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COMPARISION OF RENAL PROFILE OF OBESE HYPERTENSIVE PATIENTS WITH NON OBESE HYPERTENSIVE PATIENTS

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ABSTRACT

Introduction: Hypertension is one of the most common disease affecting humans throughout the world and is an important public health challenge because of associated morbidity and mortality.

Materials and methods: 120 study subjects were selected and were divided into four groups consisting of Obese (n=30), Obese hypertensive patients (n=30), Non obese hypertensive patients (n=30) and Healthy controls (n=30). By using simple random method cross sectional study was done.

Results: Serum urea (Mean ± SD) was (26 ± 3.91), (36.6 ± 6.32),(21.2 ± 4.16) and(21.5 ± 4.51) ,Serum creatinine levels (Mean ± SD) was (0.84 ± 0.07), (1.11±0.23),(0.70 ± 0.07) and (0.7 ± 0.05) ,Estimated GFR (Mean ± SD) was (140.5 ±17), (96.2 ±22.2), (116.1 ±7.7) and(119.6 ±6.7) in obese, obese hypertensive patients, non obese hypertensive patients and healthy group respectively.

Conclusion: Periodic evaluation of renal function tests can help in detection of early renal damage in obese hypertensive patients.

KEYWORDS : GDM, macrosomia, SGA, hypoglycemia, polycythemia, RDS, hyperbilirubinemia, hypocalcaemia, still births, IUD

Introduction: Hypertension doubles the risk of cardiovascular diseases, including coronary heart disease (CHD), congestive heart failure (CHF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease[1]. Rising mean blood pressure is apparent in populations as they are industrialize and move from rural to urban areas.

Overall global burden of hypertension according to worldwide epidemiological data in the year 2000 was 26.4% of the adult population and projected to have 29.2% by 2025[2]. In India rising prevalence of hypertension was demonstrated through serial epidemiological study conducted during the year 1994,2001, 2003 (30%, 36%, 51% among males and 34%, 38%, 51% among females respectively)[3,4,5].

Both environmental and genetic factors may contribute to regional and racial variations in blood pressure and hypertension prevalence. Obesity and weight gain are strong independent risk factors for hypertension[6]. It has been estimated that 60% of hypertensives are >20% overweight[6].

Aging in the developed world is commonly associated with the occlusion of coronary and systemic blood vessels; the reasons for this include obesity, insulin resistance, smoking, hypertension, and diets rich in lipids leading to deposition of circulating lipids in the arterial and arteriolar circulation producing local inflammation and fibrosis of small blood vessels[7]. When the renal arterial circulation is involved, the glomerular microcirculation is damaged leading to chronic nephrosclerosis. As many as 27% of patients with end-stage kidney disease have hypertension as a primary cause[8].

Obesity is a common nutritional disorder, characterized by the excess fat deposition due to increased energy intake and decreased energy expenditure. Excess weight gain which causes change in the functions and adaptation of different organs including cardiov ascular and renal system leads to progression of hyperten sion and chronic kidney disease[9].

Prevalence of obesity among western countries is more and now prevalence in developing countries is increasing due to sedentary life style and unhealthy food habits.

Obesity is associated with glomerular hyper-filtration in animal models[10].

Human studies[11-13] also revealed abnormal renal hemodynamic in obese subjects showing increased glomerular filtration rate (GFR), increased renal blood flow or both. One of the previous study demonstrated that in subjects with severe obesity, weight loss results in a decrease in GFR, renal plasma flow (RPF), filtration fraction (FF) and arterial pressure[14].

Some of the studies shown Potential mechanisms for obesity in the development of hypertension and progression of renal disease. Obesity is an important risk factor for development of diabetes mellitus and hypertension, which are the most common etiologies of kidney failure. Although there is not a clear correlation between the extent or duration of hypertension and the risk of end-organ damage.

Based on a careful history, physical examination, urine analysis, and some serum testing, the diagnosis of chronic nephrosclerosis is usually inferred without abiopsy[8].

Studies on isolated role of obesity causing renal impairment are only a few and hence the present study was conducted to know the individual effect of obesity on renal profile along with hypertension.

