



Sex Related Assessment and Differentiation of Atherogenic Risk Factors

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Authors' contributions

All the authors equally contributed in the study design, analysis, literature search, read and approved the final manuscript.

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ABSTRACT

Aims: Atherosclerosis is latent precursor of clinical cardiovascular disease. The present study aimed to assess modifiable and non-modifiable atherogenic risk factors in both sexes.

Study Design: Cross sectional design.

Place and Duration of Study: It was conducted at Exservicemen Contributory Health Scheme (ECHS) Polyclinic, Sultanpur Lodhi, Kapurthala, Punjab, India from June, 2013 to Oct, 2013.

Methodology: This study was undertaken to assess age, education, employment, socioeconomic status, physical activity, body mass index, dietary habits, family history, sleep, stress, dysglycaemia, hypertension and dyslipidemia as determinants of atherogenic risk factors. The level of significance was defined by $P < .05$ with Chi Square test.

Results: All patients (N=351) were divided into male (49.58%) and female (50.42%) cohorts. A statistically significant males (45.41%; $P < .001$) were found >65 years old and females (43.51%; $P < .001$) in 51-65 years. Males had significant higher literacy (55.19%; $P < .001$) and employment status (55.75%; $P < .001$). Females were reported with significant positive family history (40.12%; $P < .01$), stress (25.99%), sleep inadequacy (28.82%; $P < .001$), sedentary lifestyle (83.62%; $P < .001$), and vegetarianism (74.02%;

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$P < .01$). Metabolic syndrome was more prevalent in females (19.78%) than males (14.95%). The higher prevalence of hypertension (females: 49.16%; males: 48.28%), obesity (females: 23.72%; males: 17.24%), dysglycaemia (females: 25.99%; males: 22.42%) was recorded in females; and dyslipidemia (males: 29.32%, females: 23.17%) in males.

Conclusion: Females were reported with significantly higher frequencies of atherogenic risk factors which make them highly susceptible to cardiovascular events than males. Hence, sex should be considered to assess and differentiate atherogenic risk factors, and when health professionals recommend lifestyle modifications.

Keywords: Atherosclerosis; cardiovascular disease; modifiable; non-modifiable; risk factors; sexes.

1. INTRODUCTION

Atherosclerosis is a chronic progressive disease with long subclinical asymptomatic phase [1] which is a latent precursor of clinical cardiovascular disease (CVD) including coronary heart disease (CHD), cerebrovascular disease, stroke and peripheral arterial disease affecting about 50% of men and 33% of women after age 40 [2]. Non communicable diseases have overtaken communicable diseases as the world's major disease burden, with CVD currently accounting for 17.3 million deaths per year which is expected to grow to >23.6 million by 2030 [3,4]. CVD is responsible for 10% of the disability-adjusted life years (DALYs) in low- and middle-income countries and for 18% of DALYs in high-income countries [5]. The cost of CVD is exacerbated in the developing world where it occurs in a higher proportion to working-age adults affecting both families and society due to its relation to both a loss of productivity and income of the person who has CVD and their caregiver, who may have to stop working to care for them [6].

CVD is an epidemic in India whose prevalence has increased in urban areas from about 2% in 1960 to 6.5% in 1970, 7.0% in 1980, 9.7% in 1990 and 10.5% in 2000; and it increased from 2% in 1970 to 2.5% in 1980, 4% in 1990, and 4.5% in 2000 in rural areas. It accounts for about 40% of the deaths in urban areas and 30% in rural areas. Epidemiological studies has shown the prevalence of obesity, hypertension, hypercholesterolemia, and diabetes have increased significantly in urban and gradually in the rural areas over the past 50 years with currently about 30 million CHD patients in the country [7]. The INTERHEART-South Asia study [8] identified eight established coronary risk factors--abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, low fruit and vegetable consumption, and lack of physical activity which accounted for 89% of acute myocardial infarctions in Indians at a much younger age than North Americans and Western Europeans.

Atherogenic risk factors such as age, dyslipidemia, hypertension, smoking, diabetes, obesity and physical inactivity are shared by both sexes and besides that, women have additional risk factors in the form of contraceptives use and reduced ovarian function with age [9]. However, sex itself has a significant influence on the cause, clinical manifestation and prognosis of CHD. Women have a more favorable atherogenic profile and age-specific CHD mortality rates than the same-aged men and this disparity is referred to as the female advantage [10]. This gender difference of lagging women 10 years behind those of men has been reported to decrease with age and after age 60, CHD accounts for 1 in 4 deaths in both sexes from 1 in 17 women and 1 in 5 men before age 60 [11].

Differential prevalence and impact of traditional cardiovascular risk factors have been shown to account for part but not all of the gender differences [12]. Non-modifiable CHD risk factors such as age and family history have different impact in both sexes. Modifiable risk factors further show similar trends with abdominal obesity and smoking more prevalent, and hypertension less controlled in men which increases their risk for CHD. Myocardial infarction is usually the first manifestation of CHD in men and the risk of its recurrence is twice as high in men as in women. This could explain gender differences in the CHD incidences but women significant protection against CHD during their reproductive years further contributes to the differences [13]. Estrogen has protective antioxidant and anti-inflammatory properties which provides beneficial effects on atherosclerotic plaque progression, vasodilatation and blood pressure [14]. An observed 2-fold increased CHD incidence in surgically postmenopausal versus premenopausal women of the same age has implicated a protective role of estrogen [15]. Early menopause, either spontaneous or artificial, is accompanied with an elevation in age-adjusted risk for CVD [16], but not all studies support it which may be partly explained by methodological differences [17]. However, the hormone replacement therapy use hasn't shown reduction in CHD events among postmenopausal women; and the role of endogenous estrogen in the cardioprotection of women compared with men is not completely understood [18]. Furthermore, trials in the 1960s and 1970s to prevent CHD in men by giving them high doses of estrogen had unfavorable results [19].

