Section: Physiology

Original Research Article

EFFECT OF BODY MASS INDEX AND WAIST CIRCUMFERENCE ON RENAL FUNCTION IN HYPERTENSIVE CHRONIC KIDNEY DISEASE

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ABSTRACT

Background: Objectives: Obesity and its associated cardiovascular, metabolic, and renal disorders have rapidly become a major threat to global health**.** Increased renal tubular sodium reabsorption impairs pressure natriuresis and plays an important role in initiating obesity hypertension. Increasing BMI and waist circumference were associated with reduced estimated glomerular filtration rate and CKD. The aim of the study was to investigate the association of Body Mass Index (BMI) and Waist Circumference (WC) with Hypertensive Chronic Kidney Disease. **Materials and Methods:** 40 study group with Hypertensive Chronic Kidney Disease (HT-CKD; Stage I–II) in the age group of between 18-50 yrs and 40 control group of same age were included in this study. Clinical history was recorded and physical examination including body weight, height, waist & hip circumferences were measured. Waist hip ratio (WHR)was calculated. Blood Pressure was recorded.24 hrs urine sample was collected for Albumin Excretion Rate (AER). Blood urea and Serum creatinine were estimated. Glomerular Filtration Rate (GFR) was estimated by using the Modification of Diet in Renal Disease (MDRD) formula. **Results:** There was significant negative correlation between WHR and e GFR level .Statistically significant correlation exist with WC and WHR for the parameters like e GFR, serum creatinine and AER. Statistically significant correlation between BMI with e GFR decline (negative correlation) was observed in the study group and also no significant correlation between BMI with serum creatinine level elevation and elevated AER level. **Conclusion:** This study revealed that in hypertensive chronic kidney disease (stage I-II) abdominal obesity was statistically significantly correlated with renal function decline than increase in BMI.

INTRODUCTION

A high body mass index is one of the strongest risk factors for new-onset Chronic Kidney Disease(CKD).Obesity is a chronic disorder with an increase in body weight and also an excess adipose tissue mass. A recent report by WHO stated that 1.6 billion people are overweight (BMI 25 – 30 Kg / m^2), 400 million people are obese (BMI > 30 Kg / m^2) worldwide.[1] Excess fat is stored in the abdominal region (upper body fat) or in the gluteal-femoral region (lower body fat). Intra abdominal fat cells are smaller than fat cells in subcutaneous tissue and are more sensitive to nutritional and hormonal factors than those from other regions.

Obesity is an individual's response to the environment or otherwise results from interaction of environmental and genetic factors. There are so many genes involved in the genesis of obesity such as genes controlling adipocyte differentiation, adipocyte specific adrenergic receptors, 'ob' gene that controls leptin, 'db' gene that controls receptor for leptin and mutation of 'ob' gene or 'db' gene results in obesity, 'agouti' gene which is normally expressed in skin and regulates the melanin production, mutation of which leads to obesity, 'fat' gene and mutation of carboxypeptidase – E results in obesity.[2,3,4]

Body adiposity classification based on BMI (Body Mass Index) – NIH (National Institutes of Health) and WHO (World Health Organization):,[3] Under weight - < 18.5 Kg/m2, Normal weight -18.5 – 24.9 Kg/m2 , Over weight - 25.0 – 29.9 Kg/m2 , Obesity Class I - 30.0 – 34.9 Kg/m2 , Obesity Class II - 35.0 – 39.9 Kg/m2 , Obesity ClassIII - ≥ 40.0 Kg/m2 . Body adiposity classification based on specific values for Waist Circumference (WC) - South Asians

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& Chinese- Male WC ≥ 90 cm(35 inches), Female ≥ 80 cm(31.5 inches).

