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A CROSS SECTIONAL STUDY OF ANTHROPOMETRIC, BLOOD PRESSURE PARAMETERS AND METABOLIC PROFILE AMONG MEDICAL STUDENTS-GENDER DIFFERENCE DETERMINES CARDIOVASCULAR RISK

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ABSTRACT:

Background: *There The presence of obesity and dyslipidaemia has also been reported to be higher in medical students, which requires further investigation.* ***Aims****: To study gender differences on anthropometric BP and lipid profile parameters in medical students* ***Methods****: One thirty one healthy undergraduates (age 19 ± 1 year) were recruited. After obtaining informed consent and IEC clearance, height, weight, BP, heart rate, waist circumference, hip circumference, waist-hip ratio calculated, fasting blood sugar, serum cholesterol, triglycerides and high density lipoproteins was estimated.* ***Results****: WC and HC was significantly higher in males (P=0.001) as compared to females, with males having higher height and weight compared to females. Males had significantly higher SP (P=0.001), DP (P=0.02) and heart rate as compared to females. There was no significant difference in FBG, TC, HDL, LDL levels. TG and VLDL were significantly higher in males (P=0.001 for both) as compared to females. Significant negative correlation of BMI and HDL (P=0.02) was observed in males* ***Conclusions****: Gender difference plays a role in protection against many diseases. The loss of gender difference in females with regard to biochemical parameters may predispose them to the risk of CVD at an early age.*

KEYWORDS:

Cardiovascular risk factors, Gender difference, Obesity.

INTRODUCTION

A never ending field of inquiry centres on gender-related differences like, “Why do women live longer than men?”, “Why do postmenopausal women have increased risk of coronary artery disease (CAD)? These facts reinforces the importance of gender-specific research in medicine.[1] There is a vast difference between men and women with respect to structure and function of different organ systems.[2] The risk factors, presenting symptoms, testing modalities, therapeutic interventions made for women are different from those for men.[3]

Ischemic heart disease and stroke are the most common non-communicable cause of death worldwide.[4] Mortality due to cardiovascular disease (CVD) has been reported to be on the decline in developed countries, whereas in developing countries it has been reported to increase during same period.[5] Indian subcontinent has 20% of the world’s population and is said to be one of the regions with highest burden of CVD, also, CVD manifests almost 10 years earlier in this region.[4]

The prevalence of diabetes mellitus (DM) has been reported to be higher in developing countries [4] and also, its incidence is known to increase by about 30-fold over the past 20 years among youth.[6] DM, hypertension, chronic kidney disease, dyslipidaemia, smoking, alcohol consumption, lack of physical activity are some of the risk factors that has increased the burden of CVD.[4]

Simple anthropometric measurements that have been used as index of obesity have more practical value in both clinical practice and for large-scale epidemiological studies. [7] The childhood roots of adult obesity and also CVD are widely recognized and calls for health promotion targeted at youth. Moreover, there are very few studies on the health status of medical students, a target group of particular interest as they are future physicians.[8]

MATERIALS AND METHODS

Study design: One hundred student volunteers between the age group of 19 ± 1 year (50 males, 50 females) were enrolled for this study.

Study type: Cross sectional study.

The subjects with cardiovascular disease, those on medications, bronchial asthma and any other illness that affects lipid profile and fasting blood glucose were excluded from study. Ethical clearance was obtained from institute ethics committee. The study was explained in detail to the subject and a written informed consent was taken from all subjects.

