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Clinical profile and comparison of renal insufficiency in obese and non-obese hypertensive patients: A cross-sectional comparative study

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ABSTRACT

Background: Renal failure can stem from various conditions, and detecting biomarkers associated with obesity-related disorders is crucial for early identification of the risk of renal failure and timely intervention in obese individuals. Though different studies have depicted a relationship between obesity, metabolic abnormalities and cardiovascular outcomes but there is scarcity of evidence in the Indian population as there are no published data comparing this condition in hypertensive patients with or without excess BMI. Thus, we designed a prospective study to determine the prevalence of renal insufficiency in a tertiary care hospital by correlating various socio demographic and clinical risk factors.
Materials and Methods: A total of 100 subjects were enrolled in the study as per the inclusion and exclusion criteria. We conducted a clinical and biochemical profiling to evaluate renal insufficiency in subjects.
Results: The mean duration of HTN in obese hypertensive patients were 7.50 ± 3.86 years and in non-obese hypertensive patients it was 7.22 ± 4.09 years. The mean systolic blood pressure recorded in obese and non-obese patients was 136.36 ± 12.17 and 138 ± 14.64 mm Hg respectively. Prevalence of renal insufficiency in obese and non-obese patients was found 30% and 20% respectively.
Conclusion: We can conclude on the basis of obtained results, hypertension is one of the major risk factors in the development of renal insufficiencies, and the presence of obesity may further increase its prevalence rate of kidney diseases.

1. Introduction

Obesity is described as abnormal or excessive fat accumulation that may have a negative impact on health.[1] Obesity not only increases the risk of cardiovascular events, but it also plays a key role in the development and progression of pre-existing chronic kidney disease (CKD).[2] It is regarded as a risk factor for essential hypertension (HTN), diabetes, and other concomitant diseases associated with the development of CKD.[3] Obesity causes HTN due to a variety of factors, including an increase in renal tubular sodium reabsorption, which impairs pressure natriuresis and causes volume expansion via activation of the sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone (RAS) system, as well as physical compression of the kidneys, especially when visceral adiposity is high.[4] Although there is evidence that the steady growth in CKD prevalence is strongly connected with growing obesity, the link between obesity and CKD receives far less attention. Previous research has shown that a higher prevalence of renal insufficiency (RI) is associated with higher blood pressure.[5] Obesity also adds to the fast-rising prevalence of chronic renal failure (CRF) in both men and women. Overweight (BMI > 25 kg/m^2) at age 20 was

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related with a threefold increased incidence of CRF. Obesity (BMI >30) in men and morbid obesity (BMI >35) in women at any point in their lives was attributed to a three- to four-fold increase in risk. The increased risk of CRF in obese adults appears to be primarily caused by a high prevalence of HTN and/or type 2 diabetes.[6]

To maintain sodium homeostasis, an increase in renal tubular sodium reabsorption is accompanied by a compensatory increase in glomerular filtration rate (GFR) in obese people. Chronic obesity leads to renal vasodilation and glomerular hyperfiltration, as the initial rise in GFR compensates for maintaining salt balance. However, metabolic disorders, along with a significant increase in arterial pressure, can induce renal damage, resulting in a drop in GFR.[4] Obesity was found to have a substantial connection with HTN, diabetes, and dyslipidemia, all of which act synergistically to affect a favourable renal outcome. Diabetes patients with increased blood pressure and microalbuminuria eventually advances to proteinuria and a deterioration in renal function. According to the literature, healthy obese people [BMI >30 kg/m2] were at a higher risk of developing proteinuria and losing renal function after unilateral nephrectomy than lean subjects.[7,8] To accurately detect the risk of renal failure associated with obesity-related disorders, identifying biomarkers for various conditions is crucial. Various research has shown a link between obesity, metabolic abnormalities, and cardiovascular outcomes, however there is a lack of evidence in the Indian population because there is no published data comparing this condition in hypertension patients with or without excess BMI. The current prospective study aims to assess the occurrence of renal insufficiency in a tertiary care hospital by linking socio-demographic and clinical risk variables. Furthermore, in a tertiary care hospital, we evaluated clinical and biochemical profiles of obese and non-obese hypertension patients.