Environmental factors, geography and secular trends further play a role in gender differences, where women are more likely to have difficulty with social and physical activity [20,21]. Furthermore, differences in the clinical presentation of CHD have been observed in both sexes. The initial presentation of CHD in women is frequently atypical and complicated, whereas men relatively more often present with typical symptoms of CHD. The traditional diagnostic evaluation strategies have been validated in men and may be less suited for women [22]. Indeed, recent investigations have increased our awareness that gender-specific differences may exist in the pathophysiology of CHD [23]. Hence, further enhanced insights into the differences between male and female atherogenic risk factors are essential toward achieving optimal gender-specific disease prevention and therapy. This study is designed to assess the prevalence and comparison of the sociodemographic factors, modifiable and non-modifiable atherogenic risk factors in both sexes.

2. MATERIALS AND METHODS

2.1 Study Design

Sultanpur Lodhi, a county under district Kapurthala is located in the Indian state of Punjab. Males constitute 54% of the population and females 46% as per Indian census [24]. This cross-sectional descriptive study was undertaken to assess the sex-related assessment and differentiation of atherogenic risk factors among subjects registered with Ex-Servicemen Contributory Health Scheme (ECHS) Polyclinic. It is primary care centre which provides free at the point of access primary medical services and follow up care to the retired defense personnel. A multi-parameter and pre-tested questionnaire was designed to record the clinical history, physical examination and biochemical tests by personally interviewing the participants.

2.2 Ethnic Statement

Institutional ethical committee approval was obtained prior to the start of study and informed written consent was taken from all the participants who attended the polyclinic from June, 2013 to Oct, 2013.

2.3 Study Participants

The inclusion criteria was ex-servicemen who had been retired from their defense services; their family members limited to spouse, parents and children; holds a ECHS health card, and visited during the study period with any medical complaint.

2.4 Socioeconomic Variables

Age, education, socio-economic status and employment were elaborated. Education level was classified into four categories: no/little formal, primary, secondary and graduation. Socioeconomic status was defined into lower, middle and upper on the basis of retired ranks of ex-servicemen including their household income and assets. Homemaker, retiree and/or unemployed person had been counted under being at home.

2.5 Clinical Assessment

The clinical details of physical activity, family history, sleep adequacy, stress levels, dietary habits, alcohol consumption, metabolic syndrome and its components were elaborated. The American College of Sports Medicine and the American Heart Association recommendations for doing 30 minutes of moderate-intensity physical activity (e.g., brisk walking) on 5 or more days of the week or 20 minutes of vigorous-intensity physical activity (e.g., jogging or running) on 3 or more days of the week were considered [25]. The family history was defined as positive if a first-degree male relative (e.g. father, brother) and female relative have cardiovascular diseases (e.g., heart attack, hypertension) before the age of 55 or 65 years, respectively [3]. Sleep adequacy evaluated on the basis of sleep duration (7 to 8 hrs), difficulty in initiating and maintaining sleep. Job strain, social constraints, financial un-stability and emotional distress in the form of anxiety and/or depression were included under "stress" which significantly affects the daily life activities. Metabolic syndrome was defined on the basis of modified National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATPIII) guidelines [26], where-in presence of any three following traits in the same individual would meet the criteria:- abdominal obesity: waist circumference (WC) $\geq 102\text{cm}$ ($>40\text{inches}$) in men or $\geq 88\text{cm}$ ($>35\text{inches}$) in women; serum triglycerides (TGs): $\geq 150\text{mg/dl}$ ($\geq 1.7\text{mmol/L}$); High Density Lipoprotein Cholesterol (HDL-C): $< 40\text{mg/dl}$ ($< 1.03\text{mmol/L}$) in men or $< 50\text{mg/dl}$ ($< 1.29\text{mmol/L}$) in women; fasting blood glucose level: $\geq 100\text{mg/dl}$ ($\geq 5.6\text{mmol/L}$); blood pressure (BP): $\geq 130/85\text{mmHg}$. It further includes those previously diagnosed with hypertension, dyslipidemia, impaired fasting glucose, impaired glucose tolerance or type 2 diabetes mellitus and being on treatment for these disorders [26]. However, the current study used a correspondence of a WC of 102cm to body mass index (BMI) of $\geq 29.40\text{kg/m}^2$ defined in a linear regression analysis which is similar to BMI cut-offs used in previous modified NCEP definitions [27].

2.6 Clinical Measurements

Participants were weighed to the nearest 0.1kg wearing minimal clothes and without shoes; and height was measured to the nearest 0.1cm with a wall mounted non extendable measuring tape. BMI was calculated as weight per square meter (kg/m^2) and classified into underweight ($<18.50\text{kg}/\text{m}^2$), normal ($18.50\text{--}24.99\text{kg}/\text{m}^2$), overweight ($25.00\text{--}29.39\text{kg}/\text{m}^2$), and obesity ($\geq 29.40\text{kg}/\text{m}^2$) [26]. BP was measured in the right arm with the subject seated and rested for 5 minutes using a standard mercury sphygmomanometer and a suitable calibrated cuff.