Measurement of BP indices:

Participants were asked to avoid tea or coffee or do vigorous work for about 30 minutes on the day of the study. The participants were allowed to rest for five minutes before the recording was done. BP was then measured in sitting position in both the arm of the subject and higher of the two readings was taken as actual BP. The BP was measured three times with the cuff completely evacuated and recovery allowed between readings. The average of the readings was used as, systolic and diastolic BP. Pulse pressure was calculated as Systolic BP – Diastolic BP in mm Hg. Mean arterial pressure was calculated as Diastolic BP + 1/3 Pulse pressure in mm Hg**.** Heart rate is counted for one full minute expressed as beats/ minute. Rate pressure product was calculated as (Systolic BP x Heart rate) / 100.[9]

Measurement of anthropometric parameters:

Standing height was measured using a stadiometer with a fixed vertical backboard and an adjustable head piece using standard technique**.** The head piece is locked in place and the standing height was recorded in centimetres to the nearest 0.1 cm. Weight was recorded using a digital weighing scale to the nearest of 0.1 kg. WC was measured at the level of right iliac crest using a non-stretchable measuring tape extended around the waist in a horizontal plane. Measurement was taken to the nearest 0.1 centimeters at the end of the normal expiration. HC was measured at maximum extension of the buttocks. Measurement was taken to the nearest of 0.1 centimetres. BMI is computed as using Weight (kg) / height^2 (meters^2). WHR was calculated as WC divided by HC.[9]

Measurement of biochemical parameters:

Venous blood samples (5 ml) were collected from 100 subjects after an overnight fast for determination of FBG and serum lipid profile. Estimation of FBG, TC, TG and HDL was carried out using enzymatic method. TC/ HDL and TG/ HDL ratios were subsequently calculated. LDL was calculated using Freidewald's formula[10]. LDL= TC – (HDL + TG/5). VLDL was calculated using Freidewald's formula. VLDL= TG /5. Quantitative estimation was done using semiauto analyzer (Lab Life Chem. Master) Rev: 1.3 C. All the metabolic profiles were estimated in mg/ dl.

Statistical analysis:

Statistical software for social sciences (SPSS) version 16 for windows was used for statistical analysis. Intergroup comparison was done using students unpaired t test. Mann-Whitney U test was used for the analysis of biochemical parameters as the data was highly skewed. Pearson’s test was used in studying correlation between parameters. ‘P’ value ≤ 0.05 was considered as statistically significant.

RESULTS

The present study is a cross sectional study among 100 subjects (50 males and 50 females). They were matched for age (males: 19 ± 4 years & females: 19 ± 4 years) and BMI.

Table-1: Age and anthropometric parameters of the two study groups

Variables	Males Mean ±SD	Females Mean ±SD	P value
N	50	50	
Age (yrs)	19 ± 4	19± 4	0.20
Height (meters)	1.70± 0.08	1.57± 0.05	0.001
Weight (kg)	67± 9	58± 11	0.001
BMI (kg/meter^2)	23± 3	24 ± 4	0.25
WC (centimetres)	79± 10	71± 9	0.001
HC (centimetres)	95± 8	92± 10	0.04
WHR	0.8± 0.06	0.8± 0.05	0.001

BMI: Body mass index; WC: Waist circumference; HC: Hip circumference; WHR: Waist hip ratio. Data expressed as mean ± SD. P value ≤ 0.05 considered statistically significant.

Table-1 shows the age and anthropometric parameters in the two study groups. There was a statistically significant difference in height and weight between the two groups with males having higher height and weight compared to females (P= 0.001 for both). WC and HC was also significantly higher in males (P= 0.001 and P= 0.04 respectively).

Table-2: BP indices in the two study groups

Variables	Males Mean ±S.D	Females Mean ±S.D	P value
Systolic BP (mm Hg)	122±10	116±10	0.001
Diastolic BP (mm Hg)	79± 9	75± 9	0.02
Pulse pressure (mm Hg)	43± 8	41±10	0.06
Heart rate (beats/ minute)	74± 5	71± 5	0.001
RPP	91±12	83± 11	0.001

BP: Blood pressure; RPP: Rate pressure product. Data expressed as mean +/- SD. P value ≤ 0.05 considered statistically significant.

Table-2 shows the BP indices in the two study groups. Males had significantly higher systolic and diastolic BP (P= 0.001 & P = 0.02 respectively) as compared with female subjects. Heart rate and RPP was also significantly higher in males (P = 0.001 for both). No significant difference in pulse pressure was observed between the two study groups.

