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Research Article

Sociodemographic Factors Associated With Blood Pressure and Serum Lipids in Healthy Indians

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ABSTRACT

Background: Cardiovascular diseases are the largest cause of mortality worldwide and in India the incidences of CVDs have increased for people between the ages 25-69, thus losing more productive people to these diseases.

Objective: To study the effect of age and gender on blood pressure, serum lipids and anthropometric indices among normal healthy individuals.

Material and methods: The study population consisted of 400 normal healthy individuals divided into two age-groups: 30-50years and 51-80 years. Exclusion criteria included evidence of hypertension (diastolic BP > 80 mm Hg) and use of medication. Blood samples were drawn from all the subjects following an overnight fast and TC, TG and HDL-C were measured enzymatically using "Accucare" kits. Serum LDL-cholesterol (LDL-C) was calculated by Fredrickson-Friedwald formula. Blood pressure (BP) and anthropometric measurements were measured by a standardized protocol. Statistical analysis was done using the students -'t' test.

Results: BP, serum lipids, atherogenic ratios and WHR increased with age in both genders and this rise was significant ($P<0.05$) except in the parameter TG. Also HDL-C in males, atherogenic ratios in females and BMI showed a decrease with age but this decrease was insignificant. Moreover, in both the age groups: 30-50 and 51-80, females were having lower BP, serum lipids and WHR than those of males of corresponding age groups except HDL-C and BMI. This difference between male and female in both the age groups was found to be extremely significant. ($P<0.001$)

Conclusions: In our study, both age and gender are important atherogenic factors, which indicate that with increase in age, the older individuals both male and female are more susceptible to the development of HT and CVDs.

Keywords: Age, blood pressure, BMI, gender, serum lipids, WHR.

1. INTRODUCTION

Cardiovascular diseases (CVD) in developing countries are characterized by early age of onset and greater mortality [1]. India suffers the highest loss in potentially productive years of life, as deaths due to CVD in persons in the age group of 35–64 years are high [2]. It is predicted that by the year 2015, India will have the largest burden of CVD in the world [3]. The growing burden of CVD [4] is due to the increasing prevalence of modifiable and non-modifiable cardiovascular risk factors such as age, gender, hypertension (HT), dyslipidaemia, overweight or obesity, diabetes, physical inactivity and use of tobacco. It is estimated that 30-50% of the world population above 20 years of age is afflicted with HT [5]. Recent data from the Framingham Heart Study suggest that individuals who are normotensive at age 55 have a 90% lifetime risk for developing HT [6]. The INTERHEART Study [7] reported that the mean age of myocardial infarction in South Asians was 52 years as compared to 60–65 years in European and North American cohorts. Thus CVDs has been documented across all age groups and both sexes from India and different parts of world. However there is paucity of data on blood pressure and serum lipid status among normal healthy individuals from the Saurashtra population of Gujarat. With this background, the present study was conducted to study the effect of age and gender on blood pressure, serum lipids and anthropometric indices among normal healthy individuals.

2. MATERIAL AND METHODS

2.1. Study population

The study population included subjects from Jamnagar city of Gujarat with varied lifestyle based on their occupation. A total of 400 healthy people (217 women and 183 men) participated in the study and informed consent was obtained from all participants. Individuals' ≥ 30 years of age were included in the study. Random subjects free of not only HT but any disease like Diabetes, CVD and also who were not on any particular medication were included in the study. HT was

considered to be present if the SBP ≥ 140 mm Hg or the DBP ≥ 90 mm Hg [8].

2.2. Measurement of blood pressure

The BP was measured by a doctor using a mercury column sphygmomanometer by a standardized protocol in the sitting posture, with feet on the floor and arm supported at heart level. Following the standardized technique, the doctor made two separate measurements after making the patient take proper rest. In some cases, where high BP was recorded for the first time, the physicians checked the BP more than twice and took the average of the two close readings.