Various studies suggested that excess weight gain is an important risk factor for hypertension.[5,6,7,8] INTERSALT study explained that for every 10 Kg in body weight there was 3 mmHg rise in systolic blood pressure and 2.3 mmHg rise in diastolic blood pressure (DBP).[3] WC,BMI,WHR are strongly related to blood pressure.[9,10] Various studies suggested that decline in weight leads to blood pressure reduction.[11-14] Hypertension is prevalent all over the world and target major organs of the body leading to coronary heart disease, cerebrovascular disease, kidney damage etc. Approximately 10 – 15 % population is suffered from hypertension. 5–20 % ESRD is due to hypertension.[15] Kidney is a target and cause of hypertension. SBP is mainly concerned with kidney damage.[16] NIH detection and follow up programmed observed 12 deaths by renal failure among 7825 mild hypertensive patients with DBP 90 – 104 mmHg which constitutes 0.15 % and 13 deaths by renal failure among 3175 patients with DBP > 105 mmHg that is 0.42 % deaths are due to renal failure.[17]

In individuals affected by obesity, a compensatory hyperfiltration occurs to meet the heightened metabolic demands of the increased body weight. The increase in intraglomerular pressure can damage the kidneys and raise the risk of developing CKD in the long term. CKD is defined as progressive and irreversible renal function loss. There are five stages of CKD based on e GFR values.CKD Stage I – II : e GFR ≥ 60 ml /min/1.73 m2; UAC ≥ 30 – 299 mg/g (microalbuminuria) and ≥ 300 mg / g (macroalbuminuria),CKD Stage III – IV : e GFR 15 – 59 ml / min / 1.73 m2 .Clinically microalbuminuria (a random urine ACR 30 -300 mg/g) or macroalbuminuria (a random urine ACR > 300 mg /g) are the early markers of kidney damage. They are not only indicators of renal damage but also markers of progression of kidney damage.[18-20]

Though obesity is a major risk factor for development of hypertension and CKD, less attention is paid to link between CKD and Obesity. Particularly visceral obesity / central obesity has strong link with CKD through two causes: hypertension and diabetes mellitus.

The good news is that obesity, as well as the related CKD, is largely preventable. Education and awareness of the risks of obesity and a healthy lifestyle, including proper nutrition and exercise, can dramatically help in preventing obesity and kidney disease.

Aim & Objective

The aim of the present study was to investigate the association of body mass index and waist circumference with hypertensive chronic kidney disease (stage I – II).The objective was to investigate whether waist circumference (WC) is more associated with GFR (renal function) reduction than body mass index (BMI) in hypertensive individuals with stage I – II chronic kidney disease (CKD).

MATERIALS AND METHODS

This case-control study was conducted in the Department of Physiology, Thanjavur Medical College Hospital. Forty normal healthy subjects and forty Hypertensive Chronic Kidney Disease (HT-CKD, Stage I–II) patients were recruited in the age group of between > 18 years and < 70 years. In the study group, Group 1- included patients with normal waist circumference and normal BMI, Group 2- included patients with high waist circumference and normal BMI, Group 3-included patients with normal waist circumference and high BMI. We obtained ethical committee approval and clearance. Informed written consent was obtained. The purpose of this study was explained clearly in their regional language. The history of the subjects was obtained and noted in a separate pro-forma.

Inclusion Criteria: Chronic kidney disease (Stage 1-2), Hypertension, Non-diabetic, Free from cardiovascular complications. **Exclusion Criteria :**History of any malignancy, History of or presence of inflammation, Presence of major cardiovascular event like stroke, myocardial infarction, acute ischaemic heart disease during last three months prior to the study, Diabetes mellitus, Heart failure, Heriditary renal disease