AIMS AND OBJECTIVES

1. To measure Blood pressure and to calculate BMI in study groups.
2. To compare difference in values of renal profile in study groups.

MATERIAL AND METHODS

The study groups was comprised of 120 subjects including 60 patients visited to out-patient department of Medicine and admitted cases in medical ward at Rajendra Institute of Medical Sciences, Ranchi.

Study subjects

By using simple random method, 120 study subjects were taken and the study subject was divided into following 4 groups:

Group 1: Comprised of 30 Obese individuals.
Group 2: Comprised of 30 Obese Hypertensive patients.
Group 3: Comprised of 30 Non Obese Hypertensive patients.

Group 4 : Comprised of 30 Healthy control having normal BMI.

Body Mass Index
Calculated as weight (kg)/height (meters2)

Blood pressure:
It was recorded in supine posture after a rest period of 10 minutes with the standard mercury sphygmomanometer, by the single observer. Average of two readings was taken into consideration.

SBP = At the appearance of Korotkoff's first sound (phase I)
DBP = At the disappearance of Korotkoff's sound (phase V)

Sample collection :
Under aseptic precautions 4 ml of venous fasting blood sample was collected from antecubital vein in plain vacutainer for renal parameters.

Method of analysis:
All serum samples were analyzed by Semi automatic biochemical analyzer using Erba Chem-7. The precision of the instrument was checked on many occasions. All the analytical procedures were standardized, the reagents are calibrated to the instrument before sample analysis was done

ESTIMATION OF SERUM UREA:
Methodology
Enzymatic Urease- Glutamate dehydrogenase (GLDH) method 15,16

Calculation:
Absorbance of Test

Serum Urea = ——————— X Concentration of Standard (mg/dl)
Absorbance of Standard

Reference value:
Serum urea 13-45 mg/dl

ESTIMATION OF SERUM CREATININE:
Methodology
Modified Jaffe's method[17-19]

Calculation:

(T2-T1) of Sample

Serum creatinine = ——————— X Concentration of Standard (mg/dl)
(T2-T1) of Standard

Reference range
Male: 0.7-1.4 mg/dl
Female: 0.6-1.2 mg/dl

OBSERVATION AND RESULT
Table – 1: Distribution of Variables in Study Groups

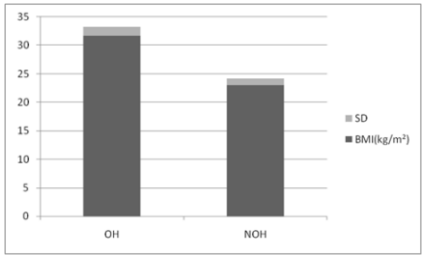
Sl. No	Variables	Values expressed as mean ± SD			
		Study Groups			
		Healthy	Obese	OH	NOH
1.	Age (years)	44.2 ± 7.03	44.6 ± 6.6	46.6 ± 9.35	46.2 ± 7.6
2.	Weight (Kilograms)	64.9 ± 5.76	92.7 ±6.15	84.5 ±4.2	64.7 ± 5.45
3.	Height (Square meters)	1.70 ±0.03	1.7 ± 0.03	1.63 ± 0.03	1.68 ± 0.04
4.	BMI	22.49 ± 1.37	31.9 ±1.07	31.7 ± 1.57	23 ±1.2
5.	Systolic blood pressure (mmHg)	117.13 ±4.8	119.6 ±6.7	140.4±6.6	137.7 ± 4
6.	Diastolic blood pressure (mmHg)	74.13 ± 5.35	74.1± 5.35	85.1± 3.6	84.2 ± 2.7
7.	Serum urea (mg/dl)	21.5 ± 4.51	26± 3.91	36.6± 6.32	21.2 ± 4.16
8.	Serum creatinine (mg/dl)	0.7 ± 0.05	0.84 ± 0.07	1.11± 0.23	0.70 ±0.07
9.	Serum uric acid (mg/dl)	3.8±0.28	4.2 ±0.56	4.2 ±0.47	3.83 ±0.30
10.	eGFR (ml/minute)	119.6 ± 6.7	140.5 ± 17	96.2 ±22.2	116.1 ±7.7