2.3. Collection of samples

5 ml of venous blood was withdrawn after an overnight fast of 12 hrs and was allowed to clot for 25-30 minutes for proper serum extraction after centrifuging for 5 minutes. This serum was used to estimate the parameters within two hour of sample collection.

2.4. Biochemical markers

Total cholesterol was measured by CHOD-PAP method, serum triglyceride by GPO-PAP method and HDL-C by HDL-C plus method using "Accucare" kits. The test was carried out according to the manufacturers' instructions. Measurements of serum cholesterol, triglycerides and HDL-C, were done on the ERBA semi-auto analyzer (Germany). LDL-C concentration was calculated using Fredrickson-Friedwald formula [9]. The cut-off values for abnormal lipid levels were considered according to the National cholesterol education programme (NCEP) guidelines Adult treatment panel III (ATP III) [10]. Atherogenic ratios [TC/ HDL] >5 and [LDL-C/HDL-C] >3.5 was taken as elevated.

2.5. Anthropometric measurements

A weighing (bathroom) scale and stadiometer were used to measure the weight (nearest 0.5 kg) and height (nearest 0.1 cm) of each subject using standard procedure. Body Mass Index (BMI) (weight in kg/ height in m^2) was calculated. The waist and hip circumference was measured to the nearest 0.5 cm in the subjects standing barefoot and wearing light clothes. The waist circumference was measured at the level of the umbilicus and the hip at the level of the greater trochanter using a non-stretchable measuring tape. Waist-hip-ratio (WHR) was calculated from the data.

2.6. Statistical analysis

Two separate analyses were performed, one by age group (30 - 50 and 51-80 years) and another by gender. Mean, standard deviation and standard error was calculated. Student't' test was applied using Minitab statistical software. '*P*' values were calculated to assess significance of the results.

3. RESULTS

In this study 400 subjects were studied. The subjects were divided into the two genders, male and female within two age-groups: 30-50 years and 51-80 years.

The BP increased with age in both genders (Table 1) and this rise was extremely significant in DBP ($P<0.001$) (Table 2) while SBP showed a minor significance ($P<0.05$) (Table 2). Moreover in all age groups females were having lower SBP and DBP than those of males of corresponding age groups (Table 1). The difference in SBP between the gender in the age group 51-80 was found to be highly significant ($P<0.01$) whereas DBP exhibited massive significance ($P<0.001$) in both the age groups.

Table 1: Effect of Age on BP, Serum Lipids, BMI and WHR in Normotensive cases

Age Group	G	N	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	VLDL-C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
30-50 (A)	M	110	117.05 ± 0.71	74.33 ± 0.46	176.0 ± 1.6	116.5 ± 3.8	41.6 ± 0.97	111.2 ± 0.97	23.31 ± 0.75	4.5 ±0.12	2.9 ±0.1	25.07 ± 0.33	0.93 ± 0.008
	F	163	116.23 ± 0.71	72.66 ± 0.3	171.8. ± 1.5	93.9 ± 2.8	46.5 ± 0.83	106.5 ±1.6	18.7 ± 0.57	3.88 ± 0.07	2.45 ± 0.06	25.39 ± 0.32	0.82 ± 0.005
51- 80 (B)	M	73	119.12 ± 0.49	78.58 ± 0.84	186.5 ± 2.7	125.1 ± 6.3	41.6 ± 1.4	119.8 ± 2.8	25.0 ± 1.3	4.86 ± 0.2	3.18 ± 0.15	24.19 ± 0.63	0.95 ± 0.007
	F	54	117.65 ± 0.65	75.41 ± 0.46	179.4 ± 1.8	97.6 ± 8.0	50.0 ± 1.5	109.9 ± 2.8	19.5 ± 1.6	3.77 ± 0.13	2.35 ± 0.12	24.54 ± 0.4	0.83 ± 0.007

Values are Mean ± S.E. M= Male; F= Female; N= Number of population

In male subjects, serum lipids and atherogenic ratios except TG and HDL-C increased with age and this increase was highly significant ($P<0.01$) (Table 2). While in female subjects only TC and HDL-C showed a tremendous significance by increasing with age. Both the age groups 30-50 and 51-80 years showed a difference in all serum lipid concentration and atherogenic ratios between the genders (Table 1) and this difference was greatly significant ($P<0.01$) (Table 2).