2. Material and Methods

2.1. Study design

The present prospective, single-centre, observational, cross-sectional study was conducted at Department of General Medicine, School of Medicine, D Y Patil Deemed to be University Navi Mumbai after seeking ethical clearance from Institutional Ethics Committee (PDDYPMC/Ethics/PG Dissert-22/2015, Dated: 15-04-2015). All ethical principles laid out by ICH GCP and ICMR guidelines were strictly adhered during the entire conduct of the study. The study population included a total of 100 patients attending the HTN OPD in the tertiary care hospital. The study was conducted over a period of 12 months.

2.2. Eligibility criteria and study groups

All the consenting subjects within the age group of 18–75 years with a BMI of 18.5-24.9 kg/m2 and a waist circumference of < 88 cm (females) and < 102 cm (males) were included in the control arm, whereas subjects with a BMI ≥ 25 kg/m2 and a waist circumference ≥ 88 cm (females) and ≥ 102 cm (males) were included in the case arm. Also, an important criterion was that subjects should be taking anti-hypertensive medications for at least 1 year in the case arm category. The study groups comprised a total of 100 subjects and were divided into the following two groups,

Group 1: 50 obese hypertensive patients.

Group 2: 50 non-obese hypertensive patients.

All the non-consenting subjects with age less than 18 years and showing up presence of other co-morbidities during enrolment to study were excluded from the study. Also, women of child-bearing age were excluded from the study.

2.3. Study procedure

BP measurements were performed using a validated semiautomatic sphygmomanometer with a cuff size adapted to the arm circumference. Patients were considered to have HTN based on their previous diagnosis and/or taking antihypertensive treatment. The presence of abdominal obesity was defined by a waist circumference ≥ 88 cm in females and ≥ 102 cm in case of males. All socio-demographic factors, medical history, habits and the presence of any previous cardiovascular disease (ischemic heart disease, heart failure, stroke, or peripheral arterial disease and any manifestation of chronic renal disease (renal insufficiency, haematuria, or proteinuria) were noted in the prescribed proforma. Baseline parameters including glucose, HbA1c, cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, serum creatinine, BUN, serum uric acid levels and examination of urine microalbuminuria were analyzed in the central laboratory, within 7 days of obtaining the informed consent. GFR was calculated using the simplified Modification of Diet in Renal Disease (MDRD) and CockroftGault equations. Renal insufficiency was defined in the protocol as a GFR by MDRD < 60 ml/min per 1.73 m^2 and the degree of renal function was graded as per the National Kidney Foundation guidelines.[9]

3. Results

The present study aimed to compare demographic and body composition characteristics of obese and non-obese individuals. A total of 50 individuals (33 males and 17 females) with a mean age of 56.98 ± 9.90 years were classified as obese, while 50 individuals (29 males and 21 females) with a mean age of 54.70 ± 10.44 years were classified as non-obese (Table 1). The results showed that

the mean body weight of the obese group was significantly higher (84.30±8.74 kgs) compared to the non-obese group (67.56 ± 9.03 kgs) (p< 0.0001). The mean height of the obese group (1.66 ± 0.08 m) was also significantly lower compared to the non-obese group (1.71 ± 0.09 m) (p= 0.0019). Additionally, the mean Body Mass Index (BMI) of the obese group (30.68±2.36) was significantly higher compared to the non-obese group (23.04±1.87) (p< 0.0001). The mean waist circumference of the obese group (100.04 ± 6.65 cm) was also significantly higher compared to the non-obese group (85.48 ± 7.71 cm) (p< 0.0001). The Table 1 shows a comparison of complaints between obese and non-obese individuals. A chi-square p value of 0.4876, suggests that there is no significant difference in the occurrence of these complaints between obese and non-obese individuals.

