Original Article

Microalbuminuria in normotensive nondiabetic overweight and obese versus nonobese adults

Kiran, Madhubala Negi

Department of General Medicine, ABVIMS and Dr. RMLH, New Delhi, India

Abstract

Introduction: Obesity has reached an epidemic stage worldwide. As the prevalence of obesity increases, the burden of its associated comorbidities increases. It has been linked to altered hemodynamics leading to glomerulopathy that results in microalbuminuria, proteinuria, and chronic kidney disease. Recent population-based surveys have shown that overweight and obesity are the risk factors for the development and progression of nephropathy in normotensive and nondiabetic adults as well. The underlying causes may involve adipogenic inflammation and endothelial dysfunction that results in microalbuminuria. The population of the Asia Pacific region appears to be at higher risk of obesity-related morbidities at lower BMI. An established indicator of early renal impairment in diabetes and hypertension is microalbuminuria (MAU). Our study aims to screen obese and overweight adults and compare them with nonobese adults to estimate the prevalence of microalbuminuria in both groups when risk factors such as diabetes and hypertension are excluded. **Material and Methods:** This study was a comparative cross-sectional study carried out in a tertiary care hospital from January 2021 to May 2022. An appropriate statistical method was applied to calculate the sample size. A total of 75 obese individuals were taken as cases and 75 age- and sex-matched nonobese adults as controls were taken for the study.

Results and Conclusion:

- The prevalence of microalbuminuria was found to be 24% in cases compared to 5.33% in controls, which was statistically significant, with a *P*-value of 0.002.
- The distribution of microalbuminuria was comparable between overweight and obese (20% vs 28.57% respectively) (*P*-value = 0.386).
- Overweight and obese adults were 5 times more likely to develop microalbuminuria compared to nonobese adults.
- This study highlights the urgent need to reverse the epidemic of obesity among young adults in India considering its role as a risk factor for cardiovascular diseases and progression of renal disease.

Keywords: BMI, estimated glomerular filtration rate, microalbuminuria

Introduction

Obesity has reached an epidemic stage worldwide. As the prevalence of obesity increases, the burden of its associated comorbidities increases.[1] Lower BMI thresholds have been proposed for the Asia Pacific region for overweight and obesity because this population appears to be at higher risk of obesity-related morbidities at lower BMI.[2] Obesity is now recognized as a disease in itself due to the increased risk of associated morbidities and mortalities.[3]

Around the world, 650 million adults are obese and over 1.9 billion are overweight. Obesity-related fatalities are estimated to have caused 2.8 million deaths worldwide.[4] There are more than 135 million obese people in India. According to the 2015 ICMR-INDIA study, the prevalence rates of obesity and central obesity range from 11.8% to 31.3% and 16.9% to 36.3%, respectively.[5] Obesity is an established risk factor for diabetes and hypertension. An established indicator of early renal impairment

Address for correspondence: Dr. Kiran, Fourth Floor, House No 8, Gali No 8, B2 Extension, Krishna Nagar, Safdarjung Enclave, New Delhi - 110029, India.
E-mail: doctorkiranyadav@gmail.com

Received: 10-09-2024 **Revised:** 23-10-2024
Accepted: 01-11-2024 **Published:** 24-09-2025

Access this article online

Quick Response Code:

Website: http://journals.lww.com/JFMPC

DOI: 10.4103/jfmpc.jfmpc_1562_24

How to cite this article: Kiran, Negi M. Microalbuminuria in normotensive nondiabetic overweight and obese versus nonobese adults. J Family Med Prim Care 2025;14:3199-204.

in diabetes and hypertension is microalbuminuria (MAU). The underlying causes of the connection between obesity and the development of nephropathy are currently unknown. It may involve adipogenic inflammation and endothelial dysfunction that results in microalbuminuria.[6] The changes include increased intraglomerular capillary pressure, glomerular hyperfiltration, glomerulosclerosis, and increased urine albumin excretion.[7-9] The prevalence of microalbuminuria in overweight and obese populations are 3.1% and 12.1%, respectively, in a population-based study.[10]

