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Original Article

A CLINICAL COMPARATIVE STUDY OF RENAL PARAMETERS OF OBESE AND HYPERTENSIVE PATIENTS WITH NON-OBESE HYPERTENSIVE PATIENTS

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ABSTRACT

Objective: Obesity is associated with an early onset of glomerulomegaly, hemodynamic changes of a hyper-filtering kidney, and increased albuminuria. It is an important risk factor for incident CKD and increase risk of ESRD. The aim of the present study is to compare the influence of renal parameters among the obese and non-obese hypertensive patients attended to a tertiary care hospital.

Methods: The study was conducted on 120 patients who were divided into 4 groups Group 'O' comprises of obese individuals; Group 'OH', which comprises of 30 obese and hypertensive individuals; Group 'NO, H' which comprises of non-obese and hypertensive individuals and Group 'C' which comprises of 30 healthy control group having normal BMI.

Results: The mean±SD of serum creatinine was 0.88±0.06; 1.13±0.1; 0.7±0.08; 0.7±0.06 of all the 4 groups and the Serum urea mean±SD was 26.4±3.8; 36.6±5.42; 21.2±3.2; 20.4±3.2 and the Serum uric acid mean±SD levels were 4.3±0.64; 4.3±0.56; 3.8±0.3; 3.7±0.26 of all the 4 groups and eGFR values were 139.5±1.8; 95.3±23.20; 116.2±7.4;118.6±6.2 in obese, obese hypertensive, non-obese hypertensive and control subjects respectively. The results showed that there was a significant influence of renal parameters among the four tested groups.

Conclusion: The strategies to prevent weight gain and to induce weight loss in patients who are already obese would ultimately reduce the incidence of obesity-mediated renal disease.

Keywords: BMI, Renal parameters, Hypertension, Obese, Non-obese

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INTRODUCTION

Obesity is a known risk factor for chronic kidney disease, diabetes and hypertension. The prevalence of obesity in India has been steadily increasing due to changes in lifestyle, dietary habits, and socioeconomic factors. Estimates suggest that about 20-30% of the urban population and 10-15% of the rural population in India is obese. Obesity rates tend to be higher in women compared to men. The prevalence of obesity is higher among adults aged 30-60 y. According to Zajak *et al.* obesity is an abnormal growth of adipose tissue due to enlargement of fat cell size or an increase in fat cell number or combination of both [1]. It is a global pandemic problem and results in a shortened life span related to adverse health outcomes. It is an important risk factor for incident chronic kidney disease (CKD) and increase risk of End-stage Kidney disease (ESRD) [2]. Body mass index, (BMI) is a globally accepted anthropometric measure for obesity classification. Obesity is associated with the early onset of glomerulomegaly, hemodynamic changes of a hyper-filtering kidney, and increased albuminuria, which are potentially reversible with weight loss. There is substantial evidence that excess visceral fat is the main driving force for almost all of the disorders associated with metabolic syndrome, including CKD [3, 4]. Obesity can significantly influence renal parameters and contribute to various renal disorders. In obese individuals often have an increased GFR, a condition known as hyperfiltration, which can be an early sign of kidney damage. Obese individuals often have increased muscle mass, which can lead to higher baseline levels of serum creatinine. Since creatinine is a byproduct of muscle metabolism, more muscle can mean more creatinine production. Obesity can lead to elevated serum urea and uric acid levels through increased production, decreased excretion, and associated metabolic conditions. Regular monitoring and appropriate lifestyle or medical interventions can help manage these levels and reduce the risk of related complications. Obesity causes hypertension 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 and the renin-angiotensin-aldosterone system, as well as physical compression of the kidneys, especially when visceral adiposity is high [4]. Previous research has shown that a higher prevalence of renal insufficiency is associated with higher blood pressure [3].

MATERIALS AND METHODS

A prospective single-centre observational cross-sectional study was carried out among 120 subjects who are attending outpatients. The study was conducted at Government General Hospital, Kurnool over a period of five months from July 2024 to November 2024.

Exclusion criteria

- Patients not giving consent
- Presence of other co-morbidities during enrolment of the study
- Women of childbearing age

Inclusion criteria

- All the selected patients were within the age group of 20-75Y with BMI 18.5.
- All hypertensive patients should be on treatment for at least 1 y in cases group.
- A total of 120 individuals (Males: 81, females: 19) were included in the study and are divided into 4 groups. Group 'O' comprises of obese individuals; Group 'OH' which comprises of 30 obese and hypertensive individuals; Group 'NO, H' which comprises of non-obese and hypertensive individuals and Group 'C' which comprises of 30 healthy control group having normal BMI.