Table 2 represents the mean duration of hypertension that was found to be 7.50 ± 3.86 years for the obese group and 7.22 ± 4.09 years for the non-obese group with a p value of 0.7255, indicating no significant difference between the two groups. 60% of the obese individuals had a mixed diet while 40% followed a vegetarian diet, while 46% of the non-obese individuals had a mixed diet and 54% followed a vegetarian diet with a p value of 0.2293, also indicating no significant difference. 100% of both obese and non-obese individuals had normal sleep patterns. 14% of the obese individuals and 6% of the non-obese individuals had a history of smoking, with a p value of 0.3178, showing no significant difference between the two groups. Similarly, 14% of the obese individuals and 10% of the non-obese individuals had a history of alcoholism, with a p value of 0.7583, indicating no significant difference between the two groups. No individuals fromeither group had a history of other addictions. Both obese and non-obese individuals had normal body temperatures.

Further the clinical parameters between obese and non-obese individuals were also compared.

The results showed that there was no statistically significant difference in SBP (p=0.5439) and DBP (p=0.523) between the two groups (Table 3). The pulse rate in non-obese individuals was higher compared to obese individuals, but the difference was not statistically significant (p= 0.0634) whereas FBS was higher in obese individuals compared to non-obese individuals, and was revealed to be statistically significant (p=0.0123). HBA1c, total cholesterol and triglycerides was higher in obese individuals compared to non-obese individuals, and this difference was statistically significant. HDL-C was lower in obese individuals compared to non-obese individuals, contradicting the LDL-C levels that was found to be elevated in non-obese individuals. This difference was statistically significant. Serum urea was higher in obese individuals compared to non-obese individuals, and this difference was statistically significant (p=0.047) (Table 3). There was no statistically significant difference in serum creatinine, serum uric acid and respiratory rate when compared between the two groups. The prevalence of proteinuria was higher in obese individuals compared to non-obese individuals, but the difference was not statistically significant (p=0.1931). The prevalence of pallor, oedema or any abnormality in respiratory system, cardiovascular system, PA system and CNS system was not statistically significant between the two groups (Table 3). These findings suggest that obesity is associated with several metabolic and cardiovascular risk factors.

Table 4 presents glomerular filtration rate (GFR), abdominal USG and prevalence of renal insufficiency in obese and non-obese subjects. The GFR was found to be 85.47 ± 38.26 ml/min for the obese group and 87.60 ± 35.64 ml/min for the non-obese group, with a p value of 0.7742, indicating no significant difference between the two groups. The abdominal ultrasound findings showed that 10% of the obese individuals had echogenic kidneys compared to 4% of the non-obese individuals, with a p value of 0.079. 10% of the obese individuals and 14% of the non-obese individuals had a fatty liver, while 14% of the obese individuals and 6% of the non-obese individuals had echogenic kidney and fatty liver. 20% of the obese individuals had cirrhosis compared to 12% of the non-obese individuals. 8% of the obese individuals and 10% of the non-obese individuals had normal abdominal ultrasound findings. The prevalence of renal insufficiency was 30% in the obese group and 20% in the non-obese group, with a p value of 0.3556, showing no significant difference between the two groups.

The Table 5 presents the electrocardiogram (ECG) characteristics of 50 obese and 50 non-obese subjects. The data shows that the incidence of arrhythmias was higher in the obese group (14%) compared to the non-obese group (6%), but the difference was not statistically significant (p=0.0939). Among other ECG characteristics, left ventricular hypertrophy (LVH) was the most prevalent, with a similar incidence in both groups (36%). The incidence of first-degree heart block, left bundle branch block (LBBB), right ventricular hypertrophy (RVH), ST-T changes, and T-wave depression were also comparable between the two groups. However, a higher percentage of non-obese subjects had a normal ECG (26%) compared to obese subjects (6%).

