Original Article

Increased Body Mass Index is Independently Associated with Chronic Kidney Disease among People with Type 2 Diabetes

Abstract

Background: The alarming increase in the prevalence of obesity has implications for chronic kidney disease (CKD) progression in type 2 diabetes (T2D). This study aimed to assess if increased body mass index (BMI) can be an independent risk factor for CKD and T2D in the Indian context. **Materials and Methods:** In this cross-sectional study, 602 (M:F = 378:224) participants were screened using Kidney Disease Improving Global Outcomes (KDIGO) from January to October 2023 in Chennai. Demographic, anthropometric, biochemical, clinical details, and comorbidities were recorded. T2D with CKD low risk was taken as control group, and CKD moderate and high risks were the study groups. BMI was classified based on the Asian criteria into normal (18.5–22.9), overweight (23–24.9), and obese (≥25 kg/m^2). **Results:** Majority of participants in moderate and high risk categories were obese compared to the low risk category (60.5% and 66.4% vs. 39.1%; $p < 0.001$). A higher proportion of participants was on antihypertensive drugs in the high risk group and in the obese category ($p < 0.001$). Comorbidities and diabetic complications were higher in the high risk group ($p < 0.001$). Multivariate logistic regression revealed that age of ≥ 60 years [OR(95% CI); 6.3(2.2–18); $p = 0.009$]; increased BMI as overweight [3.6(2.1–6.3); $p < 0.001$] and obese [5.2(3.3–8.3); $p < 0.001$]; smoking [4.2(1.7–10.2); $p = 0.002$]; increased duration of diabetes of 5–15 years [2.3(1.2–4.5); $p = 0.013$], 16–25 years [4.8(2.2–10.4); $p < 0.001$], and >25 years [4.2(1.4–13); $p = 0.011$]; systolic blood pressure [1.01(1.0–1.03); $p = 0.02$]; and hemoglobin A1c [1.2(1.1–1.3); $p < 0.001$] were independent risk factors for the progression of CKD. **Conclusion:** Increased BMI was independently associated with CKD in T2D. Overweight and obese individuals are four to five times at risk for CKD progression. Early identification, lifestyle intervention, and weight-lowering drugs may reduce the complications of obesity in T2D and CKD.

Keywords: *BMI, Chronic kidney disease, Type 2 diabetes, India*

Vijay Viswanathan[1], Sivashankari Selva Elavarasan[2], Satyavani Kumpatla[2]

[1]Department of Diabetology, [2]Diabetic Kidney Disease Research, M.V. Hospital for Diabetes and Prof. M. Viswanathan Diabetes Research Centre (IDF Centre for Excellence in Diabetes Care), Royapuram, Tamil Nadu, Chennai, India

Corresponding author: *Vijay Viswanathan, Department of Diabetology, M.V. Hospital for Diabetes and Prof. M. Viswanathan Diabetes Research Centre (IDF Centre for Excellence in Diabetes Care), Royapuram, Chennai, Tamil Nadu, India. E-mail: drvijay@mvdiabetes.com*

DOI: 10.25259/IJN_319_2024

Received: 21-06-2024
Accepted: 04-09-2024
Online First: 25-10-2024
Published: ***

Supplementary available on: https://dx.doi.org/10.25259/IJN_319_2024

Introduction

There are 537 million people aged between 20 and 79 years affected globally by diabetes, and these numbers are expected to multiply rampantly by the year 2045.[1] A recent study by the Indian Council of Medical Research—India Diabetes (ICMR-INDIAB) states that the overall prevalence of diabetes in India was found to be 11.4%, with approximately 101 million people affected by the disease.[2] These numbers are expected to rise exponentially due to the increase in the prevalence of obesity and the recent trends in the modification of lifestyle as well as urbanization.[3] There are around 650 million people who are obese and two billion adults who are overweight.[4] The highest increase in the rates of obesity was observed in the Southeast region, which includes India with nearly 135 million people living with obesity.[5] The prevalence of obesity in India was found to be 40.3%, with the highest prevalence recorded in the southern region (46.5%).[6] This alarming increase in the prevalence of obesity has substantial implications for the progression and development of chronic kidney disease (CKD), especially among people with type 2 diabetes mellitus (T2D).[7–9]

Diabetic kidney disease (DKD) is one of the most common and serious complications of T2D and is also considered as one of the major reasons for CKD as well as end-stage renal disease (ESRD) affecting nearly 30–40% of people with T2D.[10] There exists a strong association between