Sample collection

Under aseptic precautions, 5 ml of venous blood was collected from anticubital vein in a plain bulb and kept undisturbed for one and half

hour till it clots. Then, the clotted blood was subjected to centrifugation at 5000rpm for 5 min. These rum thus separated, is used for investigation. BMI calculated as weight in Kg/height in meter$^{2.}$ The estimation of serum urea was done by glutamate dehydrogenase (GLDH) method [5]. The estimation of serum creatinine was done by modified Jaffe's method [6] whereas estimation of uric acid in the serum sample was performed by uricase method. eGFR was calculated using the simplified Modification of Diet in Renal Disease (MDRD) formula. Renal insufficiency was defined in the protocol as a GFR by MDRD<60 ml/min per 1.73 m^2 [7].

Statistical analysis

All the data was collected and complied in Microsoft excel and analysis was done using parametric est. for qualitative data chi^2 test was used and for quantitative data student't' test was used. The 'p'value was considered as statistically significant when p<0.05 and highly significant when p<0.01.

RESULTS

The present study aimed to compare the renal parameters between obese hypertensive and non-obese hypertensive patients. All variables are expressed in (Mean±SD). There is no significant statistical difference of their demographic data between the groups p>0.05 (table 1). Table 2 shows the mean BMI in obese hypertensive group was 32.1±1.4 kg/meter^2 and in the non-obese hypertensive group 22.8±2.6 kg/meter^2 which was statistically highly significant 'p'<0.0001. There is no clinically significant difference between the two groups in their SBP and DBP p>0.05. In obese hypertensive group serum creatinine was 1.13±0.1 mg/dl, whereas in non obese hypertensive group it was 0.7±0.08 mg/dl which was a statistically significant p<0.0001. The blood urea in obese hypertensive group was 36.6±5.42 mg/dl and in non obese hypertensive group it was 21.2±3.2 mg/dl, which was statistically highly significant p<0.0001. Serum uric acid in obese hypertensive group and non obese hypertensive group was 4.3±0.56 mg/dl and 3.8±0.3 mg/dl, respectively, which is also statistically highly significant p<0.0001. Table 2, fig. 1 shows the eGFR in obese hypertensive group was 95.3±23.2 ml/min and in non-obese hypertensive group it was 116.2±7.4 ml/dl it is also statistically significant p<0.0001. Table 3 shows the comparison of BMI, renal parameters between. Obese and control groups. All these parameters are significantly higher in obese group than control group p<0.0001. Table 4 shows the comparison between control and non-obese hypertensive group. There was no significant difference present between these two.

Table 1: Demographic and anthropometric measures

Variable	Group 'O' (n=30)	Group 'OH' (n=30)	Group 'N,OH' (n=30)	Group 'C' (n=30)
Age (Y)	41.8±6.2	66.2±6.8	65.8±6.9	54.2±7.8
Males:	18	21	20	22
Females:	12	9	10	8
Weight	92.4±5.6	89.2±6.6	68.2±7.8	65.2±4.2
BMI (Kg/meter^2)	32.6±1.5	32.1±1.4	22.8±2.6	22.46±1.26

Table 2: Independent 't' test between obese hypertensive and non-obese hypertensive

Variable	Group OH (n=30)	Group NOH (n=30)	t-value	p-value
Age (Y)	66.2±6.8	65.8±6.9		
BMI (Kg/m^2)	32.1±1.4	22.8±2.6	17.25	<0.0001
SBP (in mmhg)	141.4±6.2	138.2±3.6	2.44	0.01
DBP (in mmhg)	86.1±3.4	85.2±2.6	1.15	0.25
Serum urea(mg/dl)	36.6±5.42	21.2±3.2	13.5	<0.0001
Serum creatinine (mg/dl)	1.13±0.1	0.7±0.08	18.39	<0.0001
Serum uric acid (mg/dl)	4.3±0.56	3.8±0.30	4.31	0.0001
eGFR(ml/min)	95.3±23.2	116.2±7.4	4.7	<0.0001

Table 3: Independent 't' between group C and group O

Variable	Group C (n=30)	Group O (n=30)	t-value	p-value
Age (Y)	54.2±7.8	41.8±6.2		
BMI (Kg/m^2)	22.46±1.26	32.6±1.5	28.35	<0.0001
SBP (in mmhg)	116.16±4.6	118.12±5.6	1.48	0.14
DBP (in mm hg)	74.12±5.2	73.18±5.2	0.7	0.48
Serum urea(mg/dl)	20.4±3.2	26.4±3.8	6.61	<0.0001
Serum creatinine (mg/dl)	0.7±0.06	0.88±0.06	11.61	<0.0001
Serum uric acid (mg/dl)	37±0.26	4.3±0.64	4.75	<0.0001
eGFR(ml/min)	118.6±6.2	139.5±1.8	17.73	<0.0001

