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Atherosclerotic Effects of Smoking and Excess Weight

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Abstract

Background: Metabolic syndrome is a systemic atherosclerotic cascade terminating with multi-organ failures.

Methods: Consecutive patients with Coronary Heart Disease (CHD) were studied.

Results: Study included 1,620 females and 1,240 males. Prevalences of CHD were similar in both sexes (3.8% versus 4.4%, respectively, $p>0.05$). Mean ages of CHD cases were 61.5 versus 63.5 years in both sexes, respectively ($p>0.05$). Smoking and Chronic Obstructive Pulmonary Disease (COPD) were higher in males with CHD (54.5% versus 9.6%, $p<0.001$ and 18.1% versus 6.4%, $p<0.05$, respectively). On the other hand, body mass index (BMI) and white coat hypertension (WCH) were higher in females with CHD (29.7 versus 28.3 kg/m^2 and 30.6% versus 23.6%) but differences were nonsignificant ($p>0.05$ for both) probably due to small sample sizes of the groups. Whereas low density lipoprotein cholesterol (LDL-C) and triglyceride (TG) were higher in females with CHD, significantly (132.6 versus 115.6 mg/dL, $p=0.008$ and 250.3 versus 150.1 mg/dL, $p=0.002$, respectively). Similarly, hypertension (HT) and diabetes mellitus (DM) were also higher in females with CHD, significantly (58.0% versus 30.9%, $p<0.001$ and 51.6% versus 38.1%, $p<0.05$, respectively).

Conclusion: Metabolic syndrome is a systemic atherosclerotic process exaggerated by some metabolic disorders. Smoking and excess weight may be the major triggering causes of the syndrome, and they come with similar degree of clinical severity in front. Smoking and COPD were higher in males with CHD against the higher BMI, WCH, LDL-C, TG, HT, and DM in females, resulting with similar prevalences of CHD in both sexes.

Keywords: Smoking; Excess weight; Atherosclerosis; Metabolic syndrome

Introduction

Due to the prolonged survival, systemic atherosclerosis may be the main health problem of the human being in this century, and an association between systemic atherosclerosis and some metabolic disorders and smoking is known for many years, and called as the metabolic syndrome [1,2]. The syndrome is characterized by a low-grade chronic inflammatory process probably initiating in early life [3], and exaggerated by some metabolic disorders, smoking, and aging. Although the syndrome can not be prevented completely due to the possible effects of aging alone, it can be slowed down with appropriate nonpharmaceutical approaches including lifestyle changes, diet, and exercise [4]. The metabolic syndrome may contain White Coat Hypertension (WCH), Impaired Fasting Glucose (IFG), Impaired Glucose Tolerance (IGT), hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, overweight, and smoking like reversible risk factors for the development of terminal diseases including Hypertension (HT), Diabetes Mellitus (DM), obesity, Chronic Obstructive Pulmonary Disease (COPD), hepatic cirrhosis, Chronic Renal Failure (CRF), Peripheric Artery Disease (PAD), Coronary Heart Disease (CHD), and stroke [5]. In another view, the syndrome is probably the most significant disease of human life decreasing its quality and duration, now. The syndrome has become increasingly common all over the world, for example, 50 millions of people in the United Sates may have it [6]. The syndrome induced symptomatic atherosclerosis is the leading cause of death for both sexes. For example, CHD is the leading cause of death in developed countries. During the average life span, males and females probably have the same risk of mortality from CHD [5]. Although CHD may be equally seen in both sexes, there may be some gender differences in the risk factors of CHD. We tried to understand whether or not there are some gender differences according to the atherosclerotic risk factors in cases with CHD in the present study.