Indian obese people are recognized to have a unique biochemical profile compared to people of other races. Information on the prevalence of microalbuminuria in healthy, obese, and overweight Indian adults is lacking. Whether nondiabetic normotensive obese adults deserve targeted identification, screening, and clinical intervention for microalbuminuria is a debatable issue. Our study aimed to screen obese and overweight adults and compare them with nonobese adults to estimate the prevalence of microalbuminuria in both groups, when risk factors such as diabetes and hypertension are excluded.

Methods

It is a comparative cross-sectional study conducted in a tertiary care hospital. This study was approved by the Institutional Ethics Committee. Patients attending outpatient or inpatient departments of medicine were taken as study subjects. Those satisfying inclusion criteria were screened for hypertension and diabetes mellitus. The sample size was calculated based on a relatable statistical formula for comparative study. Taking the prevalence of microalbuminuria in obese and nonobese subjects to be 11.6% and 3.3% respectively in a study conducted by Bhatt *et al.*,[10] the sample size came out to be 150. Considering the feasibility and time constraint in mind and taking into account inclusion and exclusion criteria, a sample size of a minimum of 75 obese and overweight adults as cases and 75 age- and sex-matched non-obese adults were taken as controls for the study.

$$\text{Sample size } (n) = [Z_{1-\alpha/2}\sqrt{\{2p(1-p)\}} + Z_{1-\beta}\sqrt{\{p_1(1-p_1) + p_2(1-p_2)\}}]^2/(p_1-p_2)^2$$

where n is the sample size, Z1-α/2 and Z1-β are the critical values of the given level of confidence at two-sided. Test and power of the study; p1 and p2 are the proportions in treatment and control groups, P is the average value of p1 and p2, d is the effect size. The confidence interval is taken to be 95% and the power is taken 80%.

Individuals having diabetes mellitus, hypertension, ischemic heart disease, congestive heart failure, clinical and laboratory evidence of renal disease, fever, urinary tract infections, history of strenuous physical exercise during the previous 24 h before the test, on nephrotoxic drugs, and pregnancy were excluded from the study. Cases included overweight and obese subjects as per case definitions by the World Health Organization (WHO) for the Asia Pacific region.[2] Overweight was defined as body mass index (BMI) in the range of 23–24.9 kg/m^2. Those with a BMI of >25 kg/m^2 and/or WC of >90 cm in males and >80 cm in females were defined as obese. Adults having a BMI in the range of 18.5–22.9 kg/m^2 and having a waist circumference of less than 90 cm in males and less than 80 cm in females were taken as a control group.

Spot urinary sample was taken for urine routine and microscopy examination and estimation of urinary albumin creatinine excretion. The urine albumin reagent was used for the quantification of spot urinary albumin by turbidimetric method on the Beckman Coulter clinical chemistry Auto Analyzer. Urinary creatinine was measured by the Jaffe kinase method. The calculated ratio between urinary albumin and creatinine was taken for urine albumin creatinine ratio (UACR) determination.

UACR was calculated as milligrams of albumin per gram of creatinine. Microalbuminuria was taken as a UACR of 30–300 mg/g (ADA criteria, 2014).[11] eGFR was calculated in each patient by MDRD formula: eGFR = 186 × S. creatinine$^{-1.154}$ × age$^{-0.203}$ × (0.742 if female).

The data were analyzed using SPSS (2015 version). A descriptive statistical analysis was conducted. Correlations between the quantitative variables were examined by the Pearson correlation coefficient test. The groups were compared using the *t*-test for the continuous variables and the Chi-square test for the categorical variables. *P*-values of 0.05 or less were considered statistically significant.

Results

A total of 75 cases were taken that included both overweight and obese adults and 75 age- and sex-matched adults were taken as controls.

