiple measurements typically conducted over 3 to 6 months [13].

The American Diabetes Association, the National Institutes of Health, and the National Kidney Foundation advise measuring albumin in urine using the albumin-to-creatinine ratio method [14].

Obesity: WHO Criteria for global population [15].

Underweight: BMI < 18.5 kg/m², Normal weight: BMI

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

18.5–24.9 kg/m², Overweight: BMI 25–29.9 kg/m², Obesity Class I: BMI 30–34.9 kg/m², Obesity Class II: BMI 35–39.9 kg/m², Obesity Class III (Morbid Obesity): BMI ≥ 40 kg/ m².

Central obesity & WHR:

Central obesity refers to the accumulation of excess fat around the abdominal region and is commonly evaluated using the waist-to-hip ratio (WHR). The World Health Organization (WHO) defines central obesity as a WHR greater than 0.90 in men and greater than 0.85 in women, which is linked to a higher risk of metabolic and cardiovascular diseases [16].

Sedentary individuals:

Adults should regularly participate in physical activity. To achieve significant health benefits, they should aim for 150–300 minutes of moderate-intensity aerobic exercise, 75–150 minutes of vigorous-intensity aerobic activity, or a combination of both, spread across the week. Additionally, performing muscle-strengthening exercises targeting all major muscle groups at a moderate or higher intensity on at least two days per week provides additional health benefits. For even greater advantages, adults can increase their moderate-intensity aerobic activity beyond 300 minutes per week or engage in more than 150 minutes of vigorous-intensity activity, or a mix of the two. Individuals who do not meet these recommendations are classified as sedentary [17].

Statistical analysis:

Data were collected using a standardized data collection form. Quantitative data were summarized as mean and standard deviation, while qualitative data were presented as frequency distributions and percentages. Statistical analysis was performed using Windows-based software, specifically SPSS-27 (SPSS Inc., Chicago, IL, USA). The Mann–Whitney U test was used to compare two groups for quantitative variables that did not follow a normal distribution. The Chi-square test and Fisher's Exact test were utilized to compare categorical variables across groups. Spearman's correlation coefficient (r) was applied to evaluate the relationship between two non-normally distributed quantitative variables or between a quantitative and a qualitative variable. A p-value of less than 0.05 was considered statistically significant.

Results

The study included a total of 67 participants, with a mean age of 35.88 ± 8.34 years, with the majority (47.8%) belonging to the >30–40 years age group. Most participants were male (55.2%), and more than one-third were students (Table 1). All participants had normal blood pressure levels, with mean systolic and diastolic pressures of 117.52 ± 7.48 mmHg and 75.87 ± 5.23 mmHg, respectively. The participants had a mean BMI of 34.84 ± 3.47 kg/m² and a mean waist-to-hip ratio of 0.92 ± 0.03 (Table 2). Among the participants, 13.4% were classified as having morbid obesity, while more than 95% had central obesity (Figure 1). Additionally, 86.6% of participants exhibited low HDL levels. More than half had elevated triglyceride levels, while over one-third presented with high LDL levels. Elevated total cholesterol levels were observed in 6.0% of the participants (Figure 2). The participants had a mean ACR of 20.93 ± 29.45, ranging from 2.50 to 209.50. At baseline or enrollment, 8 participants (11.9%) were suffering from high levels of urinary ACR, while approximately 47.8% had elevated eGFR (Table 3). To exclude transient microalbuminuria, the urinary ACR levels of 8 participants with high urinary ACR at baseline were rechecked 3 months after the initial assessment. Of these, only 7 were confirmed to have persistent microalbuminuria. The 8 participants with high urinary ACR at baseline had a median (range) urinary ACR of (22.18–296.34) at 3 months. However, these 7 participants also exhibited high eGFR levels at 3 months. The mean BMI of the participants at 3 months was 36.90 ± 3.02 kg/m² (Table 4).