Table-3: FBG and lipid profile parameters in the two study groups

Variables	Males Mean ±S.D	Females Mean ±S.D	P value
FBG (mg/dl)	88± 10	88± 11	0.88
TC (mg/dl)	161±25	163± 31	0.94
TG (mg/dl)	92±37	82±64	0.001
HDL (mg/dl)	51±9	53±8	0.19
LDL (mg/dl)	96±26	95±30	0.63
VLDL (mg/dl)	19±7	15±6	0.001
LDL /HDL	1.93±0.67	1.84±0.7	0.42
TG/HDL	1.06±0.64	1.58±1.05	0.001
TC/HDL	3.24±0.79	3.14±0.77	0.45

FBG: Fasting blood glucose; TC: Total cholesterol; TG: Triglycerides; HDL: High density lipoprotein cholesterol; LDL: Low density lipoprotein cholesterol; VLDL: Very low density lipoprotein cholesterol. Data expressed as mean ± SD. P value ≤ 0.05 considered statistically significant.

Table-3 shows FBG and lipid profile parameters in the two study groups. TG and VLDL was found to be significantly higher in males as compared to females (P = 0.001 for both). TG/ HDL ratio was significantly higher in females (P = 0.001). No statistical difference was observed for FBG, TC, HDL, LDL, LDL / HDL, and TC/ HDL between either genders.

Table-4: Correlation of BMI with anthropometric, biochemical, BP parameters:

Correlation of BMI with	Males	Females
WC	0.84**	0.52**
HC	0.83**	0.53**
HDL	-0.27	
LDL		0.25*
TC		0.34**
FBG		0.34**
SP		0.28**
DP		0.31**

WC: Waist circumference; HC: Hip circumference.

HDL: High density lipoprotein cholesterol; LDL: Low density lipoprotein cholesterol; TC: Total cholesterol; FBG: Fasting blood glucose. P value ≤ 0.05 considered statistically significant.

Table-4 shows correlation of BMI with anthropometric, BP and metabolic parameters. A significant positive correlation of BMI with WC and HC was observed in both males and females (P = 0.001). A significant positive correlation of BMI with systolic and diastolic BP was observed in females with P = 0.03, P = 0.02 respectively. A significant negative correlation of BMI with HDL was observed in males (P = 0.02). Also, there was a significant positive correlation of BMI with LDL, TC and FBG in females with P= 0.05, P= 0.01, P = 0.01 respectively.

Table-5: Correlation of WC with biochemical, BP parameters:

Correlation of WC with	Males	Females
VLDL	0.25*	
DBP		0.36**
RPP		0.25*

VLDL-C: Very low density lipoprotein cholesterol. RPP: Rate pressure product. P value ≤ 0.05 considered statistically significant.

Table-5 shows correlation of WC with BP, biochemical parameters. A significant correlation of WC with diastolic BP and RPP was observed in females with P= 0.01 and P = 0.05 respectively. A significant correlation of WC with VLDL-C was observed in males with P = 0.04.

Table-6: Correlation of HC with biochemical, BP parameters:

Correlation of WC with	Males	Females
HDL	-0.25*	
VLDL	0.26*	
LDL/HDL		0.26*
TC/HDL		0.26*
DBP		0.29*

TC: Total cholesterol; HDL: High density lipoprotein cholesterol; LDL: Low density lipoprotein cholesterol; VLDL: Very low density lipoprotein cholesterol. P value ≤ 0.05 considered statistically significant.

Table-6 shows correlation of HC with biochemical parameters. A negative correlation of HC with HDL was observed in males (P = 0.04). A significant positive relation was observed between HC and VLDL in males (P = 0.03). A significant positive correlation between HC and LDL/HDL, TC/HDL was observed in females with P= 0.04, P= 0.04 respectively.

TABLE-7: Correlation of diastolic BP with Heart rate & RPP:

Correlation of diastolic BP	Males	Females
Heart rate	0.35**	0.54**
RPP	0.54**	0.73**

RPP: Rate pressure product. P value ≤ 0.05 considered statistically significant.

