

Assessment of Anthropometric Indices, Salt Intake and Physical Activity in the Aetiology of Prehypertension

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ABSTRACT

Introduction: Globally, prehypertension is responsible for approximately 62% of cardiovascular and 49% of ischemic heart disease (IHD) episodes. Current data from the Indian subcontinent suggests that prevalence of prehypertension was 47% amongst young urban residents. Considering its serious prognosis, the current study was undertaken to assess risk factors such as for cardiovascular diseases in prehypertensive adult males in Uttarakhand region.

Materials and Methods: This cross-sectional analytical study was conducted in the Department of Physiology, HIMS, Dehradun, over a period of 12 months. Volunteers (20-40 years) were divided into two groups; Group I (Controls): normotensives and Group II (Cases): prehypertensives based on JNC VII classification.

Results: Exercise capacity, determined by peak VO_2 consumption was significantly lower in prehypertensive group than the normotensive group ($p < 0.001$). Daily salt intake of prehypertensives was significantly greater than the normotensive subjects ($p < 0.001$). Multiple Linear Regression analysis revealed that average baseline SBP increases by 0.34 mmHg for every 1 kg increase in weight and average baseline DBP increases by 0.25 mmHg for every 1 year increase in age.

Conclusion: Exercise capacity was found decreased in prehypertensives and they have high daily salt intake. Also, weight and age emerged as independent risk factors for SBP and DBP respectively.

INTRODUCTION

Globally people suffering from hypertension are approximately 1 billion and it is responsible for 7.1 million deaths yearly. According to WHO, prehypertension is responsible for approximately 62% of cardiovascular and 49% of ischemic heart disease (IHD) episodes worldwide. Current data from the Indian subcontinent suggests that prevalence of prehypertension is approximately 47% amongst young urban residents [1,2]. Prehypertension is not a disease but an entity to identify individuals who are at high risk of developing hypertension. Joint National Committee (JNC VII) categorized prehypertensives as individuals with systolic pressure (120-139 mm Hg) and/or a diastolic pressure of (80-89 mm Hg) [3]. They are not the candidates for drug therapy but for lifestyle modifications [3]. Longitudinal data from Framingham Heart Study [2,4] have suggested that prehypertension is associated with more than twofold increase in relative risk from CVD as compared to normal individuals. The aetiology of progression from prehypertension to hypertension is multifactorial. It includes genetic predisposition and other environmental factors such as smoking, alcoholism, diet, physical activity, salt intake, stress, sedentary life style, and weight gain etc [5,6]. It is strongly suggested that the current consumption of salt (9-12g/day) is one of the major factors in increasing blood pressure and subsequently CVD. Many studies revealed that physical exercise reduces the incidence of coronary events by 80% [7] and a low level of physical fitness increases risk of CVD mortality [8]. Exercise capacity was a strong and independent predictor of the rate of progression from prehypertension to hypertension. When exercise capacity exceeded 8.5 METs, the protective effects of pre hypertension, the current study was undertaken to assess risk factors such as for cardiovascular diseases in pre-hypertensive adult males in Uttarakhand region.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted in the Department of Physiology, Himalayan Institute of Medical Sciences