The results of the correlation analysis between various risk factors and the presence of renal insufficiency are presented in the Table 5. The correlation coefficient and the p-value are reported for each characteristic. The results showed a significant negative correlation between the duration of hypertension (HT), systolic blood pressure (SBP), pulse, respiratory system, and fasting blood sugar (FBS) with renal insufficiency (p<0.05). However, no significant correlation was found between age, gender, type of diet, smoking, alcohol intake, built, diastolic blood pressure (DBP), respiratory rate, cardiovascular system,

Table 1: Demographic data and complaints frequency in non-obese and obese subjects

Parameters	Obese	Non-obese	p value
Mean age (years)	56.98±9.90	54.70±10.44	0.5321
Gender			
Male	33 (66%)	29 (58%)	
Female	17 (34%)	21 (42%)	
Mean Body Weight (kgs)	84.30±8.74	67.56±9.03	<0.0001
Mean Height (m)	1.66±0.08	1.71±0.09	0.0019
Mean BMI	30.68±2.36	23.04±1.87	<0.0001
Mean Waist Circumference (cms)	100.04±6.65	85.48±7.71	<0.0001
Complaints			
Abdominal Pain	5	2	
Breathlessness	5	7	
Chest Pain	7	7	
Fever	0	7	
Giddiness	7	3	
Headache	10	6	
Palpitation	4	5	
Syncope	2	0	
Vomiting	2	3	
Anorexia	1	1	
Back Pain	1	1	0.4876
Cough Expectoration	1	1	
Diabetic Foot	1	0	
Diarrhea	0	1	
Enteritis	1	1	
Gastroenteritis	1	0	
Hematemesis	1	1	
Joint pain	0	1	
Oliguria	0	1	
Pedal Oedema	0	1	
Polyuria	1	1	

Table 2: History records of obese and non-obese subjects involved in the study

Parameters		Obese	Non-obese	p value
Mean Duration of HTN (years)		7.50±3.86	7.22±4.09	0.7255
Normal Sleep Pattern		50 (100%)	50 (100%)	
Type of Diet	Mixed	30 (60%)	23 (46%)	0.2293
	Only Vegetarian	20 (40%)	27 (54%)	
History of Smoking	Yes	7 (14%)	3 (6%)	0.3178
	No	43 (86%)	47 (94%)	
History of Alcoholism	Yes	7 (14%)	5 (10%)	
	No	43 (86%)	45 (90%)	0.7583

abdomen, central nervous system, body weight, height, body mass index (BMI), abdominal circumference and renal insufficiency (p>0.05).

4. Discussion

Hypertension is the cause of major life-threatening complications like congestive cardiac failure, ischaemic and haemorrhagic stroke, peripheral arterial disease, and renal failure. Obesity is one of the risk factors of hypertension.[10,11] An increase in BMI increases the risk of cardiovascular and renal complications especially in patients approaching obesity with BMI of 25 or more. Obesity is associated with increased cardiovascular risk and affects the renal system.[12,13] Therefore, we evaluated the prevalence of renal insufficiencies in obese and non-obese hypertensive patients. The mean age of the patient enrolled in our study was comparable to the previous published articles.[5,14] From the literature, it has been observed that a greater number of younger patients are

Table 3: Hemodynamic, biochemical parameters & body system examination of obese and non-obese subjects.