Table-7 shows correlation of diastolic BP with Heart rate & RPP. A significant correlation of diastolic BP with heart rate was observed in both males and females with P = 0.003, P = 0.001 respectively. A significant correlation of diastolic BP with RPP was observed in both males and females with P= 0.001 and P = 0.001 respectively. A significant negative correlation of diastolic BP with HDL was observed in males with P= 0.03.

Figure-1 shows distribution of BP among boys. We found 10 boys had normal BP, 24 were prehypertensives and 16 were in stage 1 hypertension when classified according to JNC-7 classification.

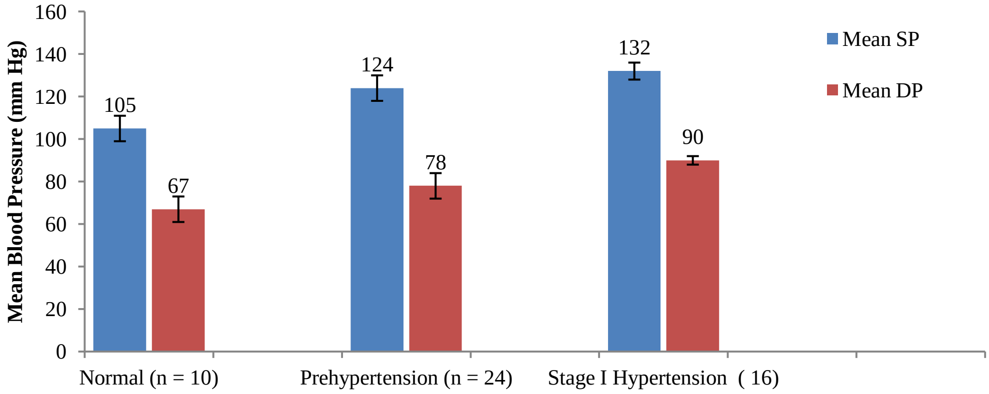


Figure-2 shows distribution of BP among girls. We found 22 girls had normal BP, 20 were prehypertensives and 8 were in stage 1 hypertension when classified according to JNC-7 classification.