Keywords: BMI, 6MWT, Peak VO_2 , Prehypertensive

(HIMS), Dehradun, over a period of 12 months. The sampling frame comprises of representative group of residents, employees and students of HIHT University. Ethical clearance from the institutional ethical committee was taken before the study. Sample size ($n=44$; each group) was calculated deploying the formula for comparing 'difference of means between the groups' ($\alpha=0.05$, power (β) = 80% and effective size (es) = 0.6) which gave a sample size of $n=44$ [10]. Volunteers (20-40 years) were selected from the aforementioned sampling frame based on the JNC VII criteria and were divided into two groups; Group I (Controls): normotensive and clinically healthy volunteers and Group II (Cases): pre-hypertensive and clinically healthy volunteers. Exclusion criteria included history of diabetes mellitus, endocrine disorders, renal diseases, and diagnosed CVD. Written informed consent was taken from the volunteers before ascertainment of study protocol. They were asked to report in the department around 9:00 am without heavy meals and preferably after a good sleep. Initially, relevant medical history was taken which was followed by the assessment of anthropometric parameters of height, weight, waist and hip circumference. Six minute walk test (6MWT) was then performed for the assessment of exercise capacity. A measured corridor course was used for walking. Subjects were instructed to walk from one end to the other at their own pace attempting to cover as much distance as possible in the allotted 6 minutes time. The walk was timed (6 minutes) and distance travelled in six minutes was recorded in meters. The subjects were allowed to stop and rest if necessary but were encouraged to proceed with the walk on recovery. Exercise capacity was then determined by calculating Work Done and Mean Peak VO_2 from the below mentioned formulae [11]; Work Done (Kg/m) = Distance travelled (m) x Body weight (Kg) and Mean Peak VO_2 (ml / kg / min) = $4.948 + 0.023 \times \text{Mean 6MWD (m)}$ [12]. Salt intake (grams/day) was calculated as daily sodium intake in food using a food frequency questionnaire (FFQ). The questionnaire was tailored as per the regional dietary habits and grouping of food on the basis of similarity profile. Value of sodium intake was calculated from the dietary record on the basis

of cooked food (National Institute of Nutrition) [13] or total no. of packets of salt consumed per month in the family (converted into per day). Composition of top up salt (table salt) per head in the family per day and published recipes for readymade food (wrapped up items) was also taken into account to limit calculation error.

STATISTICAL ANALYSIS

Statistical Analysis was done using SPSS (Statistical Package for the Social Sciences, version 17). The parameters analysed were anthropometric parameters, exercise capacity and salt intake. Independent two sample t-test was used for comparing the means for the above mentioned parameters between the normotensives and pre hypertensives. Pearson correlation was used to study the correlation between the above mentioned parameters. Multiple linear regression analysis was done to evaluate the predictor of baseline SBP after adjusting the effect of age, BMI, WHR, ΔDBP and Peak VO₂.

RESULTS

Risk Factor Analysis of our results showed that the demographic and anthropometric parameters of age, weight, height, BMI and WHR were found higher in pre-hypertensive group as compared to normotensive groups but this difference between the groups was statistically non-significant (p>0.05). Exercise capacity as determined by peak VO₂ consumption was significantly lower in pre-hypertensive group than the normotensive group (p<0.001). Daily salt intake of pre-hypertensives was significantly greater than the normotensive subjects (p<0.001). Multiple Linear Regression analysis reveals that average baseline SBP increases by 0.34 mmHg for every 1 kg increase in weight and average baseline DBP increases by 0.25 mmHg for every 1 year increase in age.

S. No	Parameter	Group I (Normotensives)	ASIA grade B
1.	Age (Years)	34.11±5.69	33.55±5.98
2.	Weight (Kg)	62.25±8.31	63.32±12.59
3.	Height (cm)	166.39±9.98	167.23±6.99
4.	BMI (Kg/m ²)	22.50±2.96	22.68±4.38
5.	WHR	0.90±0.06	0.91±0.44
6.	Resting PR (beats/min)	74.93 ± 7.10	86.16 ± 10.63***
7.	Baseline SBP (mmHg)	106.23 ± 8.19	124.86 ± 8.28***
8.	Baseline DBP (mmHg)	71.55 ± 5.54	83.18 ± 4.87***

[Table/Fig-1]: Comparative demographic, anthropometric and cardiovascular parameters among normotensives & pre hypertensives. Data presented as mean±SE; * mark indicates comparison b/w group I & II; *p < 0.05; **p < 0.01; ***p < 0.001. BMI: Body Mass Index, WHR: Waist Hip Ratio, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure

S. No	Parameter	Group I (Normotensives) n = 44	Group II (Pre-hypertensives) n = 44
1.	Mean 6MWT distance (m)	679.36 ± 9.01	615.75 ± 12.11***
2.	Work Done (Kcal/min)	16.16 ± 0.33	14.78 ± 0.40**
3.	Peak VO ₂ (ml/kg/min)	20.57 ± 0.21	19.11 ± 0.28***
4.	Salt Intake(gm/day)	9.00 ± 0.48	21.18 ± 1.18***
5.	Na+ (gm/day)	3.60 ± 0.19	8.43 ± 0.47***

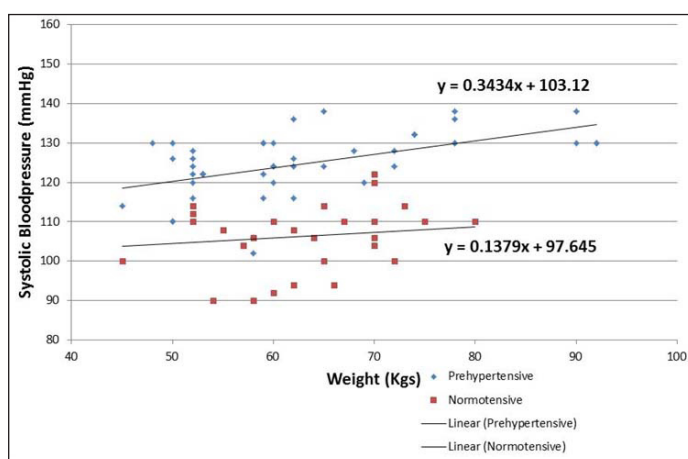
[Table/Fig-2]: Comparative exercise capacity parameters (6MWT) and salt intake among normotensives & pre-hypertensives. Data presented as mean ± SE; * mark indicates comparison b/w group I & II *P < 0.05; **P < 0.01; ***P < 0.001

[Table/Fig-1] shows a slight increase in weight, BMI and WHR among pre-hypertensives as compared to normotensives but this difference observed between the two groups was non-significant (p>0.05). But the cardiovascular parameters i.e. pulse rate, systolic blood pressure and diastolic blood pressure were found significantly higher in pre-hypertensives as compared to normotensive (p<0.001). [Table/Fig-2] compares the exercise capacity parameters and salt in take between normotensives and pre-hypertensives. In pre-

hypertensives, the distance covered in 6 minutes, total amount of work done in walking 6 minutes and peak VO₂ consumption during the test was significantly reduced as compared to normotensives (p<0.001). Also, the daily salt in take of pre-hypertensives was significantly greater than the normotensives, 21.18±1.18 gm/day (Na+= 8.43±0.47 gm/day) and 9.00±0.48gm/day (Na+=3.60±0.19 gm/day) respectively (p<0.001). [Table/Fig-3] shows correlations of age, weight, BMI, WHR, ΔDBP and peak VO₂ with each other among pre-hypertensives. Peak VO₂ exhibited significant negative correlation with anthropometric variables i.e. Age (r = -0.54, p <0.001), Weight (r = - 0.44, p <0.001) and BMI (r = - 0.46, P < 0.001). Also, there was a significant positive correlation between weight and BMI (r = 0.90, p<0.001).

Parameter	Age	Weight	BMI	WHR	ΔDBP	Peak VO ₂
Age	-	r =0.15	r =0.12	r =-0.21	r =-0.17	r =-0.54***
Weight	-	-	r =0.91***	r =0.09	r =-0.01	r =-0.44***
BMI	-	-	-	r =0.08	r =-0.02	r =-0.46***
WHR	-	-	-	-	r =-0.17	r =0.12
ΔDBP	-	-	-	-	-	r =0.11
peak VO ₂	-	-	-	-	-	-

[Table/Fig-3]: Correlation of age, weight, BMI, WHR, ΔDBP and peak VO₂ with each other among pre-hypertensives. Data was analysed using 'Pearson' correlation; 'r' representing Pearson' correlation coefficient; *p < 0.05; **p < 0.01; ***p < 0.001



[Table/Fig-4]: Multiple linear regression analysis for bsbp and weight.