The median (25th–75th percentile) of age (years) in cases was 37 (29.5–45) and controls were 35 (31–42) with no significant difference between them (P-value = 0.524). Among cases, the majority of patients 58.66% were in the 31–50 years age group followed by 26.67% in the 18–30 years and 14.67% in the 51–60 years age group. This was comparable to controls where 61.33% were in the 31–50 years age group, 24% in the 18–30 years age group, and 14.67 in the 51–60 years age group Figure 1.

Distribution of gender was comparable between cases and controls (female: 36% vs. 38.67% respectively, male: 64% vs. 61.33%, respectively) (P-value = 0.736) [Figure 2].

Significant difference was observed in anthropometric parameters body mass index (kg/m^2), and waist circumference (cm) between cases and controls (P-value < 0.05) [Table 1].

A significant difference was observed in total cholesterol, LDL, and triglycerides between cases and controls (*P*-value < 0.05). The mean ± standard deviation (SD) of total cholesterol (mg/dL) in cases was 195.43 ± 24.62, which was significantly higher as compared to controls (151.41 ± 20.84) (*P*-value < 0.0001) [Figure 3.1]. The median of LDL (mg/dL), and triglycerides (mg/dL) in cases was 104.8 (94.3–123.3), and 210 (186–240), respectively, which was significantly higher as compared to controls (75.2 [62.7–86]), 178 [160–188]), respectively. No significant difference was observed in HDL between cases and controls (*P*-value = 0.464). The median (25th–75th percentile) of HDL (mg/dL) in cases was 43 (40–47) and in controls was 44 (40–49) with no significant difference between them [Figure 3.2].

The proportion of subjects with microalbuminuria was significantly higher in cases as compared to controls (24% vs. 5.33% respectively) (*P*-value = 0.002) [Figure 4]. There were 18 subjects among cases and 4 among controls, having microalbuminuria. The distribution of microalbuminuria was comparable between overweight and obese (20% vs. 28.57%, respectively) (*P*-value = 0.386) [Figure 5].

The median (25th–75th percentile) of urine ACR (mg/g) in cases was 17 (8.2–28.3), which was significantly higher as compared to controls (13.1 [6.39–20.965]) (*P*-value = 0.025) [Table 2].

Median (25th–75th percentile) of urine ACR (mg/g) in 23 to 24.99 kg/m² (overweight) was 20.25 (10.425–27.05) and ≥25kg/m² (obese) was 14.4 (7.7–37.45) with no significant difference between them (*P*-value = 0.807) [Table 3].

An eGFR of more than 120 mL/min/kg/m², has been considered as high eGFR. The proportion of patients with high eGFR was significantly higher in cases as compared to controls 52% vs. 17.33% respectively (*P*-value < 0.0001). The mean ± SD of eGFR (mL/min) in cases was 120.82 ± 18.66 and the controls were 117.65 ± 16.45 with no significant difference between them (*P*-value = 0.27) [Table 4].

Distribution of high eGFR was comparable between overweight and obese (50% vs. 54.29%, respectively) (*P*-value = 0.711) [Figure 6]. The median (25th–75th percentile) of eGFR (mL/min) in 23 to 24.99 kg/m² (overweight) was 120.44 (110.495–129.208) and ≥ 25 kg/m² (obese) was 122.03 (107.435–130.417) with no significant difference between them (*P*-value = 0.937).

A nonsignificant mild positive correlation was observed between urine ACR (mg/g) with body mass index (kg/m²) with a correlation coefficient of 0.101 [Figure 7].