Overall, 7 participants (10.4%) were found to have microalbuminuria (Figure 3). Table 5 shows that the mean serum adiponectin level was 0.93 ± 0.35 µg/ml in participants with microalbuminuria and 2.03 ± 1.35 µg/ml in participants without microalbuminuria. Participants with microalbuminuria had a significantly lower mean serum adiponectin level (p-value: 0.001). Among the moderately obese participants, 5.9% developed microalbuminuria, while 12.5% of severely obese participants and 22.2% of morbidly obese participants developed microalbuminuria. A gradual increase in the proportion of participants developing microalbuminuria was observed with higher obesity categories. However, no significant association was found between the obesity category and microalbuminuria. Similarly, there was no significant association between the waist-to-hip ratio and microalbuminuria (Table 6). Furthermore, a significant weak negative correlation was identified between body mass index and serum adiponectin (Spearman correlation coefficient, r: -0.370, p-value: 0.002) (Figure 4). The ROC analysis of serum adiponectin for predicting microalbuminuria, as shown in Figure 5, yielded an AUC of 0.902 (95% CI: 0.760–1.00), indicating statistically significant predictive accuracy (p = 0.001). A cut-off value of ≤1.0 µg/ml exhibited the highest Youden index (0.82), with sensitivity of 85.71%, specificity of 96.67%, overall accuracy of 95.52%, and positive and negative predictive values of 75.00% and 98.31%, respectively (Table 7). Table 8 indicates that 6 out of 7 patients with microalbuminuria had a serum adiponectin value of ≤1.0 µg/ml.

ROC analysis for the Prediction of Microalbuminuria

ROC analysis of serum adiponectin to predict microalbuminuria showed an AUC value of 0.902 (95% CI 0.760-1.00) which was statistically significant (P value: 0.001).

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

Table 1: Distribution of the participants according to demographic characteristics (N=67)

Demographic characteristics		Frequency	Percentage
Age (years)	18-30	18	26.9
	>30-40	32	47.8
	>40-50	13	19.4
	>50	4	6
	Mean ± SD	35.88±8.34	
	Median (Range)	35.0 (18-58)	
Gender	Male	37	55.2
	Female	30	44.8
Occupation	Housewife	11	16.4
	Business	17	25.4
	Service	14	20.9
	Student	23	34.3
	Others	2	3

SD: Standard deviation, Data was presented as mean± SD, Median (range), Frequency, percentage

Table 2: Distribution of the participants according to the physical parameters (N=67)

Physical parameters		Values
Systolic blood pressure (mm of Hg)	Mean ± SD	117.52±7.48
	Median (Range)	120.0 (100.0-130.0)
Diastolic blood pressure (mm of Hg)	Mean ± SD	75.87±5.23
	Median (Range)	78.0 (70.0-90.0)
Body mass index (kg/m2)	Mean ± SD	34.84±3.47
	Median (Range)	34.60 (30.10-44.90)
Waist Hip ratio	Mean ± SD	0.92±0.03
	Median (Range)	0.93 (0.79-0.98)
Waist circumference (cm)	Mean ± SD	110.93±6.87
	Median (Range)	110.0 (96.0-142)

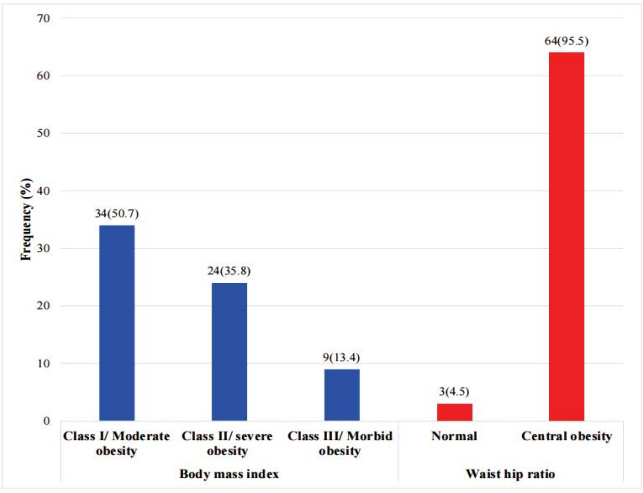

Figure 1: Distribution of body mass index, and waist-hip ratio among the study participants

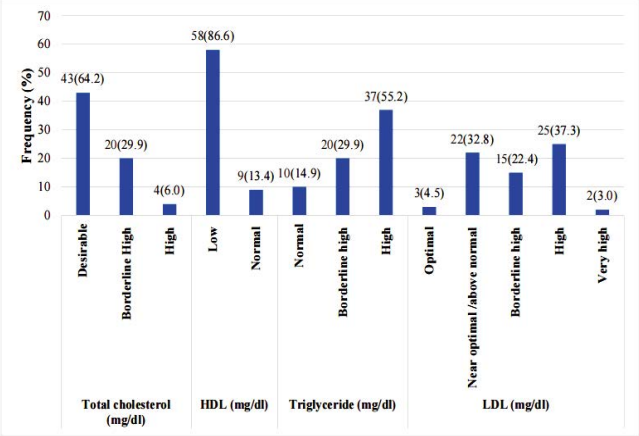

Figure 2: Distribution of the lipid profile of the study participants

Table 3: Assessment of Renal involvement at baseline (N=67)