Parameters		Obese	Non-obese	P value
SBP (mmHg)		136.36±12.17	138±14.64	0.5439
DBP (mmHg)		86.60±6.75	85.72±6.98	0.523
Pulse (bpm)		81.04±10.21	84.66±9.04	0.0634
FBS (mg/dL)		117.74±54.94	95.88±25.63	0.0123
HBA1c (%)		4.86±1.89	4.18±1.14	0.0316
Total Cholesterol (mg/dL)		237.64±39.27	202.86±62.53	0.0012
Triglycerides (mg/dL)		134.28±29.64	105.78±31.27	<0.0001
HDL Cholesterol (mg/dL)		39.04±7.51	33.90±7.93	0.0012
LDL Cholesterol (mg/dL)		115.93±18.03	99.36±16.30	<0.0001
Serum Urea (mg/dL)		41.07±14.94	34.89±15.80	0.047
Serum Creatinine (mg/dL)		1.34±0.72	1.32±1.63	0.9558
Serum Uric Acid (mg/dL)		6.26±1.16	5.60±0.90	0.0017
Respiratory rate (breaths per min)		20.46±2.70	20.26±2.82	0.7182
Proteinuria	Present	12 (24%)	6 (12%)	0.1931
	Absent	38 (76%)	44 (88%)	
Pallor	Yes	6 (12%)	4 (8%)	0.7407
	No	44 (88%)	46 (92%)	
Oedema	Yes	7 (14%)	7 (14%)	1
	No	43 (86%)	43 (86%)	
Respiratory system	Normal	44 (88%)	43 (86%)	0.7662
	Abnormal	6 (12%)	7 (14%)	
Cardiovascular System	Normal	43 (86%)	45 (90%)	0.7583
	Abnormal	7 (14%)	5 (10%)	
PA System	Normal	47 (94%)	45 (90%)	0.715
	Abnormal	3 (6%)	5 (10%)	
CNS System	Normal	49 (98%)	50 (100%)	1
	Abnormal	1(2%)	0	

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, FBS: Fasting blood sugar, PA: Per abdomen system and CNS: Central nervous system

Table 4: GFR, Abdominal USG and prevalence of renal insufficiency in obese and non-obese subjects.

Parameters		Obese	Non-obese	P value
GFR (ml/min)		**85.47±38.26**	**87.60±35.64**	**0.7742**
Abdominal USG	Echogenic Kidneys	5 (10%)	2 (4%)	0.079
	Fatty liver	5 (10%)	7 (14%)	
	Hepatomegaly	7 (14%)	7 (14%)	
	Kidney Disease	0	7 (14%)	
	Echogenic kidney and fatty liver	7 (14%)	3 (6%)	
	Cirrhosis	10 (20%)	6 (12%)	
	Normal	4 (8%)	5 (10%)	
Prevalence of Renal Insufficiency	Renal Insufficiency	15 (30%)	10 (20%)	0.3556
	No Renal Insufficiency	35 (70%)	40 (80%)	
	Prevalence	30%	20%	

GFR: Glomerular filtration rate

Table 5: Electrocardiogram (ECG) characteristics of subjects

Characteristics	Obese	Non-obese	p value
Arrhythmia	7 (14%)	3 (6%)	
1^{st} degree heart block	2 (4%)	0	
LBBB	6 (12%)	4 (8%)	
LVH	18 (36%)	18 (36%)	0.0939
RVH	1 (2%)	1 (2%)	
ST-T changes	10 (20%)	8 (16%)	
T-wave depression	3 (6%)	3 (6%)	
Normal	3 (6%)	13 (26%)	
Total	50 (100%)	50 (100%)	

LBBB: Left bundle branch block, LVH: Left ventricular hypertrophy, RVH: Right ventricular hypertrophy

Table 6: Correlation between risk factors and renal insufficiency

Characteristic/ Body Systems	Correlation Coefficient	p value	Correlation (Yes/ No)
Age	-0.1028	0.3089	No
Gender	-0.1513	0.133	No
Duration of HT	-0.3557	0.0003	Yes
Type of Diet	0.0139	0.8901	No
Smoking	0.0525	0.6035	No
Alcohol intake	-0.122	0.2266	No
SBP	-0.2001	0.0459	Yes
DBP	-0.1743	0.0828	No
Pulse	-0.1978	0.0486	Yes
Respiratory Rate	-0.1361	0.1771	No
Respiratory System	-0.3037	0.0024	Yes
CVS	0.01664	0.8695	No
Abdomen system	0.01328	0.8956	No
CNS	0.0611	0.5458	No
Body Weight	-0.1137	0.2601	No
Height	0.0895	0.3755	No
BMI	-0.2002	0.0459	Yes
Abdominal Circumference	-0.1591	0.1139	No
FBS	-0.2434	0.0147	Yes