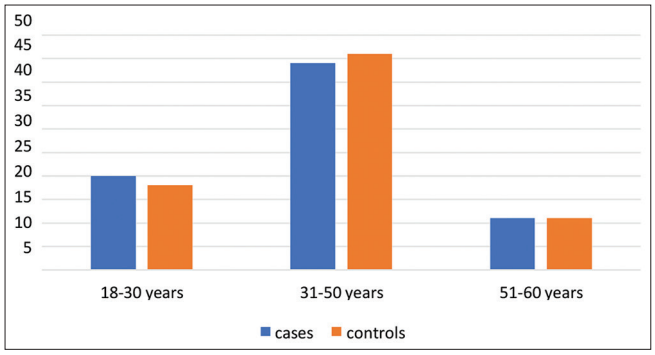

Figure 1: Age distribution among cases and controls

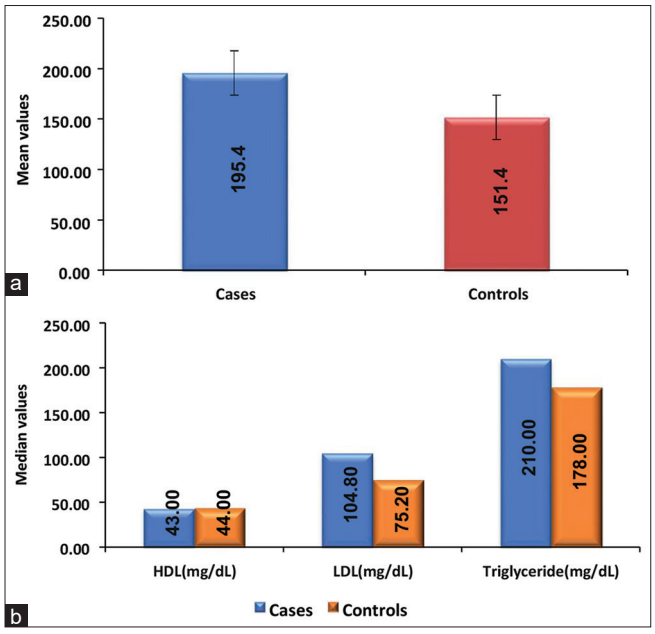

Figure 3: (a) Comparison of total cholesterol between cases and controls. (b) Comparison of lipid profile between cases and controls

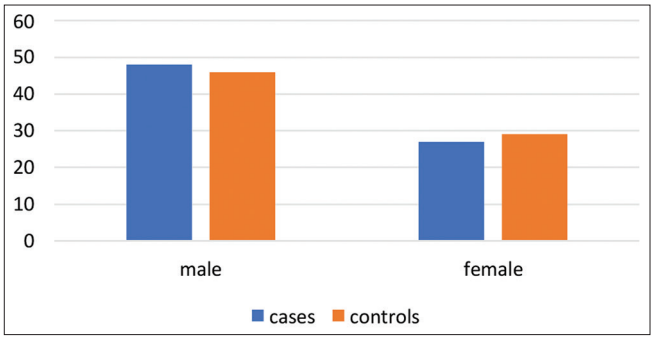

Figure 2: Sex distribution among cases and controls

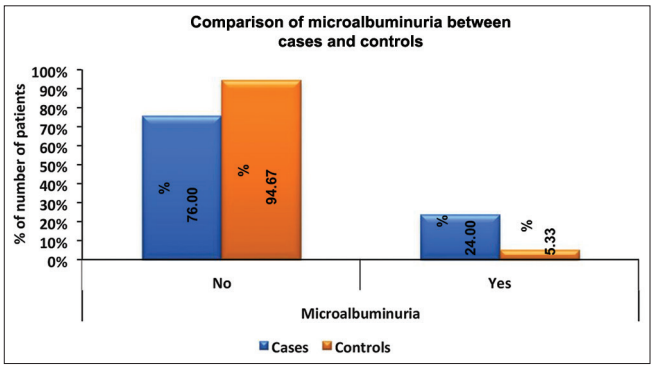

Figure 4: Comparison of microalbuminuria between cases and controls

No correlation was observed between eGFR (mL/min) with body mass index (kg/m²), with a correlation coefficient of 0.032.

