Internet Journal of Medical Update

Journal home page: http://www.akspublication.com/ijmu

Original Work

Correlation of blood pressure with Body Mass Index (BMI) and Waist to Hip Ratio (WHR) in middle aged men

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(Received 18 November 2009 and accepted 21 January 2010)

ABSTRACT: Obesity and cardiovascular risks are closely associated. Hypertension is the most common and early complication of obesity. Obesity is measured with different parameters like Body Mass Index, Waist to Hip Ratio etc. In the present study we have tried to link parameters of obesity with hypertension. We have found that in hypertensive middle aged Indian males diastolic blood pressure showed a better correlation with Waist to Hip Ratio rather than with Body Mass Index.

KEW WORDS: Obesity; Hypertension; Body Mass Index (BMI); Waist to Hip Ratio (WHR)

INTRODUCTION

Blood pressure more than or equal to 140/90 mm of Hg is defined as hypertension.[1] More than a quarter of the world's adult population is already hypertensive and this number is projected to increase to 29% (about 1.56 billion) by 2025. Almost three-quarters of this hypertensive population is going to be in developing countries.[2] Risk factors associated with development of hypertension could be internal as genetic inheritance or external like psychosocial stress, salt consumption and obesity.[3]

The incidence of obesity is rising not only in developed countries but also in developing countries. It is recognized as a risk factor for development of hypertension, IHD, DM, dyslipidemias and osteoarthritis.

Assessment of obesity is done by various tests such as Body Mass Index (BMI) [Quetelets Index][4,5], Waist Circumference (WC)[6,7], Hip Circumference (HC), Waist to Hip Ratio (WHR)[8], Skin fold thickness[4,9], Ponderal index[4,10,11], Broca Index[10], Neck circumference[12], Bioelectric impedance analysis and Hydrostatic weighing[13], Quantitative measurement of Soluble Human Leptine Receptors by ELISA[14], Dual – Energy X-ray Absorptiometry (DEXA) scan, and CT and MRI scanning[15].

This study was planned to correlate blood pressure with BMI and WHR in middle aged males.

METHODOLOGY

The study was a clinic based cross-sectional study. It was conducted in private dispensaries and the Department of Physiology of a local medical college, Pune (India). Males aged between 36 to 60 years were included in the study. The study group consisted of 100 newly diagnosed hypertensive males (Hypertensive group). Control group consisted of 100 males who were normotensive. Subjects who were suffering from major illnesses like diabetes mellitus, other endocrine disorders, and renal diseases were excluded on the basis of history and investigation reports.

Purpose of the study was explained to both the groups. Written consent was taken. Participants were interviewed by investigator using pre-test proforma. Institutional ethical

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committee approval was obtained for the study.

After giving half an hour rest to the person in the clinic or departmental laboratory, blood pressure was measured in supine position by mercury sphygmomanometer, between 10 am to 11 am. The pressure at which Korotkoff's sound first heard (Phase I) was taken as systolic blood pressure and the pressure at which these sounds disappeared (Phase V) was taken as diastolic blood pressure. Blood pressure was measured 3 times. The average of second and third readings was taken as correct systolic and diastolic blood pressure.[16]

Body weight was measured while the subject was minimally clothed and without shoes, standing motionless on a weighing scale and it was recorded to the nearest 0.1kg.

Height was measured to the nearest 0.1 cm while the subject was standing in erect position with bare feet on flat floor against a vertical scale and with heels touching the wall and head straight. BMI[4,5] was measured by weight in kilograms divided by square of height in meters (kg/m^2).

$$\mathrm{BMI} = \frac{weight\ (\mathrm{kg})}{height^2(\mathrm{m}^2)}$$

(BMI in the range of 18.50 to 24.99 kg/m^2 is considered to be normal[5].)