The HDL-C concentration increased with age in the females (Table 1) which was immensely significant ($P<0.01$) (Table 2). Moreover in both the age groups females were having higher HDL-C concentration than those of males of corresponding age groups (Table 1) and this difference was particularly significant ($P<0.001$) (Table 2). Among anthropometric indices the values of BMI decreased with age (Table 1) but vague significance was seen in females only ($P<0.05$). WHR increased with age (Table 1) and this rise was found to be slightly significant ($P<0.05$) (Table 2). Also the difference according to gender in WHR was seen to be extremely significant ($P<0.001$) (Table 2) in each of the age groups.

Table 2: Statistical Analysis of Table 1

Comparison	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	VLDL-C mg/dl	TC/ HDL-C	LDL-C/HDL-C	BMI	WHR
AM vs AF	NS	P< 0.001	P< 0.01	P< 0.001	P< 0.001	P< 0.05	P< 0.001	P< 0.001	P< 0.001	NS	P< 0.001
BM vs BF	P< 0.01	P< 0.001	P< 0.01	P< 0.001	P< 0.001	P< 0.01	P< 0.001	P< 0.001	P< 0.001	NS	P< 0.001
AM vs BM	P< 0.01	P< 0.001	P< 0.001	NS	NS	P< 0.01	NS	P< 0.05	P< 0.05	NS	P< 0.05
AF vs BF	P< 0.05	P< 0.001	P< 0.001	NS	P< 0.01	NS	NS	NS	NS	P< 0.05	P< 0.05

NS= Non Significant

4. DISCUSSION

In the present study the effect of age and gender on BP, serum lipids and anthropometric indices were studied on normal healthy individuals. Aging is the biological process common to all living organisms. It is a non-modifiable, but major risk factor in the anticipation of HT risk. The relationship between BP and risk of CVD events is continuous, consistent and is further increased in the presence of dyslipidaemia.

In the present study, SBP and DBP have been found to increase with age in both sexes as shown in table 1. These findings were in agreement to the third NHANES and Framingham data wherein a rise in SBP, with advancement of age in both genders was observed [11, 12]. In this study it was also found that SBP and DBP were higher in males than in females of the corresponding age group. There seems to be a positive correlation between BP and gender differences. It may be due to the protective effect of female sex hormones against stress. In agreement to this study, Shyamal Das *et al* also showed an influence of gender on BP among men [13]. The progressive urbanization, lifestyle modification and sedentary habits are probably some of the important factors considered to be responsible for increase in BP with advancement of age in Jamnagar city of Gujarat. Also structural, functional and biochemical changes occurring in the vascular system of human body due to aging process are some other factors which are again influenced by sex, race socioeconomic, etc. [13]

Serum lipids (cholesterol, triglyceride) and lipoprotein levels (VLDL, LDL, HDL) do have immense importance in coronary artery disease patients because variation from normal levels can predict the cause of CAD to a great extent [14]. In the present study an increase in the serum lipids (TC &TG) and lipoproteins (VLDL, LDL) are observed with the advancement of age in both sexes. A similar observation was revealed by Ritu Sharma *et al*. [14]. According to the study by Reddy [15] TG showed a slight increase with increase in age, but females were possessing higher TG values than males. While in this present study females always showed a value significantly lower to the males of the corresponding age groups of serum lipids (TC & TG) and lipoproteins (VLDL-C & LDL-C) (Table 2) which was in agreement with LRC data for North American population [16] and Reddy's [15] findings for urbanization of Andhra Pradesh.