How to cite this article: Viswanathan V, SelvaElavarasan S, Kumpatla S. Increased Body Mass Index is Independently Associated with Chronic Kidney Disease among People with Type 2 Diabetes. Indian J Nephrol. doi: 10.25259/IJN_319_2024

DKD and obesity because it can cause proteinuria and glomerular hypertrophy as well as injury,[11,12] making it one of the predominant risk factors in the development and progression of DKD. Obesity is recognized as the most important risk factor for the development of proteinuria as well as ESRD.[13] Studies have indicated that being overweight can lead to glomerular hyperfiltration and renal hyperperfusion, both of which can cause focal segmental glomerulosclerosis as well as proteinuria.[14] There is limited literature available on whether an increased body mass index (BMI) can be attributed to the development of proteinuria in people with DKD. It remains unclear if increased BMI can be considered as a risk factor for the development of CKD in the Indian context using Kidney Disease Improving Global Outcomes (KDIGO).[15] This study aims to assess if increased BMI is an independent risk factor associated with CKD among people with T2D.

Materials and Methods

In this cross-sectional study, 1028 participants were screened using KDIGO guidelines from January to October 2023 in a tertiary care center for diabetes in Chennai. A total of 602 (M:F = 378:224) participants were included for the final analysis based on the inclusion and exclusion criteria. The schematic representation of patient recruitment has been shown in a flowchart [Supplementary Figure 1]. Written informed consent was obtained from all the participants and the study was approved by the Ethics Committee (IEC/N-006/01/2023). Considering the prevalence of obesity among the population affected as 46.5%,[6] with an acceptable margin of error of 5%, 95% CI, and an estimated power of 80%, the sample size was calculated to be 612.

Demographic, anthropometric details like age, gender, duration of diabetes, habits like smoking (smoking status was classified as nonsmokers, current, and ex-smokers) and alcohol consumption, and the presence of other comorbidities were also recorded. Chest X-rays, electrocardiogram (ECG), echocardiogram, and laboratory investigations were recorded for all the study participants. A brief history of coronary artery disease and details of their treatment regime were also recorded. Any abnormalities detected in the investigations or history recorded as diagnosed by a cardiologist was considered as the presence of cardiovascular disease.

BMI was calculated by dividing the weight measured in kilograms by the square of the height measured in meters during their hospital visit and expressed in kg/m.^{2} According to the Asian Pacific cut-off points, participants were classified based on their BMI into underweight (< 18.5), normal (18.5–22.9), overweight (23–24.9), and obese (≥ 25).[16] Biochemical parameters such as fasting and postprandial glucose, hemoglobin A1c (HbA1c), lipid profile, urea, creatinine, urine albumin-creatinine ratio (uACR), and urine protein-creatinine ratio (uPCR) were recorded. All the biochemical parameters were estimated using a BS-400 automated biochemistry analyzer. HbA1c was estimated using the high performance liquid chromatography (HPLC) procedure by using an automated glycohemoglobin analyzer (Adams HA-8180V-Arkray). Serum creatinine was measured by Jaffe's kinetic method and urinary albumin was measured using immunoturbidimetry. Urinary protein was estimated using the pyrogallol method. Estimated glomerular filtration rate (eGFR) was calculated based on the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[17] Participants were grouped into three categories: CKD as low risk, CKD as moderate risk, and CKD as high risk based on KDIGO[15] [Supplementary Figure 2].

Statistical analysis

Median, minimum, and maximum values for continuous variables, numbers, and percentages were reported for categorical variables. The chi-square test and Kruskall-Wallis test were performed as applicable for comparing the variables between different groups. Multiple logistic regression analysis was used to analyze the association of CKD with other potential risk factors, including BMI. The dependent variable entered was CKD as low risk versus CKD as moderate and high risk. The independent variables entered were gender (male vs. female), smoking (no vs. yes; nonsmokers vs. current and ex-smokers), age in categories of ten units (ref < 50 years vs. 51–59 and ≥ 60 years), BMI categories (normal vs. overweight and obese categories), duration of diabetes in ten units (ref < 5 years vs. 5–15, 16–25, and > 25 years), systolic and diastolic blood pressure, and HbA1c were entered as continuous variables. A P value of < 0.05 was considered to be statistically significant. Data was analyzed using SPSS (version 29.0, Illinois, USA) software.