(A)			
Parameters		**Values**	
Urinary ACR	Mean ± SD	20.93±29.45	
	Median (Range)	13.0 (2.50-209.50)	
S creatinine (mg/dL)	Mean ± SD	1.02 ±0.14	
	Median (Range)	1.04 (0.60-1.29)	
eGFR	Mean ± SD	120.48±19.54	
	Median (Range)	117.0 (92.0-181.0)	
(B)			
Parameters		**Frequency**	**Percentage**
Urinary ACR	Normal	59	88.1
	High	8	11.9
eGFR	High	32	47.8
	Normal	35	52.2

Data was presented as mean± SD, Median (range), Frequency, percentage

Table 4: Assessment of body mass index and renal involvement at 3 months (N=8)

(A)		
Parameters		**values**
Urinary ACR (n=8)	Mean ± SD	85.18±89.70
	Median (Range)	44.87 (22.18-296.34)
Serum creatinine (mg/dl) (n=8)	Mean ± SD	0.86±0.19
	Median (Range)	0.90 (0.60-1.10)
eGFR (n=8)	Mean ± SD	154.25±22.30
	Median (Range)	159.0 (118.0-183.0)
(B)		
Parameters	**Frequency**	**Percentage**
Urinary ACR (n=8)	Normal	1	12.5
	High	7	87.5
eGFR (n=8)	High	7	87.5
	Normal	1	12.5

Data was presented as mean± SD, Median (range), Frequency, percentage

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

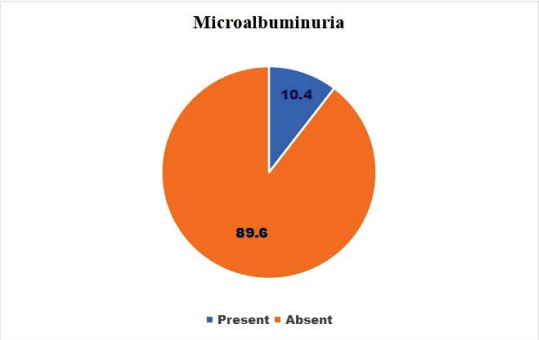

Figure 3: Distribution of microalbuminuria among the study participants

Table 5: Distribution of the serum Adiponectin among participants with or without microalbuminuria (N=67)

Parameters		Microalbuminuria		P value
		Yes	No	
		(n=7)	(n=60)	
Serum Adiponectin (µg/ml)	Mean ± SD	0.93±0.35	2.03±1.35	0.001
	Median (Range)	0.83 (0.61-1.66)	1.60 (0.41-7.0)	

Mann Whitney U test was done, Data was presented as Mean ± SD, median (range)

Table 6: Association of Body mass index with Microalbuminuria (n=67)

Variables		Microalbuminuria		P value
		Yes	No	
		(n=7)	(n=60)	
Body mass index	Class I/ Moderate obesity	2 (5.9)	32 (94.1)	[a] 0.333
	Class II/Severe obesity	3 (12.5)	21 (87.5)	
	Class III/morbid obesity	2 (22.2)	7 (77.8)	
Waist hip ratio	Normal	0 (0.0)	3 (100.0)	[b]>0.99
	Central obesity/ risk	7 (10.9)	57 (89.1)	

[a]Chi-square test and b Fisher's exact test was done, Data was presented as frequency, percentage