HDL-C levels in the present study did not vary with increase in age in males while it increased in females. (Table 1) In females the elevated HDL-C may be due to estrogens and thus they might have lower incidence of CAD before the age of 50 [17]. Also a study by B.M. Gandhi [18] showed a marginal increase of HDL-C with age but the difference between the two sexes was insignificant while in our study the difference between the gender where highly significant. Abdul Rehman [19] showed an average serum HDL-C level of 58.4% with significant variation between age groups 20 to 49 years in Kuwaiti males. Factors associated with weight change during puberty due to differential effect by gonadal hormones, smoking and sucrose intake play a major role in the difference in HDL-C and TC concentration according to sex [20]. Thus

the sex difference in lipoproteins may be resulted from difference in the effects of factors that influence lipoprotein levels between the two sexes. In communities like Japanese and Greenland Eskimos [21] with little or no CHD had minimal sex difference in lipoprotein levels which was in absolute agreement to this study.

Age and Sex related changes in LDL-C levels documented in this study closely resemble those for total plasma cholesterol. (Table 1) This is because 70% of TC is transported in this lipoprotein in the ultra centrifugal low density range. LDL-C in males and females increased with age in our study which was in agreement with B. M. Gandhi *et al*. [18] Brown and Goldstein [22] reported that increase in LDL-C with age is a consequence of a reduction in LDL-C receptors mediated catabolism due to decrease in numbers of LDL-C receptors with age. VLDL-C concentration demonstrated little but gradual change with age in males and females and resembles almost with respect to TG as VLDL-C was estimated with the use of Friedwald formula. Increased concentration of LDL-C in the plasma is the primary factor for increase in TC while a small part is due to an increase in VLDL-C with age which is also a form in which a little amount of cholesterol is transported. In women, LDL-C and VLDL-C concentration increases more slowly, thus accounting for the major differences in the genders [23] in plasma TC concentration.

According to Simons [24] in an individual population TC and HDL-C estimations were enough to predict the risk for CHD. In a study by K.P. Misra *et al* [25] it emphasizes the significance of HDL-C and more so of the ratios of TC/HDL-C and LDL-C/HDL-C as major lipid risk factors for CHD. This is understandable in view of the proposed hypothesis that HDL facilitates the uptake of cholesterol from peripheral tissues and helps in its transport to the liver for degradation and excretion [26]. Therefore for efficient transport of tissue cholesterol to the liver the proportion of HDL-C to TC and LDL-C is more important than the individual values of any of these. Also TC/HDL-C and LDL-C/HDL-C were used to compare and explain the international differences observed in the rate of development of CHD in various population studies. In this study the data confirmed the rise in these atherogenic ratios in the males with increase in age.

Obesity predispose to many disease conditions like HT, diabetes, coronary and vascular disease. The percent of fat in a young individuals body weight is somewhere between 15 to 25% and this tends to increase with age which may not be desirable. When the body fat content is greater than 25 to 30%, an individual is termed to be obese. Measuring the BMI and WHR in an individual is necessary to identify them as obese. In this study BMI decreased with age but showed no significance while WHR increased with age. Obesity has more recently been shown to decrease life expectancy by 7 years at the age of 40 years [27]. The increase in risk of death with each unit increase in BMI declines progressively with age but remains substantial until the age-group of 75 years and older [28]. Central adiposity is a predictor of CVD independently of major risk factors, including BMI [29]. Also dyslipidaemic individuals are more frequently "centrally obese" i.e. with a high WHR [30].