Table 4: Independent 't' between C and NOH patients

Variable	Group C (n=30)	Group NOH (n=30)	t-value	p-value
Age (Y)	54.2±7.8	65.8±6.9		
BMI (Kg/m^2)	22.46±1.26	22.8±2.6	0.71	0.47
SBP (in mmhg)	116.16±4.6	138.2±3.6	20.66	<0.0001
DBP (in mm hg)	74.12±5.2	85.2±2.6	10.43	<0.0001
Serum urea(mg/dl)	20.4±3.2	21.2±3.2	0.96	0.33
Serum creatinine (mg/dl)	0.7±0.06	0.7±0.08	0	1
Serum uric acid (mg/dl)	37±0.26	3.8±0.30	1.38	0.17
eGFR (ml/min)	118.6±6.2	116.2±7.4	1.36	0.17

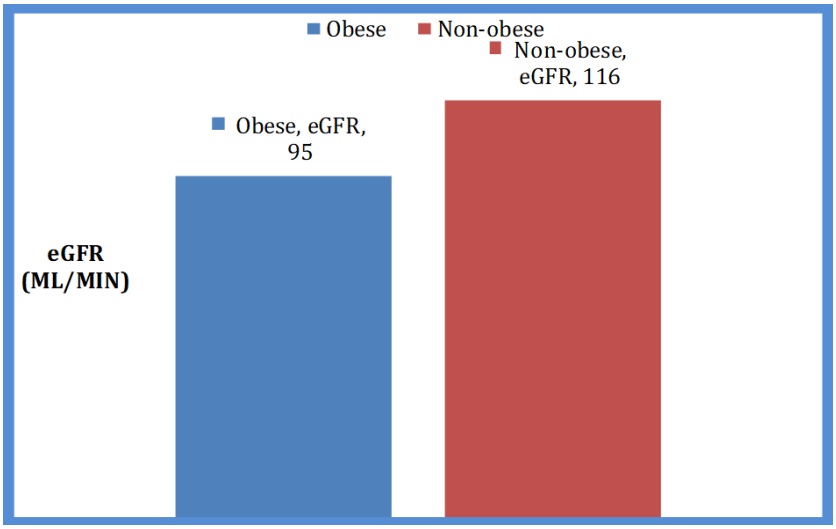

Fig. 1: Comparison of eGFR in obese and non-obese hypertensive individuals

DISCUSSION

Obesity-induced renal dysfunction will become an increasingly difficult clinical dilemma as the prevalence of obesity continues to increase. Even modest weight loss of 5% to 10% has been show to produce substantial benefits, including reductions in blood pressure and risk of diabetes. Several pathophysiological factors, such as heamodynamic and inflammatory factors, genetic factors and co-morbidities intricately intertwined in the initiation and progression of obesity-induced hypertension and CKD [7, 8]. Shetty *et al.* [9] conducted a cross-sectional comparative study of renal insufficiency in obese and non-obese hypertensive patients; they found the incidence of chronic renal disease higher in patients with obesity and overweight than in hypertensive patients with ideal weight. Our studies are very well correlated with Shetty *et al.* [9] study. In our studies, serum creatinine in obese group was 1.13±0.1 mg/dl, whereas in non obese hypertensive group it was 0.7±0.08 mg/dl, which was a statistically significant $p<0.0001$. The blood urea in obese hypertensive group was 36.6±5.42 mg/dl and in non obese hypertensive group it was 21.2±3.2 mg/dl, which was statistically highly significant $p<0.0001$. Serum uric acid in obese hypertensive group and non obese hypertensive group was 4.3±0.56 mg/dl and 3.8±0.3 mg/dl, respectively, which is also statistically highly significant $p<0.0001$. The eGFR in obese hypertensive group was 95.3±23.2 ml/min and in non-obese hypertensive group it was 116.2±7.4 ml/dl it is also statistically significant $p<0.0001$. Gomez *et al.* [3] in their study showed that 22.8% obese hypertensive patients had renal insufficiency. Osafo C *et al.* [10] in their research showed that 46.9% of obese hypertensive patients had renal insufficiency. Our study was very well correlated with Gomez *et al.* [3] and Osafo *et al.* [10] study. Our study findings of renal parameters between obese hypertensive and non obese hypertensive patients are in accordance with findings of Sandhu *et al.* [11] study who also found the significant correlation of BMI with renal profile among obese individuals. Kaufmann *et al.* [12] study also shows a significant association between BMI and renal parameters between obese hypertensive and non-obese hypertensive patients our studies are also very well correlated with their study. Hsu *et al.* [13] showed the relation between excess weight and risk of ESRD. Kambham *et al.* [14] in their studies concluded that obesity-related glomerulopathy is an emerging epidemic. Our study findings coincide to their findings.

CONCLUSION

Obesity is a known risk factor for adverse health outcomes, including chronic kidney disease, diabetes and hypertension. Periodic evaluation of renal function tests can help in detection of early renal damage in obese hypertensive patients. Modest weight loss of even 5% to 10% has been shown to produce substantial benefits, including reductions in blood pressure and risk of diabetes.

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AUTHORS CONTRIBUTIONS

All authors have contributed equally

CONFLICT OF INTERESTS

Declared none

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