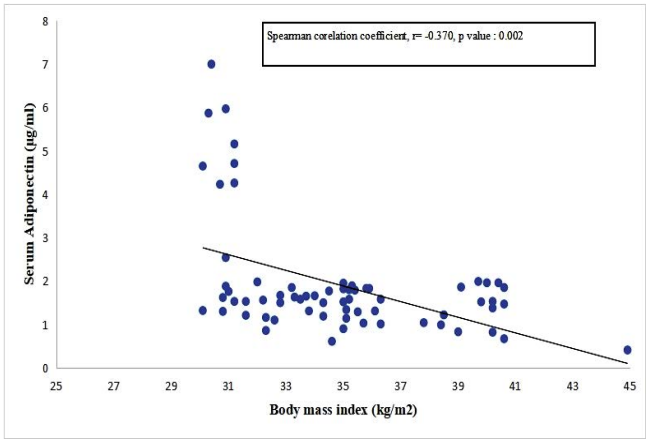

Figure 4: Correlation of body mass index with Serum Adiponectin

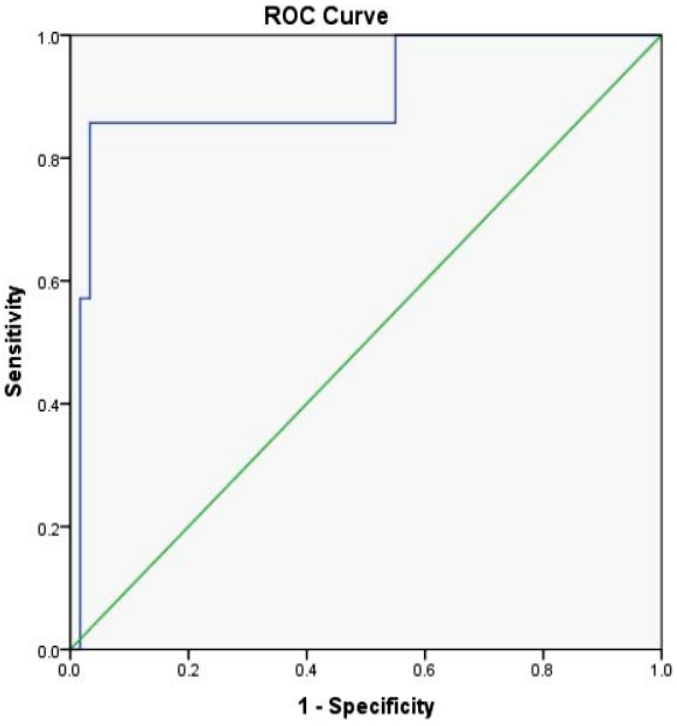

Figure 5: Receiver operator characteristics (ROC) curve of serum adiponectin to predict microalbuminuria

Cut-off points

Table 7: Determination of cut-off value with Youden index.

Cutoff value	Sensitivity	Specificity	PPV	NPV	Accuracy	Youden index
						(j=sen+spe-1)
≤0.945	0.7143	0.9667	0.7143	0.9667	0.9403	0.68
≤1.0	**0.8571**	**0.9667**	**0.75**	**0.9831**	**0.9552**	**0.82**
≤1.02	0.8571	0.95	0.6667	0.9828	0.9403	0.81

Citation: Abdur Rahaman, Omar faroque, Kabir Hossain, Ferdous Jahan, Nourin sultana, Bedar Uddin, Muhammad Nazrul Islam.Association of Adiponectin with Renal Involvement in Sedentary Obese Individuals. Archives of Nephrology and Urology. 8 (2025): 18-27.

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

Table 8: Cross-tabulation of microalbuminuria with serum adiponectin value based on derived cut-off value.

Serum adiponectin (µg/ml)	Microalbuminuria		Total
	Yes	No	
≤1.0	6	2	8 (TP+FP)
>1.0	1	58	59 (FN+TN)
Total	7 (TP+FN)	60 (FP+TN)	67 (TP+FP+FN+TN)

Discussion

This study observed the association between adiponectin and microalbuminuria, as well as evaluated the predictive value of adiponectin for the presence of microalbuminuria in sedentary obese individuals. This cross-sectional study included 67 sedentary obese individuals without diabetes, hypertension, or hypothyroidism. This study described the demographic characteristics of the participants, with an average age of 35.88 ± 8.34 years. The majority were in the 30–40 years age group (47.8%), with a male predominance (55.2%). Approximately one-third of the participants were students (34.3%), suggesting a population in early mid-life, likely pursuing postgraduate studies. This demographic distribution may indicate a higher risk of metabolic issues in males in early mid-life, potentially due to lifestyle factors such as sedentary behavior or dietary habits. Similar age and sex distributions were observed in a study conducted by de Almeida et al. [18].