Results

Table 1 shows the demographic, clinical, and biochemical details of the study participants based on KDIGO classification. The participants in the moderate and high risk groups of CKD were older than the low risk CKD group ($p < 0.001$). The majority of the participants in CKD moderate and high risk categories were obese when compared to the low risk category (60.5% and 66.4% vs. 39.1%; $p < 0.001$). The median duration of diabetes (12, 15 vs. 10; $p < 0.001$) and the habit of smoking (8.6%, 11.4% vs. 3.6%; $p = 0.009$) were significantly higher in advancing groups of CKD. The levels of fasting plasma glucose (156 vs. 137; $p = 0.004$) and HbA1c (8.7 vs. 8.1; $p = 0.014$) were significantly higher in group 2 than in group 1. The levels of triglycerides, serum urea, serum creatinine, and uACR were higher in group 3 when compared to group 1. As expected, the values of eGFR were significantly lower in high risk category when compared to low risk category (49 vs. 77; $p < 0.001$). Though the proportion of participants taking weight-reducing drugs like SGLT2i was higher in high risk group when compared to the other groups, there was no statistical significance and BMI in the CKD; the high risk group continued to remain high. There was an increased

Table 1: Demographic, clinical, and biochemical details of the study participants based on KDIGO classification

Characteristics	CKD low risk (n = 220)	CKD moderate risk (n = 162)	CKD high risk (n = 220)	P value between groups
Age (years)	54 (36,70)	58 (38,69)	62 (34,71)	< 0.001
M:F = 378:224	147:73	91:71	140:80	0.099
BMI (kg/m^2)	23.9 (17.8,32.9)	27.9 (18.4,41.2)	27.3 (17.7,54.6)	< 0.001
BMI categories				
Normal	91 (41.4)	30 (18.5)	24 (10.9)	< 0.001
Overweight	43 (19.5)	34 (21)	50 (22.7)	< 0.001
Obese	86 (39.1)	98 (60.5)	146 (66.4)	< 0.001
Blood pressure (mm Hg)				
Systolic	120 (97,180)	130 (90,200)	130 (77,192)	< 0.001
Diastolic	79 (50,110)	80 (50,120)	80 (57,130)	0.454
Duration of DM (years)	10 (2,32)	12 (2,34)	15 (2,44)	< 0.001
Smoking*	8 (3.6)	14 (8.6)	25 (11.4)	0.009
Current smoker	5 (2.3)	9 (5.5)	17 (7.7)	0.03
Ex-smoker	3 (1.3)	5 (3.1)	8 (3.7)	0.3
Alcohol consumption*	13 (5.9)	17 (10.5)	21 (9.5)	0.218
Plasma glucose (mg/dl)				
Fasting	137 (81,270)	156 (38,464)	154 (54,573)	0.004
Postprandial	236 (72,397)	244 (70,530)	238 (102,655)	0.293
HbA1c (%)	8.1 (5.6,14.6)	8.7 (5.7,14.9)	8.6 (5.3,16.4)	0.014
Total cholesterol (mg/dl)	164 (80,329)	155 (70,259)	154 (55,320)	0.349
LDL-C (mg/dl)	84 (33,182)	81 (35,120)	80 (16,175)	0.433
HDL-C (mg/dl)	49 (28,76)	53 (26,82)	49 (16,81)	< 0.001
TG (mg/dl)	102 (29,327)	111 (34,553)	134 (36,826)	< 0.001
eGFR (ml/min)	77 (61,134)	66.5 (45,108)	49 (15,59)	< 0.001
Serum creatinine (mg/dl)	1 (0.4,1.3)	1.1 (0.6,1.6)	1.5 (1,4.2)	< 0.001
Urine albumin/Creatinine ratio (µg/mg)	12 (2,29)	36 (2,263)	51 (2,257)	< 0.001
Urine protein/Creatinine ratio (mg/mg)	-	-	2.1 (0.1,8.6) (n = 34)	-
Weight reducing drugs*				
SGLT2i	52 (23.6)	41 (25.3)	65 (27.9)	0.58
GLP1-RA	5 (2.3)	1 (0.6)	1 (0.4)	0.13
No. of antihypertensive drugs*				
Nil	142 (64.5)	78 (48.1)	44 (20)	< 0.001
1	24 (10.9)	8 (4.9)	96 (43.6)	< 0.001
2	54(24.5)	76 (46.9)	63 (28.6)	< 0.001
≥3	-	-	17 (7.7)	-
Comorbidities*				
Hypertension	78 (35.5)	84 (51.9)	177 (80.5)	< 0.001
Dyslipidemia	131 (59.5)	112 (69.1)	175 (79.5)	< 0.001
Coronary artery disease	42 (19.1)	36 (22.2)	116 (52.7)	< 0.001
Neuropathy	85 (38.6)	99 (61.1)	166 (75.5)	< 0.001
Retinopathy	52 (23.6)	52 (32.1)	120 (54.5)	< 0.001