Materials and Methods: Detailed clinical history was evaluated and physical examination was done for every participants. Anthropometric measures like height (meters), weight (kilograms), waist circumference (centimeters) and hip circumference (centimeters) were measured. Waist circumference (in centimeters),[21] was measured at the midpoint between the lower margin of least palpable rib and the top of iliac crest at the end of normal expiration in standing position with arms at the sides and feet positioned closed together .The optimal waist circumference for males < 90 cm (35 inches) and for females < 80 cm (31.5 inches) for South-Asian ethnic groups. WC is an important measurement of central obesity. Hip circumference (in centimeters),[21] (HC) was measured around the widest portion of the buttocks with tape parallel to the floor. BMI (in Kg / m^2) was calculated by dividing weight in Kg by height in meter square ,Waist hip ratio (WHR) was calculated by dividing waist circumference by hip circumference and WHR is expressed in centimeters. The normal BMI range is 18.5 – 24.9 Kg / m^2.Bloodpressure (BP) is a dynamic physiological function and it varies with each heart beat. For measurement of blood pressure (BP in mmHg),[22] appropriate size cuff is important. Appropriate size cuff was selected based on mid arm circumference (MAC) of the participants. The blood pressure was measured in a quiet and relaxed setting after five minutes of rest with feet relaxed and flat on the floor. European Society of Cardiology Guidelines for management of Hypertension and Joint National Committee Guidelines for Detection, Evaluation and Management of Hypertension defined hypertension

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as SBP ≥ 140 mmHg and DBP ≥ 90 mmHg. For serum creatinine estimation (mg / dl),[23] 5 ml of venous blood was taken from the medial cubital vein in the anti-cubital fossa of the participants under sterile condition. Serum creatinine was measured by using Jaffe's kinetic method by using auto analyzer. The normal range of serum creatinine for males 0.6 – 1.2 mg/dl, and for females 0.4–1.0 mg/dl. The blood urea (in mg /dl),[23] was estimated by enzymatic(kinetic) method by using autoanalyzer. The normal range is 15–45 mg/dl.[24]

Then to assess renal function 24-hours urine was collected for the estimation of Albumin Excretion Rate (AER).The importance of the test was explained clearly to the participants. Heavy physical work and high protein diet intake were restricted on the previous day and the day of urine collection. Normal fluids and food intake was allowed. Fluid was not forced to avoid of very diluted urine. The participants were asked to empty the bladder completely on awakening and discard the first urine specimen and all the urine voided over the next 24 hours was collected in a large sterile container which contained a preservative (thymol) to reduce bacterial action or chemical decomposition. This 24 hours urine collection was delivered to the laboratory without delay. Normal AER is < 30 mg . 24-hours urine Albumin Excretion Rate (AER in mg/24hrs).[24]

Microalbuminuria is defined as 30 – 300 mg albumin excretion / 24 hours urine. Macroalbuminuria is defined as urine albumin excretion > 300 mg / day.

GFR was estimated by using MDRD formula,[18,25]

e GFR (ml/min/1.73m^2) = 186 × [Serum Creatinine level]$^{-1.154}$ × [Age]$^{-0.203}$ × [0.742 if patient is female] × [1.212 if the patient is black].

The normal average GFR is 125 ml / min. Normal glomerular filtration rate (GFR) ranges from 90 – 140 ml / min for adult male and ranges from 80 – 125 ml / min in females.

Thus the anthropometric and renal function parameters were measured to compare the effect of BMI and waist circumference on renal function decline in hypertensive chronic kidney disease.

RESULTS

Control group consisted forty healthy adults and the study group included forty hypertensive chronic kidney disease (stage I – II) patients. Statistical analysis was done by using the Statistical Package for Social Sciences (SPSS) X version. The results were analyzed by using student 't' test and ANOVA study. Data are expressed in mean with standard deviation. Correlation of BMI and WC, WHR with various renal function by using Pearson's Correlation test

Figure-1 shows the comparison of Weight, WC, HC, BMI in the study and the control groups.

Analysis of weight, body mass index, waist circumference, hip circumference, waist hip ratio of study groups by ANOVA study is shown in Table-1and Figure-2

The mean BMI of study group-3 (26.75 ± 1.06 Kg/m2) was more than that of study group-2 (23.11 ± 0.97 Kg/m2) and study group-1 (21.84 ± 1.18 Kg/m2) which was statistically significant P < 0.05.

The Waist Circumference of study group-2 (100.0 ± 3.86 cm) was statistically significantly (P < 0.05) higher than that of study group-1(81.93 ± 2.37 cm) and study group-3 (84.25 ± 2.22 cm).

The Hip Circumference and WHR of study group-2 were statistically significantly (P < 0.05) high than that of study group-1 and study group-3.