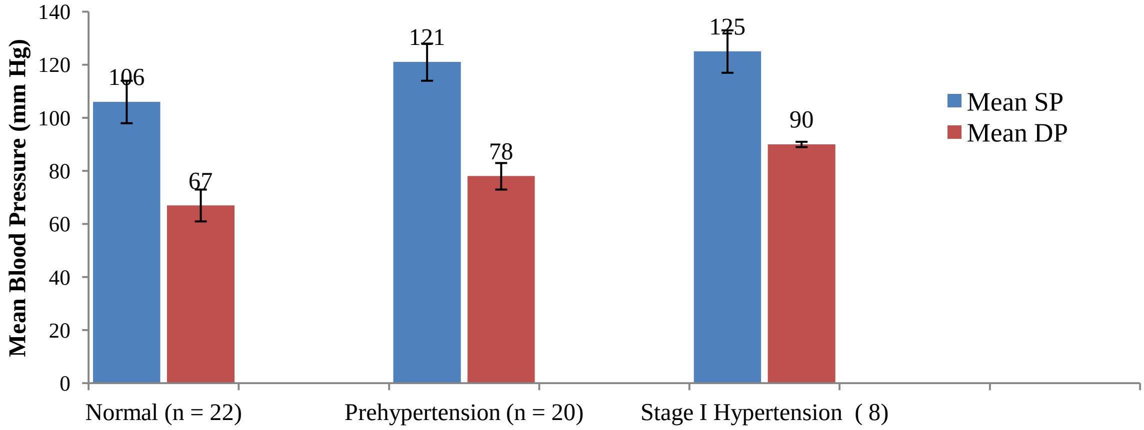


Figure-3 shows classification of BMI as per WHO guidelines among boys

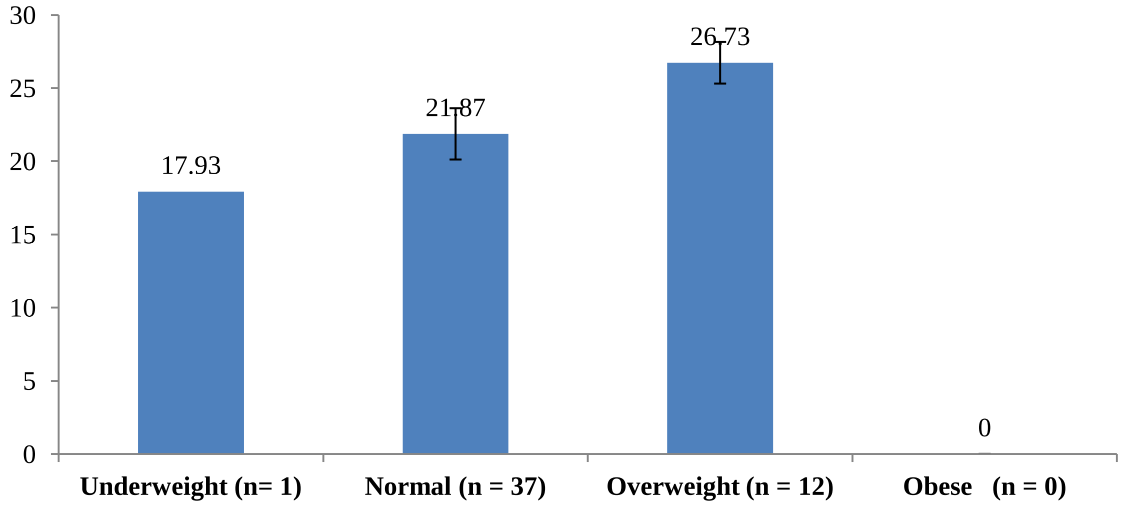
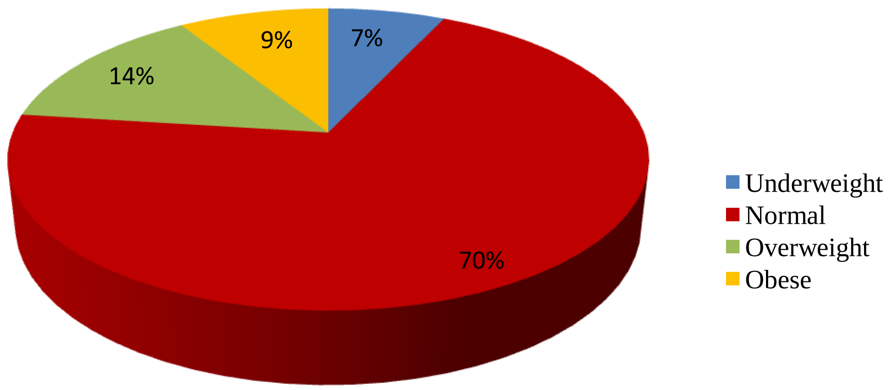


Figure-4 shows classification of BMI as per Who guidelines among girls expressed as percentage



DISCUSSION

Research has established the importance of gender in pathophysiology of various diseases and its treatment. Various forms of gender bias in most of the biomedical research carried out resulted in a situation where guidelines based on the study of one gender has be generalized and applied to both.[9] There is well known structural and functional differences in body composition of men and women.[10]

CVD is the major cause of morbidity and mortality for both men and women.[11] CVD in developing countries are characterized by early age of onset and greater mortality. [11] The childhood roots of adult obesity and also CVD are widely recognized and calls for health promotion targeted at youth. Assessing the risk for the presence of major CVD risk factors in young adults is of particular importance, since it would allow us to promptly identify persons at high risk for development of clinical CVD later in life.[8]