Median (Min, Max); * Values are n (%) LDL: low density lipoprotein, HDL: high density lipoprotein, TG: triglycerides; SGLT2i: sodium-glucose cotransporter 2 inhibitors; GLP1-RA: Glucagon like peptide 1-receptor agonist, BMI: body mass index, DM: diabetes mellitus, HbA1c: hemoglobin A1c, CKD: chronic kidney disease, eGFR: estimated glomerular filtration rate

usage of antihypertensive drugs with the advancing groups of CKD ($p < 0.001$). Comorbidities like hypertension, dyslipidemia, coronary artery disease, and microvascular complications like neuropathy and retinopathy were significantly higher in the high risk group than in other groups of kidney disease ($p < 0.001$).

Table 2 shows the demographic, clinical, and biochemical details of the study participants based on BMI categories. Participants in the obese category were older than the other two groups. The proportion of participants with obesity increased with the advancing categories of CKD ($p < 0.001$). The systolic and diastolic blood pressure in

Table 2: Demographic, clinical and biochemical details of the study participants based on BMI categories

Characteristics	Normal (18.5–22.9) (n = 145)	Overweight (23–24.9) (n = 127)	Obese (≥25) (n = 330)	P value between groups
Age (years)	55 (34,71)	57 (34,71)	59 (36,71)	0.006
M:F = 378:224	103:42	78:49	197:133	0.06
BMI (kg/m^2)	20.8 (17.7,22.9)	24.1 (23,25)	29.7 (25,71.4)	< 0.001
BMI in CKD stages*(kg/m^2)				
1	91 (62.8)	43 (33.9)	86 (26.1)	< 0.001
2	30 (20.7)	34 (26.8)	98 (29.7)	0.13
3	24 (16.6)	50 (39.4)	146 (44.2)	< 0.001
Blood pressure (mm Hg)				
Systolic	120 (90,180)	130 (90,190)	130 (77,200)	0.043
Diastolic	78 (50,100)	80 (60,110)	80 (50,130)	0.043
Duration of DM (years)	10 (2,32)	11 (2,44)	12 (2,44)	0.24
Smoking*	10 (6.9)	10 (7.9)	27 (8.2)	0.89
Current smoker	6 (4.1)	5 (3.9)	20 (6.1)	0.54
Ex-smoker	4 (2.8)	5 (4)	7 (2.1)	0.56
Alcohol consumption*	11 (7.6)	10 (7.9)	30 (9.1)	0.83
Plasma glucose (mg/dl)				
Fasting	144 (68,573)	146 (76,550)	147 (38,440)	0.91
Postprandial	245 (70,655)	238 (112,598)	234 (99,524)	0.22
HbA1c (%)	8.5 (5.6,15.7)	8.2 (5.6,15.9)	8.3 (5.3,16.4)	0.71
Total cholesterol (mg/dl)	164 (83,329)	161 (80,283)	154 (55,320)	0.036
LDL-C (mg/dl)	84 (36,167)	84 (33,166)	79 (16,182)	0.013
HDL-C (mg/dl)	49 (26,78)	50 (28,82)	50 (16,81)	0.53
TG (mg/dl)	106 (29,327)	115 (51,826)	119 (36,628)	0.009
eGFR (ml/min)	76 (21,107)	61 (21,106)	57 (15,134)	< 0.001
Serum creatinine (mg/dl)	1.1 (0.7,3)	1.1 (0.6,3.2)	1.2 (0.4,4.2)	< 0.001
Urine albumin-Creatinine ratio (µg/mg)	21 (2,250)	27 (2,240)	25 (2,263)	0.09
Urine protein- Creatinine ratio (mg/mg)	1.9 (1,2.8)	2.6 (1.8,6.1)	1.9 (0.1,8.6)	0.26
Weight reducing drugs*				
SGLT2i	27 (18.6)	33 (25)	98 (29)	0.05
GLP1-RA	4 (2.8)	-	3 (0.9)	0.15
No. of antihypertensive drugs*				
Nil	97 (66.9)	47 (37)	120 (36.4)	< 0.001
1	15 (10.3)	34 (26.8)	79 (23.9)	0.001
2	31 (21.4)	44 (34.6)	118 (35.8)	0.007
≥3	2 (1.4)	2 (1.6)	13 (3.9)	0.19
Comorbidities*				
Hypertension	48 (33.1)	81 (63.8)	210 (63.6)	< 0.001
Dyslipidemia	84 (57.9)	89 (70.1)	245 (74.2)	0.002
Coronary artery disease	36 (24.8)	44 (34.6)	114 (34.5)	0.091
Neuropathy	68 (46.9)	65 (51.2)	217 (65.8)	< 0.001
Retinopathy	52 (35.9)	45 (35.4)	127 (38.5)	0.77