2.8 Biochemical Measurements

A venous blood sample was obtained from all individuals after 8–10 hours of fasting. Blood tests were measured using bacteriological incubator, Erba glucose kit (GOD-POD method, end point), Erba triglyceride Des-kit (GPO-Trinder method, end point) and cholesterol kit (Phosphotungstic acid method, end point). Dysglycaemia was diagnosed on the basis of fasting blood glucose levels $>100\text{mg}/\text{dl}$ ($\geq 5.6\text{mmol}/\text{L}$). Dyslipidemia was evaluated on the basis of high TGs ($\geq 150\text{mg}/\text{dl}$ or $\geq 1.7\text{mmol}/\text{L}$) and/or low HDL levels ($<40\text{mg}/\text{dl}$ or $<1.03\text{mmol}/\text{L}$ in men or $<50\text{mg}/\text{dl}$ or $<1.29\text{mmol}/\text{L}$ in women) [26].

2.9 Statistical Analysis

The results were analyzed by Chi Square test. The value of statistical significance was calculated as $P<.05$.

3. RESULTS

All patients ($N=351$) were divided into male (49.58%) and female (50.42%) cohorts. Table 1 shows 12.65%, 32.19%, 48.86% and 06.33% of males had no/little, primary, secondary and graduation level education; and females had 48.03%, 27.12%, 21.47% and 03.39% for the same education categories, respectively. This shows a statistically significant higher literacy among males (55.19%; $P<.001$) than females (24.86%; $P<.001$). Majority of males (58.63%) and females (68.37%) belonged to the middle socioeconomic status in the present study. A statistically significantly employment and unemployment were seen in males (55.75%; $P<.001$) and females (96.62%; $P<.001$) respectively.

Table 1. Comparisons of the socio-demographic variables among males and females*

Category	Males (174)	Females (177)	P value
Education			
No/Little	12.65 (22)	48.03 (85)	80.70
Primary	32.19 (56)	27.12 (48)	(<.001)
Secondary	48.86 (85)	21.47 (38)	
Graduation	06.33 (11)	03.39 (06)	
Socioeconomic status			
Middle	58.63 (102)	68.37 (121)	3.58
Upper	41.38 (72)	31.64 (56)	
Occupation			
Stays at home	44.26 (77)	96.62 (171)	116.05
Employed	55.75 (97)	03.90 (06)	(<.001)

parentheses represent absolute number of the subjects in a sample

Table 2 reflects the comparison of non-modifiable atherogenic risk factors in both sexes. 02.88%, 16.09%, 35.64% and 45.41% of males were in 20-35years, 36-50 years, 51-65 years and >65 years of age group; and females had 04.52%, 29.38%, 43.51% and 22.60% for the same age groups, respectively. A statistically significant higher age group (>65years) was reported in males (45.41%; $P<.001$), and females (43.51%; $P<.001$) among 51-65 years of age groups. Males (73.56%; $P<.01$) and females (40.12%; $P<.01$) were observed with a statistically significant negative and positive family history, respectively.

Table 2. Comparison of the non-modifiable atherogenic risk factors in both sexes*

Category	Males (174)	Females (177)	P value
Age (years)			
20-35	02.88 (05)	04.52 (08)	22.30
36-50	16.09 (28)	29.38 (52)	(<.001)
51-65	35.64 (62)	43.51 (77)	
>65	45.41 (79)	22.60 (40)	
Family history			
Positive	26.44 (46)	40.12 (71)	7.42
Negative	73.56 (128)	59.89 (106)	(<.01)

parentheses represent absolute number of the subjects in a sample

Fig. 1 and Table 3 represent the comparisons of modifiable atherogenic risk factors in both sexes. 05.18%, 45.41%, 32.19% and 17.24% of males were in <18.50kg/m², 18.50-24.99kg/m², 25.00-29.39kg/m² and ≥29.40kg/m² of BMI; and females had 05.65%, 40.68%, 29.95% and 23.72% for the same BMI groups, respectively. Males (32.19%) and females (23.72%) were reported with a higher prevalence of overweight and obesity, respectively. A statistically significant active and sedentary lifestyle was noticed in males (55.75%; $P<.001$) and females (83.62%; $P<.001$), respectively.

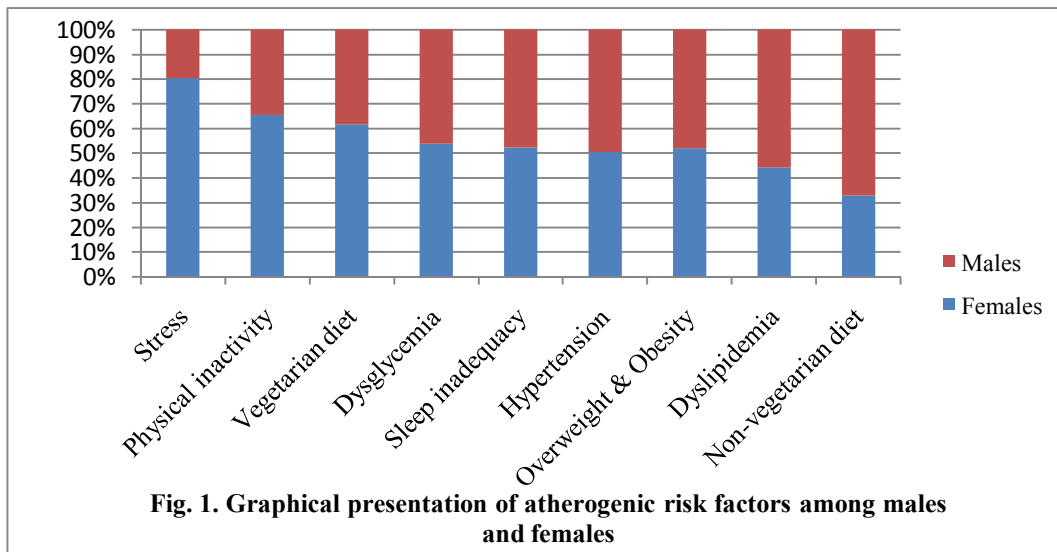


Fig. 1. Graphical presentation of atherogenic risk factors among males and females

Table 3 further displays males (53.45%; $P<.001$) and females (74.02%; $P<.001$) had a statistically significant dietary preference for omnivores and vegetarian food, respectively.