HT: Hypertension; SBP: Systolic blood pressure, DBP: Diastolic blood pressure, CVS- Cardiovascular system, CNS- Central nervous system, FBS- Fasting blood sugar

having higher chances of developing HTN. In contrast to the other researcher's data, our study contained more male participants in the obese hypertensive group.[5,14] The mean duration of HTN in obese hypertensive patients in our study was 7.50 ± 3.86 years, and in non-obese hypertensive patients it was 7.22 ± 4.09 years, whereas previous articles have reported it to be 4 years, which clearly reflects that patients today with HTN are showing improved and extended life expectancy with newer anti-hypertensive medications.[14,15] There is no literature detailing the addiction and food preferences of the obese and non-obese hypertensive participants, which have been considered in our study.

The mean SBP recorded in obese and non-obese patients was 136.36±12.17 and 138±14.64 mm Hg respectively. Gomez P et al, in 2006, also observed very similar SBP in obese patients i.e. 145.84 ± 18.2 mmHg.[5] Higher median BP was reported by another study in which the median SBP was 150 (100-280) mm Hg for non-obese patients, while the mean DBP in obese patients was noted as 86.60 ± 6.75 and 85.72 ± 6.98 mmHg in non-obese patients.[14] Gomez P et.al, reported very similar DBP readings as 85.5 ± 10.8 mm Hg in their obese patients.[5] The median DBP observed by Osafo C et al. was 90 (60-100) mm Hg, which is similar to our study.[14] The SBP and DBPs in obese HTN was slightly lesser than non-obese this may be due to the variable treatment received by the patients in the past.

Obese patients had a higher percentage of pallor (12%) compared to non-obese patients (8%) and similar incidence of oedema (14%). Mean pulse rate was lower in obese patients (81.04 ± 10.21 bpm) than in non-obese patients (84.66 ± 9.04 bpm), while mean respiratory rate was similar in both groups. Most patients had normal cardiovascular and PA systems, but a small proportion had abnormal respiratory

systems. These findings suggest an association between obesity and pallor and respiratory system abnormalities, but further research is needed to confirm these associations. In our study, 98% of patients had a normal CNS system, with only one non-obese patient showing abnormalities. These findings have not been previously described by other authors. Our study found that obese patients had a higher mean body weight

whereas mean body height was 1.66 ± 0.08 m and 1.71 ± 0.09 m in obese and non-obese patients, respectively. The BMI was 30.68 ± 2.36 kg/m2 and 23.04 ± 1.87 kg/m^2 in obese and non-obese patients, respectively. A higher mean BMI index for obese patients has been reported previously,[5] while the median BMI for non-obese hypertensive patients was 29.7 (12.2- 67.4) reported in another study.[14] A parallel observation with literature was recorded in term of waist circumference, fasting glucose level, mean HB1Ac in obese patients versus non obese ones [5]. We observed higher mean cholesterol levels in obese patients compared to non-obese patients. Similarly, mean triglyceride, HDL and LDL levels were also higher in obese patients compared to non-obese patients. A likewise finding about cholesterol, triglyceride and HDL has been published previously albeit no reports presents LDL cholesterol levels.[5]