Median (Min, Max); * Values are n (%), LDL: low density lipoprotein; HDL: high density lipoprotein; TG: Triglycerides; SGLT2i: sodium-glucose cotransporter 2 inhibitors; GLP1-RA: glucagon like peptide 1-receptor agonist; BMI: body mass index, DM: diabetes mellitus, HbA1c: Hemoglobin A1c, CKD: chronic kidney disease, eGFR: estimated glomerular filtration rate

the overweight and obese categories was higher than in participants with normal BMI. Although the levels of total cholesterol and low-density lipoprotein-cholesterol (LDL-C) were higher in people with normal BMI, the levels of triglycerides (TG) were significantly higher in the obese category ($p = 0.009$). Serum urea and creatinine increased with the increasing BMI. However, eGFR was significantly lower [57(15,134) vs. 76(21,107); $p < 0.001$] among the obese category as compared with the normal group. The number of antihypertensive drugs used was higher among people in the obese category, which was statistically significant ($p < 0.001$). Though there was a higher proportion of people in the obese category on weight-reducing drugs like SGLT2i, there was no statistical significance. Comorbidities like hypertension, dyslipidemia, and neuropathy were all significantly associated with obesity ($p < 0.001$). Though the proportion of participants with coronary artery disease and retinopathy was higher in the overweight and obese categories, there was no statistical significance between the groups.

Spearmann correlation analysis in the total study participants showed that age ($r = 0.13$, $p = 0.002$), serum creatinine ($r = 0.24$, $p < 0.001$), serum urea ($r = 0.17$, $p < 0.001$), and male gender ($r = 0.1$, $p = 0.03$) were positively associated with BMI, whereas eGFR showed significant negative correlation with BMI ($r = –0.30$, $p < 0.001$).

Table 3 shows the results of multivariate logistic regression analysis depicting the factors influencing the progression to CKD. In multivariate model, after adjustment for variables with $p < 0.05$ on univariate analyses, age category [≥ 60 years, OR(95% CI); 6.3(2.2–18); $p < 0.001$], increased BMI as overweight [3.6(2.1–6.3); $p < 0.001$] and obese [5.2(3.3–8.3); $p < 0.001$], smoking [4.2(1.7–10.2); $p = 0.002$], increased duration of diabetes in 5–15 years [2.3(1.2–4.5); $p = 0.013$], 16–25 years [4.8(2.2–10.4); $p < 0.001$], > 25 years [4.2(1.4–13); $p = 0.011$], higher SBP [1.01(1.0–1.03); $p = 0.02$], and HbA1c [1.2(1.1–1.3); $p < 0.001$] were independently associated with CKD progression.

Table 3: Results of multivariate logistic regression analysis [CKD low risk vs. CKD risk (moderate + high); as dependent variable]

Significant Variables	β	Odds ratio (OR) [95% confidence interval (CI)]	p value
Age categories (years)			
<50 (reference)			
51–59	0.50	1.6 (0.6–4.5)	0.334
≥60	1.84	6.3 (2.2–18.0)	<0.001
BMI categories (kg/m^2)			
Normal < 23 (reference)			
Overweight (23–24.9)	1.29	3.6 (2.1–6.3)	<0.001
Obese (≥25)	1.65	5.2 (3.3–8.3)	<0.001
Smoking (no vs. yes)	1.42	4.2 (1.7–10.2)	0.002
(Nonsmokers vs. Current + Ex-smokers)			
Duration of diabetes (years)			
<5 (reference)			
5–15	0.84	2.3 (1.2–4.5)	0.013
16–25	1.56	4.8 (2.2–10.4)	<0.001
>25	1.45	4.2 (1.4–13.0)	0.011
Systolic blood pressure (mmHg)	0.01	1.01 (1.0–1.03)	0.02
HbA1c (%)	0.19	1.2 (1.1–1.3)	<0.001

Nonsignificant variables: Gender and diastolic blood pressure, BMI: Body Mass Index, HbA1c: Hemoglobin A1c, CKD: Chronic Kidney Disease.