Sleep inadequacy was more frequently self-reported by females (28.82%) than males (26.44%). Similarly, a statistically significant stress was self-reported by females (25.99%; $P < .001$) than males (06.33%; $P < .001$). Furthermore, females (19.78%) were more frequently diagnosed with metabolic syndrome than males (14.95%); however, no statistically significant relation was revealed.

Table 3. Comparison of the modifiable atherogenic risk factors in both sexes*

Category	Males (174)	Females (177)	p value
Body mass index			
Underweight	05.18 (09)	05.65 (10)	2.40
Normal weight	45.41 (79)	40.68 (72)	
Overweight	32.19 (56)	29.95 (53)	
Obesity	17.24 (30)	23.72 (42)	
Physical activity			
Adequate	55.75 (97)	16.39 (29)	59.07
Inadequate	44.26 (77)	83.62 (148)	(<.001)
Dietary habits			
Vegetarian	46.56 (81)	74.02 (131)	27.66
Omnivores	53.45 (93)	25.99 (46)	(<.001)
Sleep			
Adequate	73.57 (128)	71.19 (126)	0.27
Inadequate	26.44 (46)	28.82 (51)	
Stress			
Yes	06.33 (11)	25.99 (46)	24.95
No	93.68 (163)	74.02 (131)	(<.001)
Metabolic syndrome			
Yes	14.95(26)	19.78(35)	1.41
No	85.06(148)	80.23(142)	

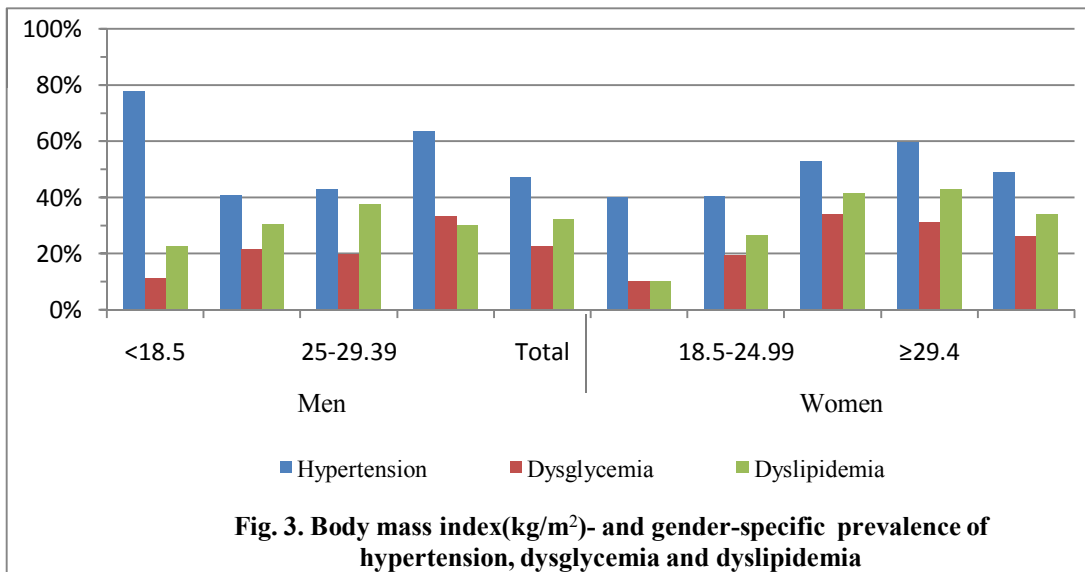
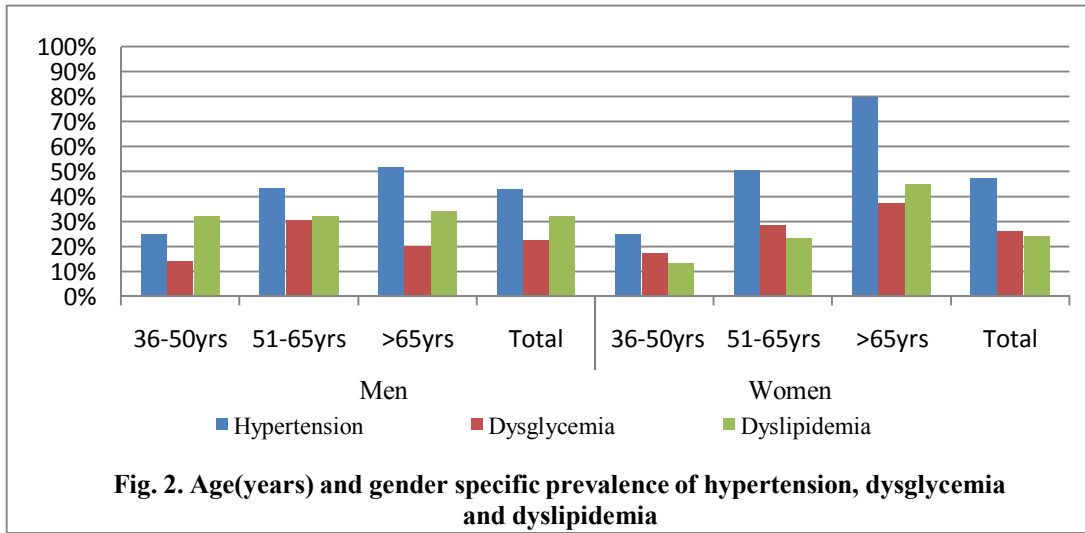
parentheses represent absolute number of the subjects in a sample

Table 4 elaborates the comparison of the components of metabolic syndrome in both sexes. The prevalence of hypertension was approximately the same in both males (48.28%) and females (49.16%). Dyslipidemia was more frequently diagnosed in males (29.32%) than females (23.17%). Conversely, females had a higher predisposition to dysglycemia (25.99%) and obesity (23.72%) than males (dysglycemia: 22.42%; obesity: 17.24%) in the present study. The age and BMI specific prevalence of hypertension, dysglycemia and dyslipidemia is shown in Fig. 2 and Fig. 3 respectively.