[Table/Fig-4] compares dependency of systolic blood pressure on weight between pre-hypertensives and normotensives using multiple linear regression analysis. Among pre-hypertensives, the slope of best fitted regression line 'β' was estimated as 0.34 which means that average baseline systolic blood pressure increases by 0.34 mmHg for every 1 kg increase in weight. As for normotensives, the slope of best fitted regression line 'β' was estimated as 0.13 which means that average baseline systolic blood pressure increases by 0.14 mmHg for every 1 kg increase in weight. Thus, the increased steepness of the slope and difference in the aforementioned β coefficient value suggests that weight acts as an independent predictor for increase in baseline systolic blood pressure among pre-hypertensives. Similarly, average baseline DBP increases by 0.25 mmHg for every 1 year increase in age.

DISCUSSION

The current study investigating the risk factors associated with raised blood pressure in pre-hypertensive young adult male population revealed higher weight, BMI and WHR in pre-hypertensives as compared to normotensives. Bracho M et al., conducted a prospective randomized control study on 3167 patients and evaluated the role of anthropometric measures as predictors of prehypertension. They found significant higher measurements of

weight, body mass index, hip circumference, waist circumference and hip waist index ($p < 0.001$) among pre-hypertensives with BMI being the strongest predictor of prehypertension [14]. This argument is further supported by Carmem Cristina Beck et al., which determined predictive power of anthropometric indicators as discriminators of high blood pressure. They suggested waist circumference as a predictor of high blood pressure (above 95th percentile), especially in adolescents [15]. Multiple Linear Regression analysis of our results showed a rise of 0.3 mmHg in baseline systolic blood pressure and 0.3 mmHg increase in baseline diastolic blood pressure with every 1 kilogram increase in weight. This pertains to the classic Framingham Study which found 10% rise in body weight with an overall 7 mmHg increase in SBP [16]. And, every kilogram excess body weight loss was associated with a decline of 0.3 mmHg and 0.4 mmHg in SBP and DBP, respectively [17]. In Egypt, blood pressure of 5534 subjects were studied by Azza Mohamed Sarry El-Din et al., in relation to obesity and found that percentages of hypertensive individuals increased with age and degree of obesity emerged as an important risk factor for hypertension [18]. The above mentioned factors cause adjustments in renal and cardiovascular functions such as blunted reflex response of heart rate and lumbar sympathetic nerve activity to volume expansion and thus, enhancing afferent arteriolar reactivity to angiotensin II and catecholamines. These adjustments play a key role in both initiation and maintenance of blood pressure [5,6].

Previously, ample evidence suggests that excess salt intake is positively associated with elevated blood pressure and it can be lowered with reductions in dietary salt [19]. In our study, daily salt intake of prehypertensives (21.2 ± 1.2 gm/day) was significantly greater than normotensive subjects (9.0 ± 0.5 gm/day); $p < 0.001$. This is in close coherence to a study conducted in the hilly population of Tehri Garhwal by Saxena P et al., which found that 22% of hypertensive population was consuming salt > 8 gm/day and 9% between 6-8 gm/day [20]. Our values of salt consumption are on a higher side as per World Health Organization (WHO) salt intake guidelines [21] and can probably be explained on the basis of sample population which mostly included subjects consuming junk food. A meta-analysis by Altun B and Arici Memphasized that salt reduction is associated with 1–5.5 mmHg reduction in SBP with more profound reductions (1.5–6 mmHg) observed in hypertensives [22]. In yet another community trial in Portugal, a salt intake reduction from 21g/day to 12 g/day was associated with a significant decline in blood pressure (13 mmHg in systolic blood pressure and 6 mmHg in diastolic blood pressure) [23]. Excess salt intake poses greater challenge for kidneys to excrete it, failure of which leads to a rise in blood pressure [24]. It is worth mentioning that kidney's capacity to excrete sodium declines with age and thus small increase in salt intake induces greater arterial pressure [25]. This can be in part due to an accelerated fall in glomerular filtration rate (GFR) with age which begins around the age of 30 years and declines to almost 40% at 80 years and also due to attenuation in the number of functioning nephrons. As there is no reduction in salt consumption with age, sodium balance is maintained by increasing fractional excretion of sodium by increased ANP and decreased angiotensin and aldosterone [26]. Thus, owing to these involuntal and structural changes in kidney, sodium retention happens once salt consumption exceeds the compensatory mechanism of sodium excretion.