Discussion

Our study highlights the importance of obesity being an independent and major risk factor among the various risk factors that can influence the development and progression of CKD. Nearly 60% of the participants in our study were obese in the moderate and high CKD risk categories. Hyperglycemia, dyslipidemia, hypertension, increased albuminuria, smoking, and obesity are considered as some of the crucial risk factors for CKD.[18] Our study findings are consistent with the results of the Framingham cohort study, which reported that increased BMI can independently predict the risk of kidney disease.[19] The major difference is the assessment of kidney disease by using Kidney Disease Outcome Quality Initiative (KDOQI) guidelines where GFR was calculated using the Modification of Diet in Renal Disease (MDRD) equation. In our study, eGFR was calculated using CKD-EPI based on creatinine estimation, which is more widely accepted and routinely used in clinical practice to assess kidney function.[20] In this study, we used KDIGO guidelines to assess kidney function using eGFR and ACR.[15]

Increased BMI is considered a predominant risk factor for various metabolic disorders because people living with obesity are more likely to develop more diabetic microvascular complications.[21] In our study, obesity was more common among people with proteinuria and was also associated with the increased usage of antihypertensive drugs, which was similar to the findings of a cross-sectional study conducted in a tertiary nephrology center in Ireland.[22] Obesity can increase the progression of ESRD, especially for those in the high risk CKD group. In our study, overweight was associated four times and obesity five times with the advanced CKD categories. Among the Japanese population, people with increased BMI had a higher risk of developing ESRD after adjusting for proteinuria and hypertension.[13]

Kawamoto *et al*. suggested that increased BMI was consistently and strongly associated with a graded decrease in eGFR, similar to the findings of our study.[23] The glomerular filtration rate declines and the excretion

rate of urinary albumin increases, which can lead to the development of microalbuminuria and subsequently proteinuria contributing to the progression of DKD.[24] However, few studies state that obesity-related indices are associated with albuminuria,[25] but it was associated with proteinuria in our study; proteinuria was observed only among people with CKD—high risk group and more common among obese people.

Weight loss can occur as a sequel of CKD, and can even induce early mortality which is associated with a lower BMI.[26] A study from Northeast Thailand reports a negative association of BMI with CKD.[27] However, older age, hypertension, and microalbuminuria were positively associated with CKD, which is similar to our study. In a study conducted among the Chinese population, an inverse association was observed between CKD and BMI, wherein a low BMI was associated with an increased risk of development of CKD.[28] The inconsistent findings may be due to the design of the study and the population studied.

Hu *et al*. identified duration of diabetes, TG, SBP, glycated HbA1c, and BMI to be independent risk factors of diabetic nephropathy among people with T2D and obesity,[29] which are quite similar to the findings in our study. In addition, older age and smoking were also found to be independently associated with DKD in our study. Our study also revealed that complications such as coronary artery disease, neuropathy, and retinopathy were significantly higher in people in the moderate and high risk categories of CKD.

The impact of the obesity pandemic has contributed significantly to the advent of new drugs like SGLT2is and GLP1-RAs, the two important classes of glucose-lowering drugs that have an indispensable role in reducing the body weight, apart from their beneficial role on both the kidneys as well as the heart. Most of the participants in our study were on sulphonylureas, DPP4-inhibitors, and insulin rather than newer drugs like SGLT2is and GLP1-RAs. On average, only less than 30% of the participants were on SGLT2i in our study, so it becomes the need of the hour to encourage the use of these drugs to improve cardiorenal outcomes and thereby prevent complications. The probable reason for their infrequent usage could be the high cost of these novel drugs.