Discussion

Our study aimed to compare the prevalence of microalbuminuria among normotensive nondiabetic overweight and obese adults with nonobese healthy adults. In our study, the majority of the cases were in the younger age groups 18–30 years (26.67%), 31–50 years (58.66%) which was comparable to the controls, 18–30 years (24%), 31–50 years (61.33%). The study conducted by Bhatt *et al.*[10] had the majority of study participants in the age group 20–30 years (53.33%) and 31-40 years (36.66%). In our study, among cases, 64% were males and 36% were females and among controls, 61% were males and 38.67% were females. This was comparable to a study conducted by Bhatt *et al.*,[10] in which among cases 60% were male and 40% were female, and among controls, 56.66% were male and 43.33% were female.

In our study, the proportion of overweight (BMI of 23 to 24.99 kg/m²) individuals was 53.33% and that of obese (BMI of more than 25 kg/m²) was 46.67% among cases. The mean BMI among cases was 26 ± 2.39 kg/m² and that among controls was 21.21 ± 1.11 kg/m². The mean waist circumference of cases was 92.03 ± 5.72 cm and that of controls was 70.31 ± 4.38 cm.

There was a significant difference noted in total cholesterol (mg/dL), LDL (mg/dL), and triglycerides (mg/dL) values between cases and controls (*P*-value < 0.05). Mean ± SD of total cholesterol (mg/dL) in cases was 195.43 ± 24.62, which was significantly higher as compared to controls (151.41 ± 20.84). The mean LDL, and triglycerides value among cases was 108.79 ± 23.47 mg/dL and 214.92 ± 56.39, and that among controls was 73.26 ± 16.4 mg/dl and 171.43 ± 30.41 mg/Dl, respectively. There was no significant difference in HDL mean levels between cases and controls. This was similar to the study conducted by Bhatt *et al.*,[10] which showed higher levels of total cholesterol and LDL among cases and a significant decrease in HDL levels. In the study by Bhatt *et al.*, the mean total cholesterol was 170 ± 25.54 mg/dL in cases as compared to controls, which had a value of 155.78 ± 16.72 mg/dL. The mean low-density lipoprotein (LDL) values in cases and controls were 111.39 ± 25.39 and 87.47 ± 16.90 mg/dL, respectively. The total cholesterol and LDL were significantly higher (*P* = 0.000315 and *P* = 0.00001, respectively) in obese than in nonobese subjects. Abnormalities of lipid metabolism are commonly observed in overweight and obese individuals. Hence, patients with deranged lipid profiles could not be excluded from the study.

Table 1: Comparison of anthropometric parameters between cases and controls

Anthropometric parameters	Cases (*n*=75)	Controls (*n*=75)
Body mass index (kg/m²)		
18.5-22.99 kg/m² (normal BMI)	0 (0%)	75 (100%)
23-24.99 kg/m² (overweight)	40 (53.33%)	0 (0%)
≥25 kg/m² (obese)	35 (46.67%)	0 (0%)
Mean±SD	26±2.39	21.21±1.11
Waist circumference (cm)		
Mean±SD	92.03±5.72	70.31±4.38

§,†Mann–Whitney test, Chi-square test

Table 2: Comparison of urine ACR (mg/g) between cases and controls

Urine ACR (mg/g)	Cases (*n*=75)	Controls (*n*=75)	*P*
Median (25th–75th percentile)	17 (8.2-28.3)	13.1 (6.39-20.965)	0.025

§Mann–Whitney test

The primary objective of our study was to estimate the prevalence of urinary albumin excretion (UAE) in terms of urinary albumin creatinine ratio (UACR). The mean urine albumin excretion in our study among cases was 35.52 ± 53.23 versus 16.74 ± 20.09 mg/g of creatinine among controls. The UAE was twice as much higher in cases as compared to controls and was statistically significant with a *P*-value of 0.025. This was nearly similar to the study done by Bhatt *et al.*[10] in which, the mean UAE in cases was 21.20 ± 26.82 mg/g creatinine and in controls was 13.55 ± 9.47 mg/g creatinine.