Waist circumference (in cms) was measured at a point mid-way between the lower rib and iliac crest with the measuring tape centrally positioned 1cm below the umbilicus. Hip circumference was measured (cms) over light clothing at the widest girth of the hip. For waist and hip circumference two consecutive readings were made at each site on a horizontal plane without compression of the skin. The mean was taken as the final reading. WHR was calculated by dividing waist circumference by hip circumference.[17]

$$\mathbf{WHR} = \frac{\textbf{Waist Circumference (cms)}}{\textbf{Hip Circumference (cms)}}$$

(WHR of 0.9 is considered normal for males.[17])

Statistical analysis was done by using z test and tests of correlation.

RESULTS

There were 100 participants in each group and **table 1** shows age wise distribution of participants in our study. The maximum number of participants i.e. 60/200 (30%) were in age group of 41-45 years, of these 28 were of the study group and 32 belonged to the control group. The minimum number of participants i.e. 9/200 (4.5%) were in age group of 56-60 yrs, of these 5 belonged to hypertensive group and 4 belonged to control group.

Table 1: Age wise distribution of participants

Age (Yrs)	Study group*	Control group**	Total
36 – 40	24	21	45
41 – 45	28	32	60
46 – 50	20	24	44
51 – 55	23	19	42
56 - 60	5	4	9
Total	**100**	**100**	**200**

*Study group – Hypertensive
**Control group – Normotensive

Table 2 shows distribution of participants of our study with respect to BMI. Out of 100 participants of control group, 54 had BMI ≥ 30 while 46 were having BMI < 30. Out of 100 participants of the hypertensive group 59 had a BMI ≥ 30 while 41 had a BMI < 30. These results were not statistically significant.

Table 2: Distribution of subjects in both the groups with different BMI

BMI	Hypertensive group (n= 100)	Control group (n= 100)
≥30	59	54
<30	41	46

Table 3 shows association of WHR in hypertensive and control group. It is seen that in control group (n=100) 48 participants had a WHR ≥ 0.9 while 52 participants had a WHR < 0.9. In hypertensive group (n=100) 64 participants had a WHR ≥ 0.9 while 36 participants had a WHR < 0.9. These findings are statistically significant ($P < 0.05$).

Table 3: Distribution of subjects in both the groups with different WHR

WHR	Hypertensive group (n=100)	Control group (n= 100)
≥ 0.9	64*	48
< 0.9	36	52

*Statistically significant ($P < 0.05$)

Table 4 shows correlation of systolic BP with BMI in both the groups. There is no significant correlation between systolic BP and BMI in both the groups. **Table 5** shows correlation of diastolic BP with BMI in both the groups. There is significant positive correlation between diastolic BP and BMI in hypertensive group. There is no significant correlation between diastolic BP and BMI in control group.

Table 6 shows correlation of systolic BP with WHR in both the groups. There is no significant correlation between systolic BP and WHR in both the groups. **Table 7** shows correlation of diastolic BP with WHR in both the groups. There is highly significant positive correlation between diastolic BP and WHR in hypertensive group; i.e. diastolic BP is significantly higher in those having WHR ≥ 0.9. Correlation is borderline significant in control group.

Table 4: Correlation between systolic BP and BMI in hypertensive and control groups

Groups	Systolic BP (mmHg) with BMI ≥ 30kg/m^2	Systolic BP (mmHg) with BMI < 30kg/m^2	r Value	P value
Hypertensive Group (n = 100)	(n = 59) 167.63 ± 20.58	(n = 41) 162.0 ± 14.81	0.13	>0.05
Control Group (n = 100)	(n = 54) 120.15 ± 8.28	(n = 46) 123.43 ± 7.58	-0.17	>0.05

Table 5: Correlation between diastolic BP and BMI in hypertensive and control groups

Groups	Diastolic BP (mmHg) with BMI ≥ 30kg/m^2	Diastolic BP (mmHg) with BMI < 30kg/m^2	r Value	P value
Hypertensive Group (n = 100)	(n = 59) 94.8 ± 4.18	(n = 41) 93.03 ± 3.41	0.37	<0.01*
Control Group (n = 100)	(n = 54) 80.11 ± 2.02	(n = 46) 80.91 ± 2.97	-0.15	>0.05