Relationship between physical fitness (maximal oxygen uptake VO_{2max}) and incidence of hypertension was known for quite some time. The Six-Minute Walk Test (6MWT) is an inexpensive, quick, and a well-tolerated method of assessing physical fitness by measuring functional exercise capacity [12]. In our study, we found peak VO_{2} consumption significantly lower in pre-hypertensive (19.1 ± 0.3 ml/kg/min) than in normotensive group (20.6 ± 0.2 ml/kg/min) ($p < 0.001$). We have also calculated the work done as product of distance and

body weight to estimate functional exercise capacity and found that the total amount of work done in walking 6 minutes by the pre-hypertensive group (14.8 ± 0.4 Kcal/min) was significantly lower than the normotensive group (16.2 ± 0.3 Kcal/min; $p < 0.001$). This is in concert with a five year prospective Japanese study by Sawada S et al., who enrolled 3305 normotensive subjects and used VO_{2max} to assess their fitness levels. After 5 years of follow up, relative risk of hypertension (425 subjects) was found to be 1.9 times higher in the least fit group compared with the fittest group thus concluding that low VO_{2max} level is related to higher hypertension incidence and an improved VO_{2max} will perhaps prevent development of hypertension [27].

LIMITATIONS

Limitations of our study include that we have to rely on the subjects about quantification of salt consumption. Also, for the assessment of salt intake, owing to feasibility criteria we have used 24 hours recall questionnaire instead of the conventional 1 week diet intake chart.

CONCLUSION

It enlightens some of the modifiable risk factors in pre-hypertensives and in future if performed more systematically will surely identify risk modifiers and predict individual therapeutic benefits.