Figure-3 shows the comparison of SBP and DBP in the study and the control groups.

Analysis of blood pressure and renal functions between study group-1,group-2,group-3 by using ANOVA study are tabulated in Table 2.

The mean difference of SBP and DBP between study group-1,group-2,group-3 were not statistically significant

The mean e GFR of the control group was 139.57 ± 27.81 ml/min/1.73m2

The mean e GFR of study Group-1(78.71 ± 6.95 ml/min/1.73m2) was higher than study Group-2(61.36 ± 1.59ml/min/1.73m2) and study Group-3 (66.75 ± 3.62ml/min/1.73m2)which was statistically significant P < 0.05 . The mean e GFR of the study group-3 which had normal WC and high BMI was higher (statistically significant P < 0.05) than that of study group-2 which had high WC and normal BMI

The mean AER of the control group was 6.42 ± 3.38 mg/24 hrs.

The mean AER of study Group-2(115.29 ± 42.77 mg/24 hrs) was statistically significantly(P < 0.05) higher than study Group-3(66.50 ± 22.25 mg/24 hrs) and study Group-1(45.0 ± 6.61 mg/24 hrs). The mean e AER of the study group-2 which had high WC and normal BMI was higher (statistically significant P < 0.05) than that of study group-3 which had normal WC and high BMI.

At the same time renal function decline was more in study group-2 than study group-3.. High AER in study group-2 in this study indicates that renal damage was more in the study group with high WC that is renal function decline was more with central obesity than with high BMI. Analysis of blood urea, serum creatinine, e GFR and AER between and within the study and the control groups were done and is shown in figure-4

Table-3 shows the comparison of blood urea, serum creatinine levels, e GFR and AER of the study and control groups.

The mean serum creatinine of Group-2 (1.16 ± 0.04mg/dl) was higher than Group-3 (1.08 ± 0.05mg/dl) and Group-1(0.93 ± 0.08mg/dl).

The mean blood urea level of Group-3 (55.58 ± 5.11 mg/dl) was higher than Group-1 (47.79 ± 5.24 mg/dl) and Group-2(50.57 ± 5.84 mg/dl).

Table-4 shows Correlations of BMI & Waist circumference, WHR with renal parameters & blood. Statistically significant correlation between BMI with e GFR decline (negative correlation) was observed in the study group and also no significant

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correlation between BMI with serum creatinine level elevation and elevated AER level. There is positive correlation between BMI and blood Urea level. Statistically significant negative correlation between WC with e GFR, positive correlation between WC and serum creatinine level and positive correlation between WC and AER were observed in the study group in this present study.

There are significant negative correlation between WHR and e GFR level, positive correlation between WHR and serum creatinine level and also positive correlation between WHR and AER level were observed in the study group.

Table 1: Analysis of Weight, BMI, WC, HC, WHR between Study Groups By Anova Study

Parameters	Mean	S.D	SS	Df	MS	F	Statistical inference
WEIGHT(Kg)							
Between Groups			845.215	2	422.67	54.869	.000<0.05 Significant
Group-1(N=14)	56.29	2.091					
Group-2(N=14)	57.64	1.216					
Group-3(N=12)	66.92	4.358					
Within Groups			284.976	37	7.702		
BMI(Kg/m^2)							
Between Groups			164.572	2	82.287	71.267	.000<0.05 Significant
Group-1(N=14)	21.8429	1.17912					
Group-2(N=14)	23.1143	.97417					
Group-3(N=12)	26.7500	1.05787					
Within Groups			42.721	37	1.155		
WC(cm)							
Between Groups			2664.441	2	1332.220	153.455	**.**000<0.05 Significant
Group-1(N=14)	81.93	2.369					
Group-2(N=14)	100.00	3.863					
Group-3(N=12)	84.25	2.221					
Within Groups			321.215	37	8.681		
HC(cm)							
Between Groups			420.180	2	210.090	10.180	.000<0.05 Significant
Group-1(N=14)	93.36	5.198					
Group-2(N=14)	100.86	4.639					
Group-3(N=12)	95.33	3.473					
Within Groups			763.595	37	20.638		
WHR							
Between Groups			.150	2	.075	39.799	.000<0.05 Significant
Group-1(N=14)	.8771	.03750					
Group-2(N=14)	1.0064	.05930					
Group-3(N=12)	.8792	.02275					
Within Groups			.070	37	.002		