In the present study, anthropometric parameters like height, weight, WC and HC were found to be significantly higher in males as compared to females. Males also had higher levels of TG and VLDL. WC and HC are indices of central obesity[12,13] and a higher value in males shows the presence of central obesity in this group. These findings are in line with the findings of a similar study carried out on medical students by Bertsias et al[8], who reported central obesity levels to be significantly higher in men than women.[8] In the same study men were found to have high TG, LDL, TC/HDL and low HDL.[8] Central obesity is known to be independent predictor for CVD. This may be attributed to dietary factors, life style factors and urbanization.[14] In present study, a higher average value for WHR in males, suggest an excess of intra-abdominal adipose tissue. This could lead to changes in the lipid profile[8] although dyslipidemia was not observed in either group in the present study. Obesity in childhood is associated with obesity in adulthood and is a predictor of all the causes of obesity associated mortality from CVD in adulthood.[15] Juvenile overweight and obesity are increasing, influenced by factors such as genetic background, socioeconomic status, excessive energy intake and low energy expenditure**.**[16] In contrast to our findings, in a study by Akthar et al. the mean BMI for females is higher than males, shows obesity is more prevalent in females than in males and the authors attributed it to irregular dietary habits and hormonal changes.[17]

Owing to the presence of the hormone oestrogen, normally HDL cholesterol levels are found to be higher in females.[3] Serum HDL is higher in healthy females while serum TG is raised in healthy males. Increased HDL has a cardio protective role in female population.[17] In contrast to the above, our study group did not show any significant difference in HDL levels. From the present study a clear cut reason for the above mentioned finding cannot be explained. No significant difference was observed for FBG, TC, LDL, LDL/HDL and TC/HDL across gender in the present study. This is in line with the findings of Habib et al[3], who observed no significant difference of TC and LDL in normal male and females. The difference between the two studies is that the study by Habib et al[3] was on elderly subjects with the mean age being 47. Habib et al, have also reported the loss of gender difference in lipid profile in patients with DM. HDL < 35mg/dl in men and < 45mg/dl in women is associated with greater risk of CAD.[3] 1% reduction in serum TC reduces risk of CAD by 2 %.[17] Whether the absence of gender difference in young age especially in females in the present study may predispose them to DM in later life is a question which only further research can answer.

A well defined gender difference was observed in terms of SP, DP, HR and RPP in the two study groups in the present study. Twenty four hour mean BP has been reported to be significantly higher in men than women.[18] It has been also reported by Harshfield et al[19] that, after the onset of puberty, boys have higher BP than do age-matched girls.[19] This gender difference in regulation of BP has been attributed to hormone testosterone.[18] Heart rate has also been demonstrated to be lower in females, owing to the presence of female hormone estrogen. In line with these reports, in the present study SP, DP and HR were significantly lower in females as compared to males.

The main reasons for this gender difference may be due to difference in cardiovascular regulatory mechanisms among either gender, at all ages, women were found to have reduced sympathetic activity and enhanced parasympathetic activity when compared to men reflected by lower BP among women.[2]

RPP is an index of myocardial oxygen consumption.[20] In the present study RPP was significantly higher in males. An elevated RPP in males in the present study could have been to the fact that SP and HR was significantly higher in males. On the contrary, it can also result from elevated levels of sympathetic activity in males.[2] An increase in sympathetic activity can increase both HR and SP and their by increase the myocardial oxygen consumption. As the present study did not evaluate sympathetic activity, the presence of an elevated sympathetic activity can only be speculated.

In the present study, BMI positively correlated with SP and DP in the female group but not in males. A positive correlation between BMI and SP has been reported in overweight male subjects by Ravishankar et al.[21] The correlation between BMI and BP indices has been well established and has been found to be significant factor in obesity, hypertension.[16,22] Studies by Marina et al., have shown a relationship between increase in body weight and high BP; a 10 kilogram increase in body weight has been associated with a 3.0 mmHg higher systolic BP and 2.3 mmHg higher diastolic BP.[14] As body mass increases the demand for nutrient supply also increases, this demand eventually increases the sympathetic activity and thereby results in development of hypertension.[23] Also, activation of the renin–angiotensin system as well as physical compression of the kidney may be important factors in linking body weight and elevated BP.[24]

Population-based data on the anthropometric measurements of Palestinian adults showed BMI was significantly higher in females than in males. There was a significant positive association between BMI and age in both sexes. The mean WHR was significantly higher among males than females and the prevalence of central obesity among males was more than double that in females. Significant positive association was seen in both sexes between BMI levels and systolic BP, WHR, FBG and TG after adjusting for age, and a significant negative association with HDL-C was also seen in both sexes. TC and LDL were significantly associated with BMI only in men.[25] These findings are similar to the present study except that there was a significant negative correlation with HDL-C in males, whereas TC, LDL, FBG were significantly associated with BMI only in females. These gender differences in metabolic profile may determine the future risk of CVD among our study groups.