In this study, serum levels of urea, creatinine, and uric acid were measured in both obese and non-obese patients. Proteinuria was observed in a higher percentage of obese patients (24%) compared to non-obese patients (12%). The majority of patients in both groups did not show evidence of proteinuria. In the HTN detection and follow up programme, the incidence of chronic renal disease at 5 years, defined by proteinuria and GFR< 60 ml/min per 1.73 m^2 was higher in patients with obesity and overweight (31 and 34% respectively) than in hypertensive patients with ideal weight (28%).[16] The present study reports median haemoglobin and urine protein/creatinine ratio in obese patients, and have identified hepatic and renal abnormalities. Similar findings were observed in non-obese patients. These parameters were not previously evaluated by any authors. In obese and non-obese patients, mean eGFR was 85.47 ± 38.26 and 87.60 ± 35.64 respectively. Previous literature have reported median eGFR as 73.0 (1.68-209.5) for hypertensive patients, lower than our study.[14] Prevalence of renal insufficiency in obese and non-obese patients was found 30% and 20% respectively being comparable to the result of Gomez P et al.[5] On the other hand, other article have reported more than double prevalence rate i.e. 46.9% for renal insufficiency in hypertensive patients.[14] In a Spanish study, the occurence of GFR was 5.1%[17] similar to that performed in Canadian population but the prevalence rate increased further with HTN i.e. 16.2%.[18] Moreover, obesity is a stronger risk factor for HTN and CKD, which increases the prevalence rate of renal insufficiencies further, as indicated in our study.

In addition, certain risk factors were found to have negative intermediate correlations with renal insufficiency, including serum urea, serum creatinine, HbA1c, serum uric acid, urine protein, abdominal USG, duration of HT, and respiratory system. Weak negative correlations were also observed for systolic BP, pulse, BMI, and fasting blood when correlated with renal insufficiency. ECG findings in obese patients of our study showed arrhythmia in 7 (14%) patients, LVH in 18 (36%) patients, ST-T changes in 10 (20%) patients, and LBBH in 6 (12%) patients. ECG was done in non-obese patients which showed arrhythmia in 3 (6%) patients, LVH in 18 (36%) patients, and LBBH in 4 (8%) patients. ST-T changes were detected in 8 (16%) patients and T-wave depression in 3 (6%) patients. Limited research has investigated correlation parameters as executed in our research. In the future, further correlation studies will be needed to ascertain the role of risk factors associated with renal insufficiencies.

Lifestyle changes can be an effective method for improving patient outcomes in obese people. Salt restriction, body weight maintenance, exercise, abstaining from alcohol and smoking are all known to help prevent HTN development and consequences. Our findings also show that patients had greater rates of all of these risk variables, particularly obesity.[19] Newer anti-hypertensives should be used not just to lower blood pressure but also to protect target organs such as the kidneys, eyes, brain, and heart. This can aid in the prevention of HTN problems and the accompanying risk to the patient's life. Although the results of the present study were not significant, it is worth noting that the study was conducted on a small number of participants for a relatively short period. Nonetheless, the study identified a higher prevalence of renal insufficiencies in hypertensive patients, and the presence of obesity was observed to exacerbate the risk of kidney problems in this population. These findings provide a basis for future research with a larger sample size and longer duration to further investigate the association between hypertension, obesity, and renal dysfunction.

5. Conclusion

Our findings suggest hypertension is a significant risk factor for renal dysfunction, and obesity may worsen the risk. Despite non-significant results, the study provides a foundation for future research to develop interventions to mitigate renal failure risk in patients with these risk factors. Patients with hypertension present at a younger age. A healthy diet, daily exercise, and avoiding smoking and alcohol intake may help to reduce HTN problems. Obesity is a major risk factor for the development of CKD. The earlier onset of disorders such as HTN and obesity increases patient mortality and morbidity. More effective approaches to managing obesity and avoiding the development and progression of obesity-related CKD are

warranted. As a result, there is an expanding need for an extensive understanding of the causes of high BMI or obesity, as well as their management.

6. Source of Funding

None.

7. Conflict of Interest

Authors declare no conflict of interest

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