Table 2: Analysis of Blood pressure and renal functions between Study Groups by Anova Study

Parameters	Mean	S.D	SS	Df	MS	F	Statistical inference
SBP(mmHg)							
Between Groups			14.648	2	7.324	.403	.671>0.05 Not Significant
Group-1(N=14)	141.57	5.331					
Group-2(N=14)	142.71	3.646					
Group-3(N=12)	141.33	3.447					
Within Groups			672.952	37	18.188		
DBP(mmHg)							
Between Groups			16.757	2	8.379	1.196	.314>0.05 Not Significant
Group-1(N=14)	90.14	2.983					
Group-2(N=14)	90.57	2.875					
Group-3(N=12)	89.00	1.809					
Within Groups			259.143	37	7.004		
Within Groups			.143	37	.004		
eGFR(ml/min/1.73m^2)							
Between Groups			2199.57	2	1099.78		**.000<0.05 Significant**
Group-1(N=14)	78.71	6.955					
Group-2(N=14)	61.36	1.598					
Group-3(N=12)	66.75	3.621					
Within Groups			806.321	37	21.792		
AER(mg/24 hrs)							
Between Groups			36144.043	2	18072.021		**.000<0.05 Significant**
Group-1(N=14)	45.00	6.610					
Group-2(N=14)	115.29	42.776					
Group-3(N=12)	66.50	22.253					
Within Groups			29801.857	37	805.456		

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Table 3: Descriptive Analysis of Urea,Creatinine,e GFR, AER in Study and Control Groups

Parameters	Mean	S.D	T	Df	Statistical inference
Urea(mg/dl)					
Study group (N=40)	51.10	6.172	24.991	78	.000<0.05 Significant
Control group(N=40)	21.33	4.323			
Creat(mg/dl)					
Study group (N=40)	1.0620	.11654	20.510	78	.000<0.05 Significant
Control group(N=40)	.5823	.09113			
e GFR(ml/min/1.73m^2)					
Study group (N=40)	69.05	8.779	-15.291	78	.000<0.05 Significant
Control group(N=40)	139.57	27.817			
AER(mg/24 hrs)					
Study group (N=40)	76.05	41.121	9.496	78	.000<0.05 Significant
Control group(N=40)	6.42	3.38			

Table 4: Correlations of BMI & Waist circumference, WHR with renal parameters & blood pressure

Parameters	BMI - Correlation value	BMI -Statistical inference	WC - Correlation value	WC - Statistical inference	WHR- Correlation value	WHR- Statistical inference
CREAT	.301	p>0.05 Not Significant	.675(**)	p<0.01 Significant	.507(**)	p<0.01 Significant
e GFR	-.320(*)	p<0.05 Significant	-.689(**)	p<0.01 Significant	-.535(**)	p<0.01 Significant
AER	-.002	p>0.05 Not Significant	.685(**)	p<0.01 Significant	.601(**)	p<0.01 Significant
UREA	.356(*)	p<0.05 Significant	-.073	p>0.05 Not Significant	.101	p>0.05 Not Significant
SBP	-.139	p>0.05 Not Significant	-.060	p>0.05 Not Significant	.230	p>0.05 Not Significant
DBP	-.273	p>0.05 Not Significant	.176	p>0.05 Not Significant	.152	p>0.05 Not Significant