Table 4. Comparison of the components of metabolic syndrome in both sexes*

Category	Males (174)		Females (177)		p value
	Healthy	Diseased	Healthy	Diseased	
Hypertension	51.73(90)	48.28(84)	50.85 (90)	49.16(87)	.04
Dyslipidemia	70.69(123)	29.32(51)	76.84(136)	23.17(41)	1.69
Dysglycemia	77.59(135)	22.42(39)	74.02(131)	25.99(46)	.59
Obesity	82.76 (144)	17.24(30)	76.28 (135)	23.72 (42)	2.26

parentheses represent absolute number of the subjects in a sample



4. DISCUSSION

Atherosclerosis begins with fatty streaks, the precursor of atherosclerotic plaques which appear in the aortic intima at the three years of age, and in the coronary arteries during adolescence [28]. Thus, atherosclerosis gradually changed from a model of a chronic degenerative disease exclusively affecting patients with advanced age to a model of subclinical chronic inflammatory disease already existing in childhood [29]. The likelihood of occurrence of CVD further increases in the presence of multiple atherogenic risk factors which may be modifiable or non-modifiable [30]. The current study was designed to determine the prevalence and comparison of modifiable and non modifiable risk factors in both sexes.

The current study (Table 1) observed a significant higher education levels in males (55.19%; $P < .001$) than females (24.86%; $P < .001$). Bobak et al. [31] has shown an inverse correlation of atherogenic risk factors and education attainment, and found men and women with lower education have a higher incidence of elevated BP, BMI, total cholesterol, and smoking. Furthermore, Saeed et al. [32] has noticed subjects with a higher education level have a good knowledge and awareness level of modifiable CVD risk factors. Majority of males in the present study were employed significantly (55.75%; $P < .001$), while most of significant females were looking after their families by staying at home (96.62%; $P < .001$). Dupre et al. [33] has found unemployment status, multiple job losses, and short periods without work are all significant risk factors for acute cardiovascular events. However, Kivimaki et al. [34] has shown psychological stress at work or job strain to be moderately associated with an increased risk of CHD. Furthermore, Cho and Lee [35] noticed associations between education, occupation, and financial status and cardiovascular risk factors. Such differences may be due to underlying gender differences in biological vulnerability; social coping mechanisms; and access to material, social, and physiological resources [35]. It shows a higher illiteracy (24.86%; $P < .001$) and being at home (96.62%; $P < .001$) makes females prone to a statistically significant higher atherogenic risk in the current study.

A majority of significant males (45.41%) in the current study (Table 2) belonged to geriatric group (>65years; $P < .001$), and significant females to 51-65 years of age group (43.51%; $P < .001$). Jousilahti et al. [12] has observed an age-related increase in CHD incidence and mortality in both sexes but to a larger extent in women. This can be contributed to the menopausal state which accompanies metabolic, biochemical, and physiological alterations. Total cholesterol, LDL-C, and TGs rises, and HDL-C apparently decreases [36]. Furthermore, glucose metabolism worsens in the sense of higher blood glucose and decreased insulin sensitivity [37]. An additional change is procoagulatory state with increased fibrinogen and plasminogen activator inhibitor-1 levels [37]. On the whole, reduced glucose tolerance, abnormal plasma lipids, increased BP, increased sympathetic tone, endothelial dysfunction and vascular inflammation [38] seems to develop during the menopause which can be partly reversed by estrogen administration [39]. A higher statistically significant positive family history in females (40.12%; $P < .01$) than males (26.44%; $P < .01$) in the present study was found supported by Pohjola-Sintonen et al. [40]. Furthermore, Jousilahti et al. [41] showed that 76% of the women and 62% of the men who survived a myocardial infarction had first degree relatives with CHD at <65 years of age. This shows a postmenopausal status (66.11%; $P < .001$) and positive family history (40.12%; $P < .01$) predisposes females to a statistically significant higher atherogenic risk levels in the present study.

A higher prevalence of obesity (Table 3) in females (23.72%) than males (17.24%) in the current study has been consistent with Sani et al. [42]. Furthermore, Wannamethee et al. [43] reported significantly increased long term risk of CVD and diabetes with increasing overweight and obesity. McGill et al. [44] showed adiposity has a greater impact on atherogenesis in men than women. This difference in atherogenesis rates may be due to differences in body fat distribution where men are more prone to abdominal obesity, one of the cardiovascular risk factors and women to gluteofemoral adiposity [45]. However, females with a menopausal status experiences changes in body fat distribution from a gynaeoid to an android pattern which increases their atherogenic risk levels [38]. A statistically significant sedentary lifestyle lived by females (83.62%; $P < .001$) than males (44.26%; $P < .001$) in the current study has been persistent with Perez et al. [46]. Heavy environmental pollution, scarcity of "green spaces" or open land for public use, high traffic, no sidewalks, and even the threat of violence outside the home contributes to the lack of exercise and sedentary

lifestyle [5]. Sedentary lifestyle (83.62%; $P < .001$) and obesity (23.72%) makes females susceptible to a higher atherogenic risk levels in current study.