In our study, the proportion of subjects with microalbuminuria was significantly higher in cases (24%) as compared to controls (5.33%) with a *P*-value of 0.002. Hence, nearly five times higher prevalence was seen in cases compared to controls. This was higher as compared to the study conducted by Valensi *et al.*,[12] which showed the prevalence of microalbuminuria in the control group with BMI <25 kg/m² to be 3.1% compared to 12.1% in nondiabetic obese cases and in particular, in 19.2% of the obese patients with hypertension.

Similarly, 11.66% of patients had microalbuminuria compared to 3.33% of controls in a study done by Bhatt *et al.*[10] In the study conducted by Pavan *et al.*,[13] microalbuminuria was prevalent in 40% of obese subjects compared to 4.2% of non-obese subjects, which was higher compared to our study. This difference in the prevalence of microalbuminuria could be attributed to various factors such as the population studied, methods of measuring urine albumin, and methods of urine sample collection.

On analysis of the prevalence of microalbuminuria in subgroups of cases in our study, microalbuminuria was present in 8 (10.66%) cases of overweight and 10 (13.33%) cases of obese individuals, the difference however was not statistically significant. In a study conducted by Minoo *et al.*,[14] the prevalence of microalbuminuria

Table 3: Comparison of urine ACR (mg/g) between overweight and obese

Urine ACR (mg/g)	Overweight (*n*=40) 23-24.99 kg/m²	Obese (*n*=35) ≥25 kg/m²	Total	*P*
Median (25th–75th percentile)	20.25 (10.425–27.05)	14.4 (7.7–37.45)	17 (8.2–28.3)	0.807