Conversely, although male prevalence was observed in studies by Kawamoto et al. [19], Chen et al. [20], and Song et al. [21], the mean age in these studies was significantly higher. In contrast, studies by Mackowiak-Lewandowicz et al. [22] and El-Shaheed et al. [23] focused on a younger demographic, with participants averaging 13.06 ± 2.61 years. This difference could be attributed to variations in target sample selection, as different studies targeted distinct demographic groups. In this study, physical parameters were assessed, showing that all participants had normal systolic and diastolic blood pressure levels, with mean values of 117.52 ± 7.48 mmHg and 75.87 ± 5.23 mmHg, respectively. The mean Body Mass Index (BMI) was significantly elevated at 34.84 ± 3.47 kg/m², indicating widespread obesity among the participants. Additionally, the waist-to-hip ratio averaged 0.92 ± 0.03, further suggesting central obesity. This was confirmed by Figure 1, which showed that over 95.5% of participants exhibited central obesity, and 13.4% were classified as morbidly obese. This data underscores the significant burden of obesity-related health risks, potentially exacerbating cardiovascular and metabolic conditions. A similar BMI range (approximately 34.9 ± 7.33 to 37.5 ± 3.88 kg/m²) was reported in comparable studies [18, 23-25]. Physical parameters were also consistent with findings from other studies [21,26]. The similarity in physical parameters across studies can be attributed to the fact that individuals with comparable height, weight, and BMI tend to exhibit similar pulse and blood pressure values.

The study also presented laboratory parameters, where the mean fasting blood sugar was within normal limits at 4.93±0.31 mmol/L, and the mean blood sugar two hours after glucose intake was 6.88±0.63 mmol/L. The HbA1c levels were also normal at 5.07±0.27, indicating adequate glycemic control in this population. The median TSH level was 2.9 mIU/L, with a range of 1.15-4.62, suggesting normal thyroid function. However, concerning lipid profiles (Figure 2), 86.6% had low HDL levels, and a significant portion had elevated TGs and LDL levels, with 6.0% exhibiting high total cholesterol. These results indicate a significant prevalence of dyslipidemia, which is a key risk factor for cardiovascular disease. Low HDL and high levels of triglycerides (TG), cholesterol, and LDL were also observed in previous studies [19, 20, 18, 25, 27]. El-Shaheed et al. [23] reported that blood glucose levels were nearly similar between obese and non-obese patients.Another study found that approximately one-third of participants had an elevated albumin-to-creatinine ratio (ACR), while the remaining two-thirds had ACR levels within the normal range. Related studies also reported elevated serum creatinine in overweight or obese patients [19, 20, 25]. Studies by Kawamoto et al. [19] and Chen et al. [20] observed that about 8.78% of patients had decreased eGFR, whereas Mackowiak-Lewandowicz et al. [22] found no decrease in eGFR among their study population. Differences in renal parameters could be attributed to participants being in different categories of obesity, as various obesity categories may affect kidney function differently.

The follow-up data at three months included only those participants who were microalbuminuric at enrollment to exclude transient microalbuminuria. Of these, seven out of eight participants with initially high ACR continued to show elevated ACR (median: 22.18–296.34), while one participant became normoalbuminuric, suggesting either transient microalbuminuria or the beneficial effects of weight-loss strategies such as physical exercise and diet control. Additionally, 10.4% developed microalbuminuria, a marker of ongoing renal damage. Notably, participants with microalbuminuria had a higher mean BMI (36.95 ± 3.02 kg/m²) and higher eGFR, indicating that obesity is closely linked to renal stress and subsequent proteinuria.One study identified

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

a significant relationship between microalbuminuria and HbA1c, postprandial blood glucose, and uric acid levels, but no association with ACR, BMI, or eGFR [27]. Only Mackowiak-Lewandowicz et al. [22] found a link between microalbuminuria, eGFR, and BMI z-scores, but their study was conducted in a younger age group. Thus, limited similarities were observed concerning these variables.

The study also found that serum adiponectin levels were markedly lower in participants with microalbuminuria (0.93 ± 0.35 µg/ml) compared to those without microalbuminuria (2.03 ± 1.35 µg/ml), with a p-value of <0.001. This observation indicates that reduced adiponectin levels, commonly linked to obesity, may contribute to the development of kidney damage in these individuals. Several studies support this finding, demonstrating a strong significant relation between microalbuminuria and mean adiponectin levels [27, 23]. de Almeida et al. [18] observed that adiponectin levels measured during the basal period were significantly higher in normoinsulinemic (NI) subjects compared to hyperinsulinemic (HI) groups ($p < 0.04$). This study also revealed a significant, albeit weak, negative correlation between BMI and serum adiponectin ($r = -0.370$, $p = 0.002$), as well as between eGFR and serum adiponectin ($r = -0.303$, $p = 0.011$). These findings are supported by previous studies [19, 21, 22, 23]. These correlations suggest that obesity and altered kidney function may contribute to decreased adiponectin levels, potentially exacerbating metabolic and renal issues.