It is suggested that BMI cannot be used as a reliable indicator of CKD in people with T2D because it cannot differentiate between body fat and muscle.[30] Using BMI as a solitary method to measure the composition of the body can be quite misleading. The cross-sectional design of the study is another limitation as we could not assess other important predictors of CKD progression over time. The inaccuracies of BMI in advanced diabetic CKD where fluid accumulation is common cannot be denied. Measuring waist circumference or waist-to-height ratio can be considered to assess the influence of abdominal obesity among people with DKD. Some studies state that abdominal obesity is more closely associated with DKD than general obesity.[31] Most recently, the use of bioimpedance or dual X-ray absorptiometry (DEXA) scan can be considered for a better and more precise presentation of lean and body fat mass.[32] The main limitation of our study was the nonavailability of waist circumference measurement in our participants and we also did not perform the gold standard method to assess the body composition. The pack-years for smoking have not been recorded in this study, which is another limitation of the study. We have not measured Cystatin C to corroborate the observations of this study, which can also be considered as one of the limitations.

Intentional weight loss can provide considerable protection against the potential renal complications that develop in individuals with higher BMI. Regular and long-term tailored exercises play a crucial role in decelerating the development of hypertension, diabetes, and cardiovascular diseases as well as mortality among CKD patients.[33]

Our study highlights that overweight and obese individuals are four to five times at risk for the progression of CKD, which can have substantial implications in the development of complications. An integrated systematic and comprehensive approach to highlight the importance of reducing weight and promote the use of weight-reducing drugs like SGLT2i and GLP1-RA are required, which can prevent the adverse potential complications.

Acknowledgement

We thank Mr. S. Gurumoorthy for the help rendered during statistical analysis.

Conflicts of interest

There are no conflicts of interest.

References

1. International Diabetes Federation. IDF Diabetes Atlas, 10th edition. Brussels, Belgium; 2021. Available at: https://www.diabetesatlas.org [Last accessed on 2023 Apr 28].
2. Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, *et al*. Metabolic non-communicable disease health report of India: The ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). Lancet Diabetes Endocrinol 2023;11:474–89.
3. van Houtum WH. Barriers to the delivery of diabetic foot care. Lancet 2005;366:1678–9.
4. Boutari C, Mantzoros CS. A 2022 update on the epidemiology of obesity and a call to action: As its twin COVID-19 pandemic appears to be receding, the obesity and dysmetabolism pandemic continues to rage on. Metabolism 2022;133:155217.
5. Pradeepa R, Anjana RM, Joshi SR, Bhansali A, Deepa M, Joshi PP, *et al*. Prevalence of generalized & abdominal obesity in urban & rural India—the ICMR-INDIAB study (phase-I) [ICMR- NDIAB-3]. Indian J Med Res 2015;142:139–50.
6. Venkatrao M, Nagarathna R, Majumdar V, Patil SS, Rathi S, Nagendra H. Prevalence of obesity in India and its neurological implications: A multifactor analysis of a nationwide cross-sectional study. Ann Neurosci 2020;27:153–61.