§Mann–Whitney test

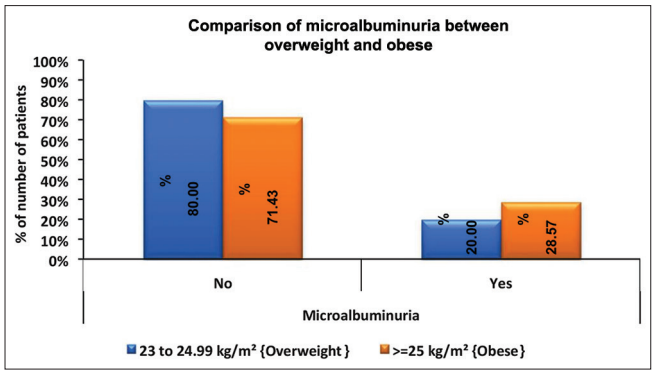

Figure 5: Comparison of microalbuminuria between overweight and obese

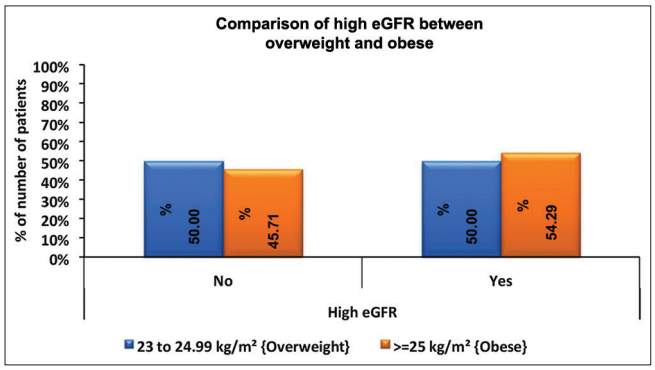

Figure 6: Comparison of high eGFR between overweight and obese

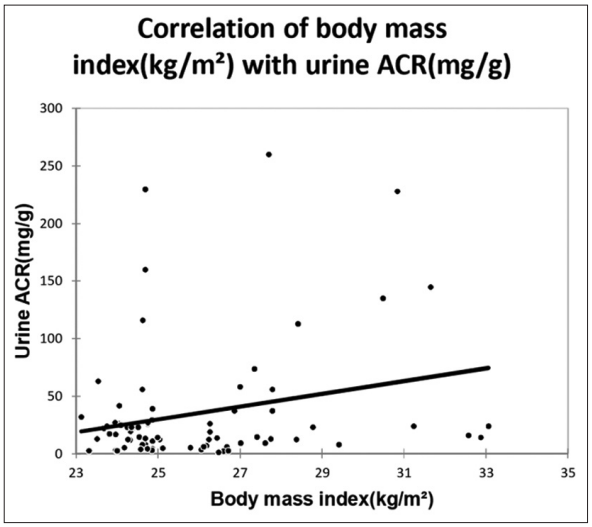

Figure 7: Correlation of BMI with urine ACR

Table 4: Comparison of eGFR (mL/min) between cases and controls

eGFR (mL/min)	Cases (*n*=75)	Controls (*n*=75)
High eGFR		
No	36 (48%)	62 (82.67%)
Yes	39 (52%)	13 (17.33%)
eGFR (ml/minute)		
Mean±SD	120.82±18.66	117.65±16.45

§Mann–Whitney test, †Chi-square test

was 11.8% overall in obese normotensive nondiabetic subjects, which was similar to our study.

Obesity leads to increased renal blood flow and glomerular hyperfiltration causing the eGFR to be abnormally high (>120 mL/min/kg/m²). In our study, the mean eGFR among cases was higher, 120.82 ± 18.66 versus 117.65 ± 16.45 among controls, the difference was not however statistically significant. The cases however had a significantly larger proportion of subjects with high eGFR than controls (52% vs. 17.33%, respectively) with a *P*-value of less than 0.0001. The eGFR was 61% higher in the obese than in the normal BMI group as evidenced by the study done by Chagnac *et al.*[15] In our study, the prevalence of high eGFR was comparable between overweight and obese (50% vs. 54.29%, respectively) with a *P*-value of 0.711.

Several studies have shown that abdominal obesity and high BMI are associated with a higher prevalence of microalbuminuria. In our study, a nonsignificant mild positive correlation was seen between urine ACR (mg/g) with body mass index (kg/m²) with a correlation coefficient of 0.101. No statistically significant correlation was observed between waist circumference and microalbuminuria among cases. In a cross-sectional survey conducted by Seo *et al.*[16] in Korea, abdominal obesity was not significantly associated with microalbuminuria in the general population. A study conducted by Bhatt *et al.*[10] also showed that an increase in waist circumference was not associated with an increase in microalbuminuria. On the contrary, in a study done by Hemayati *et al.*[17] in Iran, the prevalence of microalbuminuria increased with increasing BMI.

No significant correlation was observed between anthropometric measurements and eGFR in our study. However, in a study conducted by Bhatt *et al.*, a moderately statistically significant correlation was found between waist circumference, BMI, and eGFR. This difference may be attributed to the fact that the majority of patients in the study by Bhatt *et al.* were in the Class 1 obese group with a BMI of 25–29.9 kg/m² (78%) followed by the Class 2 obese group BMI >30 kg/m² (22%). In our study, 53.33% of cases were overweight and only 46.67% were in the obese category.

The limitations of this study were that a sample size of 150 was taken, which is small considering the high prevalence of obesity in India. The cross-sectional design limited causal inferences in this study.

Conclusion

The results of this study showed a high prevalence of microalbuminuria in young nondiabetic normotensive overweight and obese adults. They were also found to have higher eGFR compared to nonobese healthy adults suggestive of glomerular hyperfiltration. The results of this study call for lifestyle modifications in younger obese adults who otherwise do not have any other risk factors for renal disease.