A statistically significant vegetarianism (74.02%; $P < .001$) and omnivorism (53.45%; $P < .001$) was reported in females and males respectively, in the present study. Key et al. [47] has observed vegetarians had a 24% lower mortality from ischemic heart disease than omnivores due to lower serum total cholesterol, reduced LDL-C oxidation or changes in blood clotting. Tonstad et al. [48] has further added lower rate of obesity and type 2 diabetes mellitus among vegetarians. Pettersen et al. [49] has noticed lower systolic and diastolic BP among vegetarians. Thus, vegetarianism (74.02%) plays a protective role for females participating in the current study. Furthermore, a higher sleep inadequacy was self-reported by females (28.82%) than males (26.44%) in the present study. Epidemiological and pathophysiological studies [50,51] indicated a causal link between primary sleep abnormalities (sleep curtailment, shift work, and sleep-disordered breathing) and cardiovascular and metabolic diseases including hypertension, atherosclerosis, stroke, heart failure, cardiac arrhythmias, obesity, menopause, and the metabolic syndrome. This shows medical conditions like obesity (23.72%) and menopause (66.11%) might have contributed to a higher sleep inadequacy among female subjects in the present study.

A statistically significant higher stress levels were complained by females (25.99%; $P < .001$) than males (6.33%; $P < .001$) in the current study. The Framingham Study [52] was the first to describe the relationship between type A behaviour and cardiovascular disease, and found hostility and anger are significantly associated with CVD in both sexes. Nyberg et al. [53] has further found an association between job related strain and elevated Framingham risk score attributing to the higher prevalence of diabetes, smoking and physical inactivity among those reporting job strain. Marital stress, at the same time, appears to be one of the primary factors in worsening prognosis among women with CHD [54]. Furthermore, Gallo et al. [55] has shown high-quality marriages may protect against CVD by less rapid progression of carotid atherosclerosis relative to women in low-satisfying marriages. Thus, the sleep inadequacy (28.82%) and higher stress levels (25.99%; $P < .001$) among female population make them vulnerable to an elevated atherogenic risk levels.

A higher trend of metabolic syndrome in females (19.78%) than males (14.95%) in the present study was found supported by Magnat et al. [56]. Conversely, Sawant et al. [57] has found significant double the prevalence of MetS in males than females. The prevalence of hypertension (Table 4) was approximately the same among males (48.28%) and females (49.16%) in the present study. However, Castanho et al. [58] has reported higher prevalence of hypertension among women than men. Conversely, Sani et al. [42] found a contrary finding of higher blood pressure in men. However, Hsia has shown a strong association between hypertension and CHD in women [59]. A higher prevalence of hyperlipidemia in males (29.32%) than females (23.17%) in the current study was contrary to Cho and Lee [35]. Conversely, a higher prevalence of dysglycemia in females (25.99%) than males (22.42%) in the present study might be contributed to sedentary lifestyle [46], obesity [43], high stress levels [52,53,54,55] and inadequate sleep [51] among them. Diabetic women lose their "relative immunity" to CHD in relation to men, where female diabetics are two times more likely to die from CHD than male diabetics [60] which further makes females of the current population highly susceptible to an atherogenic risk. However, Sani et al. [49] has reported an equal prevalence of type 2 diabetes mellitus in both sexes.

4.1 Study limitations

The present study did not screen for other established CVD risk factors like smoking, microalbuminuria, homocysteinaemia and C reactive protein. Smoking has a very low prevalence in the study population due to practising of “Sikhism” by the study subjects in which it is considered forbidden. A cross-sectional design did not allow for the determination of concrete causal relationships in a specific time interval. Several variables like family history, physical activity, stress level and sleep adequacy were self reported by the study subjects which may had lead to an over-estimation or under-estimation of these risk factors. However, the current study findings can serve as a template for a proper community based study on the same subject especially in the view of high prevalence of atherogenic risk factors.

5. CONCLUSION

Females were reported with significantly higher conglomerate frequencies of atherogenic risk factors such as obesity, sedentary lifestyle, stress, inadequate sleep, postmenopausal status, dysglycaemia, hypertension and dyslipidemia which make them highly susceptible to cardiovascular events. Several characteristics easily obtained through self-report may identifies individuals at a higher atherogenic risk who requires aggressive risk factor intervention, including cholesterol and blood pressure reduction, management of blood glucose, and weight loss. Hence, sex should be considered to assess and differentiate atherogenic risk factors, and when health professionals promotes and recommend behavior modifications by physical activity, reducing obesity and maintaining normal BMI, dietary modifications, and stress management.

CONSENT

All authors declare that an informed written consent was taken from all the selected subjects.

ETHICAL APPROVAL

All authors hereby declare that the present study have been approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Toth PP. Subclinical atherosclerosis: what it is, what it means and what we can do about it. *Int J Clin Prac.* 2008;62(8):1246-1254.
2. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. *Lancet.* 1999;353:89-92.
3. World Health Organization. Cardiovascular Disease: Global Atlas on Cardiovascular Disease Prevention and Control. Available at: http://www.who.int/cardiovascular_diseases/publications/atlas_cvd/en/; accessed 29 March 2014.