5. CONCLUSION

The subjects showed a positive correlation with BP and serum lipids while HDL-C showed a negative correlation with the advancement of age in both genders. Thus, age is an important atherogenic factor, which indicated that with increase in age, the older individuals are more susceptible to the development of HT and CVDs. In relation to gender, males had values higher to females in all the parameters but still both are prone to develop HT and atherosclerotic disease as the values were above normal in both the genders. Thus gender specific increase of development of HT in both men and women indicate significant role of environmental factors. Thus the non-modifiable structural, metabolic and hormonal change in the body due to aging can be one of the factor which accounts for an increase in BP, dyslipidaemia and obesity. As Indians have a higher prevalence of premature CVD, it would be useful to assess the prevalence of risk factors of CVD among the population at a young age itself.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the authorship and/or publication of this article.

6. REFERENCES

1. Beaglehole R, Yach D. *Lancet,* 2003; **362:** 903-908.
2. Leeder SR, Raymond SU, Greenberg H, Lie H, Esson K. A race against time: The challenge of cardiovascular disease in developing economies. New York: The Center for Global Health and Economic Development, Columbia University; 2004.
3. Reddy KS, Yusuf S. *Circulation,* 1998; **97:** 596–601.
4. Sanderson JE, Mayosi B, Yusuf S, Reddy S, Hu S, Chen Z, *et al. Heart,* 2007; **93:** 1175.
5. Wasir HS Hypertension- The need for Primary prevention. Regional Health Forum (WHO SC-Asia). 1996; 1: 1.
6. Yasan RS, Beiser A, Seshadri S. *JAMA,* 2002; **287:** 1003-10.
7. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, *et al*.**:** *Lancet,* 2004; **364:** 937-952.
8. Chobanian AV, Bakris GL, Black HR. *JAMA,* 2003; **289:** 2560-2572.
9. Friedwald WT, Levy RI and Fredrickson DS. *Clin Chem,* 1972; **18:** 499.
10. Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High

11. Blood Cholesterol in Adults (Adults Treatment Panel III). *JAMA,* 2001; 285: 2486-2497.
12. Burt NL, Whelton P, Rocella EJ. *Hypertension,* 1995; **25:** 305-13.
12. Franklin SS, Gustin WT, Wong ND. *Circulation,* 1997; **96:** 308-15.
13. Das SK, Sanyal K, Basu A. *Int J Med Sci*, 2005; **2:** 70-78.
14. Ritu S, Mridula M and Ravi K. *Indian J Clin Biochem,* 2004; **19:** 10-13
15. Reddy KK, Bulliyya. *Ind J Clinical Biochem,* 1990; **5:** 18-29.
16. Lipids Research Clinics Program Prevention Study. LRCPPC trial. Plasma lipid distributions in North American White population. *Circulation,* 1979; 60: 427-439.
17. Third Report of the NCEP Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Available at: http://www.nhibi.nih.gov.
18. Gandhi BM. *In J Med Res,* 1982; **75:** 393-401.
19. Abdul RS. *Med Principle Practice,* 1990; **2:** 199-202.
20. Robert L, Donald CT, Israel Tamir. *Circulation,* 1980; **62(suppl IV):** 83-92.
21. Eskimos diets and diseases editorial. *Lancet,* 1983; 1: 1139-1141.
22. Brown MS, Goldstein JL. *J Clin Invest,* 1983; **72:** 743-747.
23. Heiss G, Tamir I, Davis CE. *The LRCPPS Circulation,* 1980; **61:** 302-315
24. Simons LA. *Am J Cardiology,* 1986; **57(suppl G):** 5G-10G.
25. Misra KP, Suresh S, Balachandran G, *et al. J Assoc Phys Ind,* 1980; **28:** 399-404.
26. Miller GJ, Miller NE. *Lancet,* 1976; **1:** 16.
27. Peters A, Barendregt DT, Wang C, *et al. Ann Intern Med,* 2003; **289:** 187-93
28. Stevens J, Cai J, Panuk ER. *N Engl J Med,* 1998; **338:** 1-7.
29. Han TS, Van Leer EM, Scidell JC, *et al. BMJ,* 1995; **311:** 1401-1405.
30. Bjorntorp P. *Ann Int Med*, 1985; **103:** 994-995.