**** Correlation is significant at the 0.05 level for BMI & 0.01 for WC and WHR***

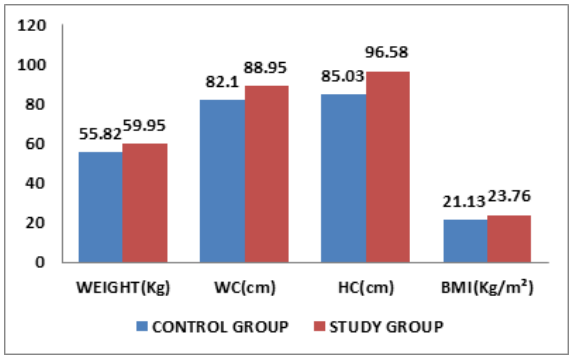

Figure 1: Comparison of Weight, WC, HC, BMI between control and study groups

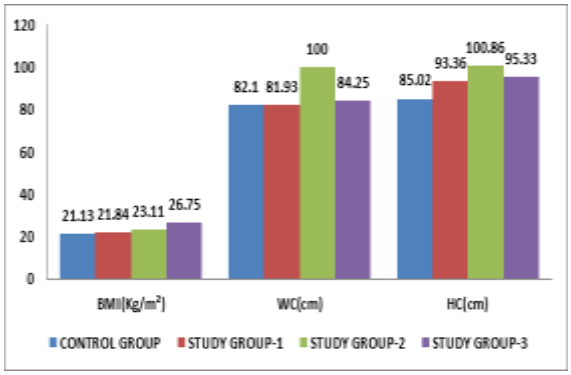

Figure 2: Comparison of BMI, WC, HC between control and Group-1, Group-2 and Group-3 of study groups

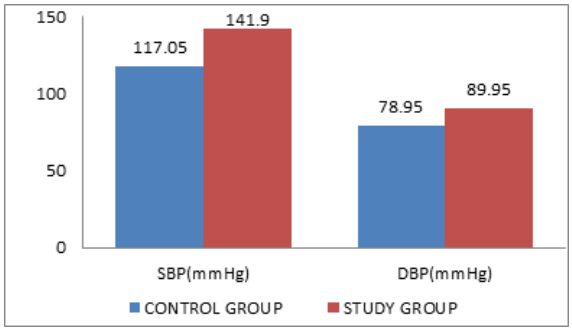

Figure 3: Comparison of SBP, DBP between control & study groups

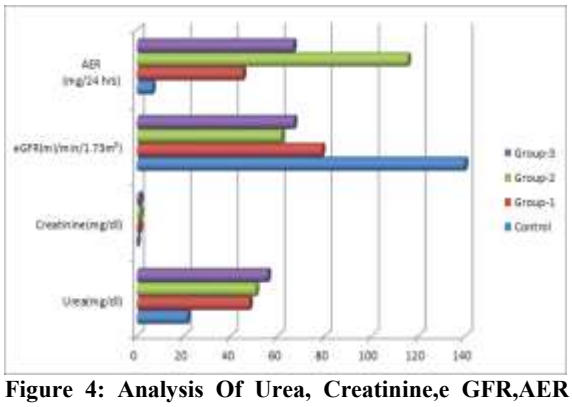

Figure 4: Analysis Of Urea, Creatinine,e GFR,AER between Study Groups and control

DISCUSSION

Obesity is a risk factor for cardiovascular diseases, hypertension, diabetes mellitus etc. But awareness of association between obesity and kidney damage is

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less. Prevalence of obesity is rapidly increasing now a days. In obesity there is increased sympathetic nervous system activity, R-A-A-S activation, renal compression by accumulation of fat around the kidney. Increased adrenergic activity plays major role in the development of hypertension in obesity. Elevated systemic BP → transmitted to glomerulus → Glomerular hypertension → ↑ glomerular damage.[4] Obesity may directly cause a specific form of glomerulopathy (focal and segmental glomerulosclerosis). Higher BMI appears associated with the presence and development of low estimated glomerular filtration rate (eGFR), with more rapid loss of e GFR over time, and with the incidence of ESRD. Elevated BMI levels, class II obesity and above, have been associated with more rapid progression of CKD in patients with pre-existing CKD. Low level of BMI thresholds for overweight and obesity have been proposed for Asians especially China and India.