4. Smith SC, Collins A, Ferrari R. Our time: a call to save preventable death from cardiovascular disease (heart disease and stroke). *J Am Coll Cardiol*. 2012;60(22):2343-2348.
5. World Heart Federation. Urbanization and Cardiovascular Disease: Raising Heart-Healthy Children in Today's Cities. Available at: <http://www.world-heart-federation.org/publications/reports/urbanization-and-cvd/>; accessed 29 March 2014.
6. Gaziano TA. Reducing the growing burden of cardiovascular disease in the developing world. *Health Aff (Project Hope)*. 2007;26(1):13-24.
7. Gupta R. Recent trends in coronary heart disease epidemiology in India. *Indian Heart J*. 2008;60(2,SupplB):B4-B18.
8. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364(9438):937-952.
9. Bush TL, Fried LP, Connor-Barret E. Cholesterol, lipoproteins and coronary heart disease in women. *Clin Chem*. 1988;34(8B):B60-B70.
10. Barrett-Connor EL, Cohn BA, Wingard DL, Edelstein SL. Why is diabetes mellitus a stronger risk factor for fatal ischemic heart disease in women than in men? The Rancho Bernardo Study. *JAMA*. 1991;265(5):627-631.
11. National Center for Health Statistics. Vital statistics of the United States, 1989. Vol. II. Mortality. Part A. Washington, DC: Government Printing Office, 1993. DHHS publication (PHS) 93-1101.
12. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Sex, age, cardiovascular risk factors, and coronary heart disease: A prospective follow-up study of 14 786 middle-aged men and women in Finland. *Circulation*. 1999;99:1165-1172.
13. Fodor JG, Tzerovska R. Coronary heart disease: is gender important? *The journal of men's health & gender*. 2004;1(1):32-37.
14. Lawton JS. Sex and Gender Differences in Coronary Artery Disease. *Semin Thoracic Surg*. 2011;23(2):126-130.
15. Colditz GA, Willett WC, Stampfer MJ, Rosner B, Speizer FE, Hennekens CH, et al. Menopause and the risk of coronary heart disease in women. *N Engl J Med*. 1987;316:1105-1110.
16. Atsma F, Bartelink ML, Grobbee DE, Van der Schouw YT. Postmenopausal status and early menopause as independent risk factors for cardiovascular disease: A meta-analysis. *Menopause*. 2006;13(2):265-279.
17. Tom SE, Cooper R, Wallace RB, Guralnik JM. Type and timing of menopause and later life mortality among women in the Iowa established populations for the Epidemiological study of the elderly (EPESE) cohort. *J Womens Health (Larchmt)*. 2012;21(1):10-16.
18. Hulley S, Grady D, Bush T, Furberg C, Herrington D, Riggs B, Vittinghoff E, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/Progestin Replacement Study (HERS) Research Group. *JAMA*. 1998;280(7):605-613.
19. Coronary Drug Project Research Group. The coronary drug project. Findings leading to discontinuation of the 2.5-mg day estrogen group. *JAMA*. 1973;226:652-657.
20. Wild SH, Laws A, Fortmann SP, Varady AN, Byrne CD. Mortality from coronary heart disease and stroke for six ethnic groups in California, 1985 to 1990. *Ann Epidemiol*. 1995;5:432-439.

21. Nassis GP, Geladas ND. Age-related pattern in body composition changes for 18- to 69-year old women. *Journal of Sports Medicine and Physical Fitness*. 2003;43(3):327-333.
22. Mosca L, Appel LJ, Benjamin EJ, Berra K, Strobos NC, Fabunmi RP, et al. Evidence-based guidelines for cardiovascular disease prevention in women. *Circulation*. 2004;109:672-693.
23. Shaw LJ, Bairey Merz CN, Pepine CJ, Reis SE, Bittner V, Kelsey SF, et al. Insights from the NHLBI-Sponsored Women's Ischemia Syndrome Evaluation (WISE) Study: Part I: gender differences in traditional and novel risk factors, symptom evaluation, and gender-optimized diagnostic strategies. *J Am Coll Cardiol*. 2006;47(3 Suppl):S4-S20.
24. "Census of India 2001: Data from the 2001 Census, including cities, villages and towns (Provisional)". Census Commission of India. Accessed 5 March 2014.
25. Haskell WL, Lee IM, Pate RR, et al. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc*. 2007;39(8):1423-1434.
26. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation*. 2005;112:2735-2752.
27. Sattar N, Gaw A, Scherbakova O, Ford I, O'Reilly DS, Haffner SM, et al. Metabolic syndrome with and without c-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland coronary prevention study. *Circulation*. 2003;108:414-419.
28. Ford ES. C-reactive protein concentration and cardiovascular disease risk factors in children: findings from the National Health and Nutrition Examination Survey 1999–2000. *Circulation*. 2003;108:1053-1058.
29. Verri J, Fuster V. Mecanismos das síndromes isquêmicas agudas e da progressão da aterosclerose coronária. *Arq Bras Cardiol*. 1997;68(6):461-467. French
30. Tolfrey K. Intraindividual variability of children's blood, lipid and lipoprotein concentrations: a review. *Prev Cardiol*. 2002;3:1445-1451.
31. Bobak M, Hertzman C, Skodova Z, Marmot M. Socioeconomic status and cardiovascular risk factors in the Czech Republic. *Int J Epidemiol*. 1999;28(1):46–52.
32. Saeed O, Gupta V, Dhawan N, Streja L, Shin JS, Ku M, et al. Knowledge of modifiable risk factors of Coronary Atherosclerotic Heart Disease (CASHD) among a sample in India. *BMC International Health and Human Rights*. 2009;9:2-8.
33. Dupre ME, George LK, Liu G, Peterson ED. The Cumulative Effect of Unemployment on Risks for Acute Myocardial Infarction. *Arch Intern Med*. 2012;172(22):1731-1737.
34. Kivimäki M, Nyberg ST, Batty GD, Fransson EI, Heikkilä K, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. *Lancet*. 2012;380(9852):1491–1497.
35. Cho CM, Lee YM. The relationship between cardiovascular disease risk factors and gender. *Health*. 2012;4(6):309-315.
36. Tremollieres FA, Pouilles JM, Cauneille C, Ribot C. Coronary heart disease risk factors and menopause: a study in 1684 French women. *Atherosclerosis*. 1999;142(2):415–423.
37. Gohlke-Baerwolf C. Coronary artery disease—is menopause a risk factor?. *Basic Res Cardiol*. 2000;95(Suppl 1):177–183.