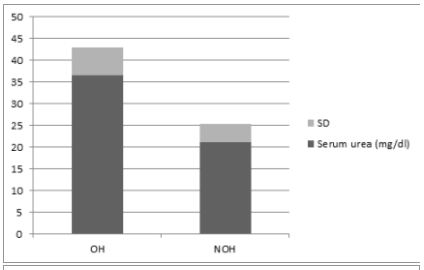
TABLE – 2: Independent ' t' test between Obese hypertensive and Non obese hypertensive patients

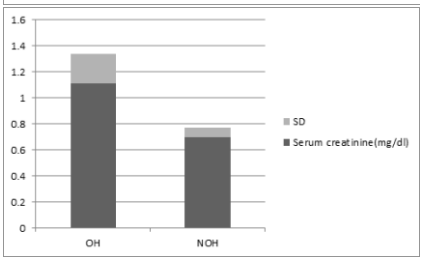
Sl. No	Test variables	Values expressed as mean ± SD		't' value	'p' value
		OH	NOH		
1.	Age	46.6 ± 9.35	46.2 ± 7.6	0.212	0.833
2.	BMI	31.7 ± 1.57	23 ±1.2	23.935	0.0001**
3.	Systolic blood pressure	140.4 ± 6.6	137.7 ± 4	1.915	0.060
4.	Diastolic blood pressure	85.1 ± 3.6	84.2 ± 2.7	1.092	0.28
5.	Serum urea (mg/dl)	36.6 ± 6.32	21.2 ± 4.16	11.09	0.0001**
6.	Serum creatinine(mg/dl)	1.11 ± 0.23	0.70 ± 0.07	9.115	0.0001**
7.	Serum uric acid(mg/dl)	4.2±0.47	3.83±0.30	3.547	0.001**
8.	eGFR (ml/minute)	96.2 ± 22.2	116.1 ± 7.7	-4.624	0.0001**

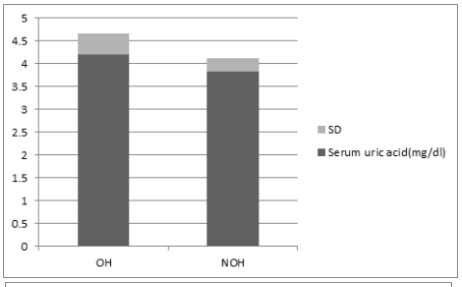
OH – Obese hypertensive patients
NOH – Non obese hypertensive patients

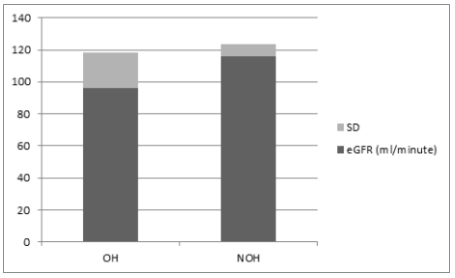
**** p value is highly significant < 0.005**











DISCUSSION

Table 1: Shows the mean values of the all measured variables in the four study groups.

Table 2: Showing comparison between obese hypertensive patients and non obese hypertensive patients. No significant difference were observed in age and blood pressure ('p' > 0.05) among these two groups but significant differences ('p' <0.05) were observed in relation to BMI and renal profile.

Hsu et al[20] showed the relation between excess weight and risk of ESRD appeared to persist even after accounting for the presence or absence of baseline diabetes and hypertension.

Neerajakambham et al[21] in their studies concluded that obesity related glomerulopathy is an emerging epidemic and is distinct from idiopathic focal segmental glomerulosclerosis consistent with presence of features of glomerulomegaly and milder foot process fusion.

Nakagawa T et al[22] in their experimental animal study showed that renal injury occurs in hyperuricemic rats, consisting of afferent arteriolopathy, mild tubulointerstitial fibrosis, glomerular hypertrophy, and eventually glomerulosclerosis and albuminuria.

CONCLUSION

From the present study it can be concluded that additive decline in renal function in obese hypertensive's is mainly due to Glomerulo sclerosis leads to changes in the renal profile. Hence periodic evaluation of renal function tests can help in detection of early renal damage in obese hypertensive patients.

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