Hypertension is one of the major modifiable risk factors for stroke, CAD, renal failure and peripheral vascular disease.[26] In our study, Diastolic BP was lower in females. Similar findings were observed by Perloff et al.[27] Here, the mechanisms responsible for these gender differences in BP was attributed to androgens and female sex hormones.[27]

More accurately, a gender difference in BP is mainly due to the effect of sex hormones on renal sodium handling and vascular resistance. Female sex hormones protects against salt induced increase in BP mainly by increasing sensitivity of pressure-natriuresis relationship and augmenting renal sodium excretion.[26] The kidneys play a major role in the regulation of BP, a decrease in renal sodium excretion or a rightward shift of the pressure–natriuresis relationship can lead to a long-term increase in BP and the development of hypertension.[28] Also, ovarian hormones maintain a normal endothelial function[11,27], reduction plasma renin activity, angiotensin converting enzyme activity, vascular angiotensin 1 receptor expression, superoxide production[27], causes structural and functional alterations in the arterial wall that reduce vascular stiffness and reduce the development of isolated systolic hypertension.[27] Various studies has reported gender differences in various components of renin-angiotensin cascade that partially explains the gender differences in BP. Plasma renin activity has been reported to be 27% higher in men suggesting that testosterone stimulates the renin-angiotensin system[29].

Subgroup analysis of the data showed that, 44% girls had normal BP, 40% were prehypertensives and 16% were in stage 1 hypertension. Also, 20% of boys had normal BP, 48% were prehypertensives and 22% were in stage 1 hypertension. Presence of large number of our study subjects, who are otherwise normal, in prehypertensive category, suggests an increased risk of hypertension in them. Also, it has been observed that males are at higher risk compared to females among our study groups. A systematic review by Reckelhoff JF shows that, individuals who have prehypertensive BP have been reported to have an increased risk of developing CVD as compared to those with normal levels. The presence of one or more cardiovascular risk factors like high levels of TC, LDL, TG, glucose, insulin, BMI and a decreased HDL have been found to increase the progression of pre hypertension to hypertension.[18] This high risk in our study group may be attributed mainly to lack of physical activity, dietary habits and sedentary life style.

In our study, 74 % boys had normal BMI, 24% were overweight and 2% were underweight when classified according to WHO guidelines. Also we found 70% girls had normal BMI, 14% were overweight and 9% were obese. Only two third of the subjects among both the genders have normal BMI. Another one third being either overweight or obese are at a greater risk of developing hypertension, diabetes mellitus and hence a need for appropriate life style modification, dietary intervention at an earliest to prevent the development of metabolic disease.

The changes in demographic and epidemiological determinants of health, particularly changes in lifestyle, urbanization have resulted in an epidemiological and nutritional transition towards a greater prevalence of non-communicable diseases. The emerging chronic diseases like CVD, diabetes mellitus poses a serious threat to population. Hence early diagnosis and prevention of these diseases is important for public health.[30]

CONCLUSIONS

Our study group being otherwise normal students are of particular interest as very few studies exists on health status of medical students. Assessing the presence of major CVD risk factors in young adults is of particular importance, since it would allow us to promptly identify persons at high risk for development of clinical CVD later in life. We concluded that absence of normal gender difference will substantially increase cardio-metabolic risk. This may be attributed mainly to sedentary life style, stress and dietary habits. Hence gender specific evaluation, treatment and prevention strategies must be implemented to reduce the CVD burden and promote health.

Competing interests:

The authors declare that they have no competing interests.

Acknowledgements: nil

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