Most of the population-based studies, correlating microalbuminuria and obesity have been done on the Western population. Further, population-based studies are required in India, with a longer duration of follow-up for ascertaining or refuting the impact of obesity on microalbuminuria in the absence of other risk factors. This study highlights the urgent need to reverse the epidemic of obesity among young adults in India considering its role as a risk factor for cardiovascular diseases and progression of renal disease.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Kushner R, Jameson L, Kasper L, Longo L, Fauci A, Hauser L, Loscalzo J, editor. Evaluation and Management of Obesity, Harrison's Principles of Internal Medicine. vol. 2. 20th ed. New York: McGraw-Hill Education; 2018. p. 2843-44.
2. The Asia Pacific perspective: Redefining obesity and its treatment, World Health Organization. International Association for the Study of Obesity and the International Obesity Task Force: St Leonards, Australia; Health Communications Australia Pty Limited; 2000. p. 22–9.
3. Flegal KM, Kit BK, Orpana H, Graubard B. Association of all-cause mortality with overweight and obesity using standard body mass index categories a systematic review and meta-analysis. JAMA 2013;309:71-82.
4. WHO Global Status Report on noncommunicable diseases. Burden: Mortality, morbidity, and risk factors. 2010. p. 144-51.
5. Pradeepa R, Anjana R, Joshi R, Bhansali A, Deepa M, Joshi P, *et al.* Prevalence of generalized and abdominal obesity in urban and rural India- the ICMR-INDIAB study (Phase-I) [ICMR-INDIAB-3]. Indian J Med Res 2015;142:139-50.
6. McGown C, Birerdinc A, Younossi ZM. Adipose tissue is an endocrine organ. Clin Liver Dis 2014;18:41-58.
7. Chagnac A, Herman M, Zingerman B, Erman A, Rozen-Zvi B, Hirsh J, *et al.* Obesity-induced glomerular hyperfiltration: Its involvement in the pathogenesis of tubular sodium reabsorption. Nephrol Dial Transplant 2008;23:3946-52.
8. Bosma RJ, Krikken JA, Homan JJ, De Jong PE, Navis GJ. Obesity and renal hemodynamics. Contrib Nephrol 2006;151:184-202.
9. Tomaszewski M, Charchar FJ, Maric C, McClure J, Crawford L, Grzeszczak W, *et al.* Glomerular hyperfiltration: A new marker of metabolic risk. Kidney International 2007;71:816-21.
10. Bhatt VR, Khese VB, Jadhav SL, Kakrani AL. Urinary albumin excretion, estimated glomerular filtration rate and prevalence of microalbuminuria in obese nondiabetic and nonhypertensive adults: A cross-sectional study. Indian J Nephrol 2019;29:166-71.
11. Tuttle KR, Bakris GL, Bilous RW. Diabetic kidney disease: A report from an ADA consensus conference. Diabetes Care 2014;37:2864–83.
12. Valensi P, Assayag M, Busby M, Paries J, Lormeau B, Attali JR. Microalbuminuria in obese patients with or without hypertension. Int J Obes Relat Metab Disord 1996;20:574-9.
13. Pavan M, Ranganath R, Chudhari A, Shetty M. Obesity as an independent risk factor for the development of microalbuminuria. Nephro Urolo Mthly 2011;3:276-9.
14. Minoo F, Mahdavi-Mazdeh M, Abbasi MR, Sohrabi S. Impact of the severity of obesity on microalbuminuria in obese normotensive nondiabetic individuals. J Renal Inj Prev 2015;4:34-8.
15. Chagnac A, Weinstein T, Herman M, Hirsh J, Gafter U, Ori Y*et al.* The effects of weight loss on renal function in patients with severe obesity. J Am Soc Nephrol 2003;14:1480-6.
16. Seo WJ, Lee GM, Hwang JH, Lee M N, Kang HC. Association between body mass index, waist circumference and prevalence of microalbuminuria in Korean adults of age 30 years and older without diabetes, hypertension, renal failure, or overt proteinuria: The 2013 Korean National Health and Nutrition Examination Survey. Korean J Family Med 2016;37:57-63.
17. Hemayati R, Kaseb F, Ghadiri A, Yosefi F. The relationship between microalbuminuria, overweight, and obesity. J Nephropathol 2020;9:e4.