The study explored the relationship between BMI and microalbuminuria, observing that although the prevalence of microalbuminuria rose with increasing obesity severity, the association was not statistically significant. This finding suggests that factors beyond BMI may influence renal health. Similarly, the waist-to-hip ratio did not show a significant correlation with microalbuminuria. Additionally, the study determined the optimal cut-off value for adiponectin as a biomarker for predicting microalbuminuria. A serum adiponectin level of ≤1.0 µg/ml demonstrated the highest diagnostic accuracy, with a Youden index of 0.82, sensitivity of 85.71%, and specificity of 96.67%. This cut-off suggests that low adiponectin levels are strongly predictive of microalbuminuria, emphasizing the role of inflammation and adipokine imbalance in the pathophysiology of renal damage in obese individuals. These findings are unique to this study. To the best of our knowledge, no prior studies have explored predictive cut-off values for adiponectin. Further exploratory studies are recommended to validate and standardize these cut-off points for this biomarker.

In the present study, 7 participants (10.4%) were found to have renal involvement in the form of microalbuminuria. The median (range) serum adiponectin level was 0.83 (0.61–1.66) µg/ml in participants with renal involvement and 1.60 (0.41–7.00) µg/ml in those without renal involvement. Serum adiponectin levels were significantly lower in participants with renal involvement ($p = 0.048$).

Conclusion

This study highlights the critical relationship between obesity, adipokine dysregulation, and renal health. A considerable number of sedentary obese individuals demonstrated renal involvement, evidenced by microalbuminuria, an essential indicator of early kidney damage. Participants with microalbuminuria exhibited significantly reduced serum adiponectin levels, supporting the hypothesis that adiponectin could play a pivotal role as a biomarker in the development of obesity-related renal dysfunction. The study revealed a weak yet significant negative correlation between BMI, eGFR, and serum adiponectin levels, suggesting that renal stress associated with obesity might lead to a decline in adiponectin levels. Furthermore, a serum adiponectin cut-off value of ≤1.0 µg/ml demonstrated high sensitivity, specificity, and diagnostic accuracy, highlighting its potential as a reliable predictor of microalbuminuria. Overall, the findings highlight the importance of implementing comprehensive management strategies aimed at addressing sedentary obesity, inflammation, and metabolic health to mitigate the risk of renal and cardiovascular complications in this population.

Limitations of the study

The study has several limitations, including a relatively small sample size, which may not provide sufficient statistical power to identify smaller yet potentially important associations. Additionally, its cross-sectional design limits the ability to establish causal relationships between obesity, inflammation, and renal dysfunction. Moreover, the study did not incorporate a detailed and comprehensive evaluation of dietary habits, lifestyle factors, physical activity levels, or genetic influences, all of which could impact adipocytokine levels and renal outcomes.

Recommendations

Future studies should involve larger, longitudinal investigations to better understand the causal relationships and explore the long-term impact of interventions aimed at reducing obesity and inflammation. Additionally, it is recommended to investigate the contribution of other potential factors influencing renal health, such as dietary patterns and physical activity levels.Future research should also consider incorporating genetic testing to identify hereditary factors that may influence both obesity and renal disease.Additionally, promoting weight loss strategies, including consistent exercise and dietary improvements, is crucial for reducing inflammation and enhancing renal health in sedentary obese individuals

Declarations

Conflict of Interest: None declared.

Ethical approval: The study was approved by the institutional Ethics committee.