7. Mohammedi K, Chalmers J, Herrington W, Li Q, Mancia G, Marre M, *et al*. Associations between body mass index and the risk of renal events in patients with type 2 diabetes. Nutr Diabetes 2018;8:7.
8. Martin WP, Bauer J, Coleman J, Dellatorre-Teixeira L, Reeve JLV, Twomey PJ, *et al*. Obesity is common in chronic kidney disease and associates with greater antihypertensive usage and proteinuria: Evidence from a cross-sectional study in a tertiary nephrology centre. Clin Obes 2020;10:e12402.
9. Liu W, Du J, Ge X, Jiang X, Peng W, Zhao N, *et al*. The analysis of risk factors for diabetic kidney disease progression: A single-centre and cross-sectional experiment in Shanghai. BMJ Open 2022;12:e060238.
10. International Diabetes Federation. IDF diabetes atlas. Available at: https://diabetesatlas.org/atlas-reports/report-year = 2023
11. Wuerzner G, Pruijm M, Maillard M, Bovet P, Renaud C, Burnier M, *et al*. Marked association between obesity and glomerular hyperfiltration: A cross-sectional study in an African population. Am J Kidney Dis 2010;56:303–12.
12. Thomson SC, Vallon V, Blantz RC. Kidney function in early diabetes: The tubular hypothesis of glomerular filtration. Am J Physiol Renal Physiol 2004;286:F8–15.
13. Iseki K, Ikemiya Y, Kinjo K, Inoue T, Iseki C, Takishita S. Body mass index and the risk of development of end-stage renal disease in a screened cohort. Kidney Int 2004;65:1870–6.
14. Hsu CY, McCulloch CE, Iribarren C, Darbinian J, Go AS. Body mass index and risk for end-stage renal disease. Ann Intern Med 2006;144:21–8.
15. Kidney Disease: Improving Global Outcomes (KDIGO) Diabetes Work Group. KDIGO 2022 clinical practice guideline for diabetes management in chronic kidney disease. Kidney Int 2022;102:S1–127.
16. Pan WH, Yeh WT. How to define obesity? Evidence-based multiple action points for public awareness, screening, and treatment: An extension of Asian-Pacific recommendations. Asia Pac J Clin Nutr 2008;17:370–4.
17. Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF, Feldman HI, *et al*. A new equation to estimate glomerular filtration rate. Ann Intern Med 2009;150:604–12.
18. Hussain S, Jamali MC, Habib A, Hussain MS, Akhtar M, Najmi AK. Diabetic kidney disease: An overview of prevalence, risk factors, and biomarkers. CEGH 2021;9:2–6. Available from: https://doi.org/10.1016/j.cegh.2020.05.016.
19. Fox CS, Larson MG, Leip EP, Culleton B, Wilson PW, Levy D. Predictors of new-onset kidney disease in a community-based population. JAMA 2004;291:844–50.
20. Kumpatla S, Soni A, Viswanathan V. Comparison of two creatinine based equations for routine estimation of GFR in a speciality clinic for diabetes. J Assoc Physicians India 2017;65:38–41.
21. Bae JP, Lage MJ, Mo D, Nelson DR, Hoogwerf BJ. Obesity and glycemic control in patients with diabetes mellitus: Analysis of physician electronic health records in the US from 2009-2011. J Diabetes Complications 2016;30:212–20.
22. Martin WP, Bauer J, Coleman J, Dellatorre-Teixeira L, Reeve JLV, Twomey PJ, *et al*. Obesity is common in chronic kidney disease and associates with greater antihypertensive usage and proteinuria: Evidence from a cross-sectional study in a tertiary nephrology centre. Clin Obes 2020;10:e12402.
23. Kawamoto R, Kohara K, Tabara Y, Miki T, Ohtsuka N, Kusunoki T, *et al*. An association between body mass index and estimated glomerular filtration rate. Hypertens Res 2008;31:1559–64.
24. de Jong PE, Verhave JC, Pinto-Sietsma SJ, Hillege HL. Obesity and target organ damage: The kidney. Int J Obes Relat Metab Disord 2002;26 Suppl 4:S21–4.
25. Ou YL, Lee MY, Lin IT, Wen WL, Hsu WH, Chen SC. Obesity-related indices are associated with albuminuria and advanced kidney disease in type 2 diabetes mellitus. Ren Fail 2021;43:1250–8.
26. Stevens J, Juhaeri None, Cai J. Changes in body mass index prior to baseline among participants who are ill or who die during the early years of follow-up. Am J Epidemiol 2001;153:946–53.
27. Zaman SB, Hossain N, Rahman M. Associations between body mass index and chronic kidney disease in type 2 diabetes mellitus patients: Findings from the Northeast of Thailand. Diabetes Metab J 2018;42:330–7.
28. Luk AO, So WY, Ma RC, Kong AP, Ozaki R, Ng VS, *et al*. Metabolic syndrome predicts new onset of chronic kidney disease in 5,829 patients with type 2 diabetes: A 5-year prospective analysis of the Hong Kong Diabetes Registry. Diabetes Care 2008;31:2357–61.
29. Hu F, Zhang T. Study on risk factors of diabetic nephropathy in obese patients with type 2 diabetes mellitus. Int J Gen Med 2020;13:351–60.
30. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr 2004;79:379–84.
31. Hu J, Yang S, Zhang A, Yang P, Cao X, Li X, *et al*. Abdominal obesity is more closely associated with diabetic kidney disease than general obesity. Diabetes Care 2016;39:e179–80.
32. Lin TY, Peng CH, Hung SC, Tarng DC. Body composition is associated with clinical outcomes in patients with non-dialysis-dependent chronic kidney disease. Kidney Int 2018;93:733–40.
33. Tamiya H, Tamura Y, Nagashima Y, Tsurumi T, Terashima M, Ochiai K, *et al*. Long-term tailor-made exercise intervention reduces the risk of developing cardiovascular diseases and all-cause mortality in patients with diabetic kidney disease. J Clin Med 2023;12:691.