REFERENCES

- [1] Yadav S, Boddula R, Genitta G, Bhatia V, Bansal B, Kongara S, et al. Prevalence & risk factors of pre-hypertension & hypertension in an affluent north Indian population. *Indian J Med Res.* 2008;128(6):712-20.
- [2] Vasan RS, Larson MG, Leip EP, Kannel WB, Levy D. Assessment of frequency of progression to hypertension in non-hypertensive participants in the Framingham Heart Study: a cohort study. *Lancet.* 2001;358:1682-86.
- [3] The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. <http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.pdf> /Accessed 2nd October 2011.
- [4] Pitzalis MV, Iacoviello M, Massari F, Guida P, Romito R, Forleo C, et al. Influence of gender and family history of hypertension on autonomic control of heart rate, diastolic function and brain natriuretic peptide. *J Hypertens.* 2001;19(1):143-48.
- [5] Dogru MT, Simsek V, Sahin O, Ozer N. Differences in autonomic activity in individuals with optimal, normal, and high-normal blood pressure levels. *Turk Kardiyol Dern Ars.* 2010;38(3):182-88.
- [6] Pal GK, Pal P, Nanda N, Lalitha V, Dutta TK, Adithan C. Sympathovagal imbalance in young prehypertensives: Importance of male-female difference. *Am J Med Sci.* 2013;345(1):10-17.
- [7] Manson JE, Hu FB, Rich-Edwards JW, et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. *N Engl J Med* 1999; 341(9):650-58.
- [8] Blair SN, Kohl HW III, Barlow CE, Paffenbarger RS, Gibbons LW, Macera CA. Changes in physical fitness and all-cause mortality. A prospective study of healthy and unhealthy men. *JAMA.* 1995;273(14):1093-98.
- [9] Faselis C, Dourmas M, Kokkinos JP, Panagiotakos D, Kheirbek R, Sheriff HM, et al. Exercise capacity and progression from prehypertension to hypertension. *Hypertension.* 2012;60(2):333-38.
- [10] Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Lawrence Erlbaum Associates, Publishers. London; 1988: Pp. 550.
- [11] Enright PL. The Six-Minute Walk Test. *Respiratory Care.* 2003;48(8):783-85.
- [12] Carter R, Holiday DB, Nwasuruba C, Stocks J, Grothues C, Tiep B. 6-minute walk work for assessment of functional capacity in patients with COPD. *Chest.* 2003;123(5):1408-15.
- [13] Gopalan G, Rama BV, Balasubramanian S C. Nutritive value of Indian foods. 1st ed. National Institute of Nutrition, Hyderabad; 1989 Pp. 68-73.
- [14] Bracho M, Esis C, Silva E, Villasmil J, Gonzalez A, Bermudez G, et al. Anthropometric measures as predictors of prehypertension in Adolescents. *Journal of Hypertension.* 2010;28:e479-80.
- [15] Beck CC, Lopes Ada S, Pitanga FJ. Anthropometric indicators as predictors of high blood pressure in adolescents. *Arq Bras Cardiol.* 2011;96(2):126-33.
- [16] Ashley FW and Kanne WB. Relation of weight change to changes in atherogenic traits. The Framingham Study. *J Chronica Dis.* 1974;27:103-04.
- [17] Stevens VJ, Corrigan SA, Obarzanek E, Bernauer E, Cook NR, Hebert P, et al. Weight loss Intervention in phase 1 of the trials of hypertension prevention. *Arch Intern Med.* 1993;153(7):849-58.
- [18] El-Din AMS, Erfan M, Kandeel WA, Kamal S, El-Banna RAE, Fouad WA. Prevalence of Pre hypertension and Hypertension in a sample of Egyptian Adults and its Relation to Obesity. *Australian Journal of Basic and Applied Sciences* 2012;6(13):481-89.

- [19] Meneton P, Jeunemaitre X, de Wardener HE, MacGregor GA. Links between dietary salt intake, renal salt handling, blood pressure, and cardiovascular diseases. *Physiol Rev.* 2005;85(2):679-715.
- [20] Saxena P, Saxena V, Saxena Y. Bio-social factors associated with hypertension in hilly population of Tehri Garhwal. *Indian Journal of community health.* 2011;23(2):81-3.
- [21] World Health Organization (WHO). Guidelines: Sodium intake for adults and children. http://www.who.int/nutrition/publications/guidelines/sodium_intake_printversion.pdf. Accessed 10th August 2013.
- [22] Altun B, Arici M. Salt and blood pressure: time to challenge. *Cardiology.* 2006;105:9-16.
- [23] Forte JG, Miguel JM, Miguel MJ, de Padua F, Rose G. Salt and blood pressure: a community trial. *J Hum Hypertens.* 1989;3:179-84.
- [24] He FJ, MacGregor GA. A comprehensive review on salt and health and current experience of worldwide salt reduction programmes. *J Hum Hypertens.* 2009;23(6):363-84.
- [25] Lindeman RD, Tobin JD, Shock NW. Association between blood pressure and the rate of decline in renal function with age. *Kidney Int.* 1984;26:861-68.
- [26] Kasiske BL. Relationship between vascular disease and age-associated changes in the human kidney. *Kidney Int.* 1987;31:1153-59.
- [27] Sawada S, Tanaka H, Funakoshi M, Shindo M, Kono S, Ishiko T. Five year prospective study on blood pressure and maximal oxygen uptake. *ClinExpPharmacol Physiol.* 1993;20(7-8):483-87.

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