References

1. Kurokawa K, Nangaku M, Saito A, et al., Current issues and future perspectives of chronic kidney disease: A view from Japan. Kidney Int Suppl 79 (2011): S21–S29.
2. Bello AK, Nwankwo E, El Nahas AM. Prevention of chronic kidney disease: A global challenge. Kidney Int Suppl 72 (2007): S11–S17.
3. Ejerblad E, Fored CM, Lindblad P, et al., Obesity and risk for chronic renal failure. J Am Soc Nephrol. 2006;17(6):1695–702.
4. Farb MG, Gokce N. Obesity-related inflammation and endothelial dysfunction in cardiovascular disease. Front Endocrinol 8 (2017): 183.
5. Kim SM, Cho KI, Park KS. Relationship between adiponectin levels and cardiovascular disease in subjects with or without metabolic syndrome. J Clin Endocrinol Metab 101 (2016): 2486–2494.
6. Yoon JH, Jeong YJ, Jeong YJ, et al. Serum adiponectin levels and the risk of metabolic syndrome: A 4-year longitudinal study. J Clin Endocrinol Metab 101 (2016): 2550–2555.
7. Ouchi N, Parker JL, Lugus JJ, et al., Adipokines in inflammation and metabolic disease. Nat Rev Immunol. 11 (2021): 85–97.
8. Bello AK, Nwankwo E, El Nahas AM. Prevention of chronic kidney disease: A global challenge. Kidney Int Suppl 72 (2007): S11–S17.
9. Xiao N, Wang Y, Zheng S, et al., Obesity and its impact on adolescent kidney health: Insights from a population-based study. J Am Soc Nephrol. 2020;31 (2020):1505–1514.
10. El-Atat F, Aneja A, McFarlane S, et al., Obesity and hypertension. Endocrinol Metab Clin North Am 35 (2017): 823–854.
11. Praga M, Hernández E, Morales E, et al., Clinical features and long-term outcome of obesity-associated focal segmental glomerulosclerosis. Nephrol Dial Transplant 31 (2016):1841–1848.
12. Freedman DS, Thornton JC, Pi-Sunyer FX, et al., The body adiposity index (hip circumference ÷ height$^{1.5}$) is not a more accurate measure of body fatness than BMI, waist circumference, or hip circumference. Obesity (Silver Spring) 21 (2013): 856–862.
13. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl 3 (2013): 1–150.
14. National Kidney Foundation. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. Am J Kidney Dis 49 (2007): S1–S180.
15. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. Geneva: World Health Organization; (2000).
16. World Health Organization. Guidelines on physical activity and sedentary behaviour. Geneva: World Health Organization; 2020.
17. World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation. Geneva: World Health Organization; 2008.
18. de Almeida AR, Monte-Alegre S, Zanini MB, et al., Association between prehypertension, metabolic and inflammatory markers, decreased adiponectin and enhanced insulinemia in obese subjects. J Hum Hypertens 28 (2014):731-735.
19. Kawamoto R, Tabara Y, Kohara K,et al., Serum high molecular weight adiponectin is associated with mild renal dysfunction in Japanese adults. J Atheroscler Thromb.17 (2010).
20. Chen S, Zhou S, Zhao Y, et al., Association between metabolically unhealthy overweight/obesity and chronic kidney disease: the role of inflammation. Kidney Blood Press Res 40 (2014): 423–430.
21. Song SH, Oh TR, Choi HS, et al. High serum adiponectin as a biomarker of renal dysfunction: results from the KNOW-CKD study. Sci Rep 10 (2020): 5598.
22. Mackowiak-Lewandowicz K, Ostalska-Nowicka D, Zaorska K, et al., Chronic kidney disease predictors in obese adolescents. Pediatr Nephrol (2022).
23. El-Shaheed A, Fahmy R, Mahfouz N, et al., Evaluation of serum leptin and adiponectin and their associations with obesity-related renal injury among Egyptian adolescents. J Arab Soc Med Res 18 (2023):134.
24. Lee BT, Ahmed FA, Hamm LL, et al., Association of C-reactive protein, tumor necrosis factor-alpha, and interleukin-6 with chronic kidney disease. BMC Nephrol 16 (2015): 77.
25. Barakat L, Shora H, El-Deen I, et al., Inflammatory biomarkers of cardiometabolic risk in obese Egyptian type 2 diabetics. Med Sci 5 (2017): 25.
26. Xiao H, Xiong C, Shao X, et al. Visceral Adiposity Index

Rahaman A, et al., Arch Nephrol Urol 2025
DOI:10.26502/anu.2644-2833090

and chronic kidney disease in a non-diabetic population: a cross-sectional study. Diabetes Metab Syndr Obes 13 (2020): 257–265.

27. Alnaggar ARLR, Sayed M, El-deena KE, et al., Evaluation of serum adiponectin levels in diabetic nephropathy. Diabetes Metab Syndr Clin Res Rev 13 (2019): 128–131.