For some obesity complications, the regional distribution of fat rather than the absolute excess adipose tissue amount appears to be important. Abdominal obesity is closely associated with type -2 DM, metabolic syndrome, cardiovascular diseases, kidney damage etc.[4,15,26] BMI provides an estimate body fat .Central obesity, which is measured by waist circumference (WC) and waist hip ratio(WHR), is a better predictor than BMI to reflect visceral fat. WC was a strong predictor for both abdominal and non-abdominal fat.

So to avoid all these complications of obesity particularly of central obesity, like hypertension and CKD, we must make awareness about physical fitness and importance of maintenance of normal body weight through regular physical activity and health education about healthy food habits .This study was done to investigate the association of BMI and WC with hypertensive-CKD (stage I – II).So by simple health education about healthy diet, physical activity, life style modification we can prevent excess body fat accumulation and related complications such as hypertension and kidney damage.

In our study, there was strong statistically significant correlation of WC and WHR which are the indicators of central or abdominal obesity, with renal function reduction in stage I & II HT-CKD patients. And we observed that both high BMI and increased WC (visceral / central obesity) were associated with reduced renal function (reduced e GFR, elevated serum creatinine level, high AER, Blood Urea) ; but in high waist circumference more decline in kidney function was observed than other groups

Hyunju oh et al,[27] In their study "Waist Circumference(WC), not BMI ,is associated with renal function decline in Korean population. Janssen et al: (28) studied about "WC and not BMI explains obesity related health risk"."Relationship between Abdominal Obesity (AO) and Microalbuminuria (MA) in elderly " was studied by Mohmoud AH and Taha HM.[29] AO is strongly associated with Microalbuminuria in Egyptian elderly. Seok Huikang et al,[30] studied " Association of visceral fat area(VFA) with CKD and Metabolic syndrome (MS) risk in the general population. "Prevalence of Renal Insufficiency (RI) in individuals with hypertension and obesity / overweight: the FATH study" conducted by Pablo Gomez et al,[31] in 5,585 pateints with hypertension and GFR ≥ 25 Kg / m2. They observed that the prevalence of RI was greater in patients with abdominal obesity. Pinto Sietsma SJ et al,[32] in their study they observed microalbuminuria (MA) was associated with abnormal renal function in non-diabetic individuals.

Due to its close association with diabetes and hypertension, obesity is a crucial contributor to renal illnesses; also, obesity and excess weight pose a severe hazard to the development of chronic kidney diseases. Obesity affects the progression of stable kidney disease because it increases the risk of developing diabetic nephropathy, hypertensive nephron sclerosis, and focal and segmental glomerulosclerosis, among other conditions. Renal hemodynamic, structural, and histological alterations are linked to obesity. Adipokines, such as leptin, adiponectin, tumor necrosis factor-, monocyte chemoattractant protein-1, transforming growth factor-1, and angiotensin-II, are produced by active adipose tissue .These substances damage the peripheral tissues. The therapeutic targets for BMI and body composition warrant more precise definition in NDD-CKD and dialysis patients, and in particular those participating in kidney transplantation (recipients and living donors). Lastly, given the compelling evidence for obesity as risk factor for incident CKD and ESRD, further studies are needed to determine the safety and effectiveness of weight loss interventions (e.g., pharmacologic, surgical, and physical activity) upon the development and progression of kidney disease.

Limitations of our study

Use of single measurement of creatinine, Need of large sample size,*Absence of gold standard for body fat assessment (VFA),*Need to follow for years to know further renal function decline due to the effect of central obesity. In our study both obesity and hypertension co-existed, so it is difficult to establish significance of these two in the development of kidney damage

CONCLUSION

In our study, we found that abdominal obesity was statistically significantly correlated with renal function decline than increase in the BMI .Abdominal obesity can be easily measured by simple methods like waist circumference, hip circumference and waist hip ratio with easily available instrument such as inch-tape .The physicians can advice the obese patients to reduce the weight and suggest the patients that weight reduction can reduce the incidence of hypertension and their complications. This study suggests, the importance of controlling the

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obesity, especially in hypertensive chronic kidney disease with initial stages of CKD and consequetively preventing the CKD progression.

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