*Significant

Table 6: Correlation between systolic BP and WHR in hypertensive and control groups

Groups	Systolic BP (mmHg) with WHR ≥ 0.9	Systolic BP (mmHg) with WHR < 0.9	r Value	P value
Hypertensive Group (n = 100)	(n = 64) 167.26 ± 18.04	(n = 36) 164.25 ± 18.89	0.07	>0.05
Control Group (n = 100)	(n=48) 122.25 ± 8.24	(n=52) 121.11 ± 8.00	0.11	>0.05

Table 7: Correlation between diastolic BP and WHR in hypertensive and control groups

Group	Diastolic BP (mmHg) with WHR ≥ 0.9	Diastolic BP (mmHg) with WHR < 0.9	r Value	P value
Hypertensive Group (n = 100)	(n = 64) 95.36 ± 4.27	(n = 36) 92.49 ± 2.85	0.40	<0.01*
Control Group (n = 100)	(n = 48) 80.17 ± 3.21	(n = 52) 80.77 ± 2.69	-0.24	<0.05*

*Significant

DISCUSSION

In the present study, there were 100 hypertensive participants in study group and 100 normotensive participants in control group. Blood pressure, Body Mass Index (BMI) and Waist to Hip ratio (WHR) were estimated in both the groups.

We have found no correlation between systolic BP with BMI and WHR in both the groups. As far as diastolic BP is concerned, it shows no correlation with BMI in control group. There is borderline correlation between diastolic BP and WHR in control group. There is significant correlation between diastolic BP and BMI as well as diastolic BP and WHR in hypertensive group. But correlation between diastolic BP and WHR is stronger (r value 0.40) than the correlation between diastolic BP and BMI (r value 0.37).

So, in a nutshell, we have found that diastolic BP correlates better with WHR than BMI. Similar finding was observed by many scientists.[18-23]

WHR is a measure of central obesity. Role of central obesity in pathophysiology of hypertension has been extensively studied by different investigators.[24-28]

Abdominal fat (visceral fat) is now considered to be an important endocrine organ producing biologically active substances with local and systemic actions. These substances are leptin, angiotensinogen, cytokines, resistin, prostaglandin (PGI2alpha), tumor necrosis factor (TNF), insulin-like growth factor, adiponectin and many others. Out of the above mentioned substances, recent studies have highlighted the importance of two adipose tissue derived hormones i.e. leptin and angiotensinogen in obesity induced hypertension.[29] Physiologically leptin is involved in regulation of appetite, food intake, sexual maturation, haematopoiesis and activity of hypothalamo-pituitary-gonadal axis. Obese individuals have high plasma leptin concentration. Leptin influences blood pressure by several mechanisms: such as activation of the sympathetic nervous system and pituitary adrenal axis; influence on water-electrolyte balance; modulation of endothelial cell function and influence on vascular remodeling.[30] Sympathetic activation appears to cause sodium retention and impaired pressure natriuresis.[31] It was found that in obese hypertensive individuals, there was reduced biological activity of atrial natriuretic peptide (ANP) probably as a result of over-expression of the clearance receptor in adipocytes.[32] So, enhanced leptin-driven renal sympathetic outflow, in combination with low ANP activity possibly due to over-expression of the natriuretic peptide clearance receptor in adipocytes, may enhance sodium retention and volume expansion, both key features in the pathophysiology of obesity associated hypertension.

Thus it can be said that visceral fat present in centrally obese Indian male subjects is probably the main contributory factor for hypertension. To detect this risk early, a simple measurement like WHR can be used as a better parameter than BMI.

We do accept some limitations of the present study, such as small sample size and its restriction to middle aged male population only. Similarly we have not estimated blood renin, angiotensin levels, catecholamine levels and leptin levels to confirm our findings.

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