38. Rosano GM, Vitale C, Marazzi G, Volterrani M. Menopause and cardiovascular disease: the evidence. *Climacteric*. 2007;10(Suppl 1):19-24.
39. Taddei S, Virdis A, Ghiadoni L, et al. Menopause is associated with endothelial dysfunction in women. *Hypertension*. 1996;28(4):576–582.
40. Pohjola-Sintonen S, Rissanen A, Liskola P, Luomanmaki K. Family history as a risk factor of coronary heart disease in patients under 60 years of age. *Eur Heart J*. 1998;19(2):235–239.
41. Jousilahti P, Rastenyte D, Tuomilehto J, Sarti C, Vartiainen E. Parental history of cardiovascular disease and risk of stroke. A prospective follow-up of 14 371 middle-aged men and women in Finland. *Stroke*. 1997;28:1361–1366.
42. Sani MU, Wahab WK, Yusuf BO, Gbadamosi M, Johnson OV, Gbadamosi A, et al. Modifiable cardiovascular risk factors among apparently healthy adult Nigerian population - a cross sectional study. *BMC Research Notes*. 2010;3:11.
43. Wannamethee SG, Shaper AG, Walker M. Overweight and obesity and weight change in middle aged men: impact on cardiovascular disease and diabetes. *J Epidemiol Community Health*. 2005;59(2):134–139.
44. McGill Jr HC, McMahan CA, Herderick EE, et al. Obesity and atherosclerosis in youth. *Circulation*. 2002;105:2712–2718.
45. Larsson B, Svardsudd K, Welin L, et al. Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: 13-year follow-up of participants in the study of men born in 1913. *Br Med J*. 1984;288(6428):1401–1404.
46. Perez LG, Pratt M, Simoes EJ, DLSHTM, Lenildo de M, Malta DC. Association between leisure time physical activity and self reported hypertension among Brazilian adults, 2008. *Prev Chronic Dis*. 2013;10:e130032.
47. Key TJ, Fraser GE, Thorogood M, Appleby PN, Beral V, Reeves G, Burr ML, et al. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *Am J Clin Nutr*. 1999;70(Suppl):516S–S524.
48. Tonstad S, Butler T, Yan R, Fraser GE. Type of Vegetarian diet, body weight, and prevalence of type 2 diabetes. *Diabetes Care*. 2009;32(5):791–796.
49. Pettersen BJ, Anousheh R, Fan J, Jaceldo-Siegl K, Fraser GE. Vegetarian diets and blood pressure among white subjects: results from the Adventist Health Study-2 (AHS-2). *Public Health Nutr*. 2012;15(10):1909–1916.
50. Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem. Institute of Medicine (US) Committee on Sleep Medicine and Research; Colten HR, Altevogt BM, editors. Washington (DC): National Academies Press (US); 2006.
51. Wolk R, Gami AS, Garcia-Touchard A, Somers VK. Sleep and cardiovascular disease. *Curr Probl Cardiol*. 2005;30(12):625-62.
52. Haynes SG, Feinleib M, Kannel WB. The relationship of psychosocial factors to coronary heart disease in the Framingham Study. III. Eight-year incidence of coronary heart disease. *Am J Epidemiol*. 1980;111(1):37–58.
53. Nyberg ST, Fransson EI, Heikkila K, Alfredsson L, Casini A, et al. Job Strain and Cardiovascular Disease Risk Factors: Meta-Analysis of Individual- Participant Data from 47,000 Men and Women. *PLoS ONE*. 2013;8(6):e67323.
54. Orth-Gomer K, Wamala SP, Horsten M, Schenck- Gustafsson K, Schneiderman N, Mittleman MA, et al. Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *JAMA*. 2000;284(23):3008–3014.

55. Gallo LC, Troxel WM, Kuller LH, Sutton-Tyrrell K, Edmundowicz D, Matthews KA, et al. Marital Status, Marital Quality, and Atherosclerotic Burden in Postmenopausal Women. *Psychosomatic Medicine*. 2003;65:952–962.
56. Mangat C, Goel NK, Dinesh KW, Neeraj A, Munesh KS, Jasbinder K, et al. Metabolic Syndrome: a challenging health Issue in highly urbanized Union Territory of north India. *Diabetology & Metabolic Syndrome*. 2010;2:19.
57. Apurva S, Ranjit M, Swarup S, Rani R, Gargi G, Himanshu R, et al. Prevalence of Metabolic Syndrome in Urban India. *Cholesterol*. 2011, article ID 920983, 7 pages.
58. Castanho VS, Oliveira LS, Pinheiro HP, Oliveira HCF, de Faria EC. Sex differences in risk factors for coronary heart disease: a study in a Brazilian population. *BMC Public Health*. 2001;1:3-10.
59. Hsia AJ. Cardiovascular disease in women. *Med Clin North Am*. 1998;82(1):1-19.
60. Kannel WB. Metabolic risk factors for coronary heart disease in women: perspective from the Framingham study. *Am Heart J*. 1987;114(2):413-419.

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