Material and Methods

The study was performed in the Internal Medicine Polyclinic of the Dumlupinar University between August 2005 and March 2007. We took consecutive patients applying for any reason at and above the age of 15 years. Their medical histories including smoking habit were learnt, and a routine check up procedure including Fasting Plasma Glucose (FPG), Low Density Lipoprotein Cholesterol (LDL-C), Triglyceride (TG), and an electrocardiography was performed. Current smokers with six pack-months and cases with a history of five pack-years were accepted as smokers. COPD was diagnosed via the pulmonary function tests in suspected cases in which the ratio of forced expiratory volume in the first second of expiration to forced vital capacity is lower than 70%. Body Mass İndex (BMI) of each case was calculated by the measurements of the same Physician instead of verbal expressions. Weight in kilograms is divided by height in meters squared [7]. Cases with an overnight FPG level of 126 mg/dL or greater on two occasions or already using antidiabetic medications were defined as diabetics. An oral glucose tolerance test with 75 gram glucose was performed in cases

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with a FPG level between 110 and 126 mg/dL, and diagnosis of cases with a two-hour plasma glucose level of 200 mg/dL or higher is DM. An Office Blood Pressure (OBP) was checked after a 5 minute rest in seated position with a mercury sphygmomanometer on three visits, and no smoking was permitted during the previous two hours. A 10 day twice daily measurement of Blood Pressure At Home (HBP) was obtained in all cases, even in normotensives in the office due to the risk of masked HT after an education about proper BP measurement techniques [8]. A 24 hour Ambulatory Blood Pressure (ABP) monitoring was not required due to its equal effectiveness with HBP measurements [9]. Eventually, HT is defined as a mean HBP value of 135/85 mmHg or greater, and WCH as an OBP of 140/90 mmHg or greater, but a mean HBP value of lower than 135/85 mmHg [8]. A stress electrocardiography was performed in cases with an abnormal electrocardiography and/or history of angina pectoris. A coronary angiography was obtained just for the stress electrocardiography positive cases. So CHD was diagnosed either angiographically or with a history of coronary artery stenting and/or coronary artery bypass graft surgery. Eventually, all cases with CHD were divided into two groups according to gender distribution, and the mean age, weight, BMI, LDL-C, and TG values and prevalences of smokers, COPD, WCH, HT, and DM were compared in between. Mann-Whitney U test, Independent-Samples T test, and comparison of proportions were used as the methods of statistical analyses.

Results

The study included 1,620 females and 1,240 males. Mean ages of them were 41.7 and 40.8 years, respectively (p>0.05). Characteristic features of the study cases were summarized in table 1. Prevalence of the CHD was similar in both sexes (3.8% versus 4.4%, respectively, p>0.05). Mean ages of the CHD were 61.5 versus 63.5 years, respectively (p>0.05). Prevalence of smoking was significantly higher in males with CHD (54.5% versus 9.6%, p<0.001). Parallel to the higher prevalence of smoking, prevalence of COPD was also significantly higher in males (18.1% versus 6.4%, p<0.05). On the other hand, although the mean weight of males with CHD was significantly higher (79.1 versus 74.4 kg, p=0.027), the females had a higher mean BMI value (29.7 versus 28.3 kg/m^2, p>0.05), but the difference was statistically nonsignificant probably due to the small sample sizes of the groups. Similarly, the mean LDL-C and TG values were significantly higher in the females, too (132.6 versus 115.6 mg/dL, p=0.008 and 250.3 versus 150.1 mg/dL, p=0.002, respectively). Although prevalence of WCH was also higher in females, the difference was nonsignificant probably due to the small sample sizes of the groups again (30.6% versus 23.6%, p>0.05). Additionally, prevalences of HT and DM were also higher in females, significantly (58.0% versus 30.9%, p<0.001 and 51.6% versus 38.1%, p<0.05, respectively).

Variables	Males with CHD*	Females with CHD	*p*-value
Prevalence	4.4% (55/1,240)	3.8% (62/1,620)	ns†
Mean age (year)	63.5 ± 10.8 (43-82)	61.5 ± 11.2 (42-88)	ns
Prevalence of smokers	*54.5% (30)*	*9.6% (6)*	*<0.001*
Prevalence of COPD‡	*18.1% (10)*	*6.4% (4)*	*<0.05*
Mean weight (kg)	*79.1 ± 12.9 (58-116)*	*74.4 ± 18.7 (42-129)*	*0.027*
Mean BMI§ (kg/m^2)	28.3 ± 4.7 (20.6-46.9)	29.7 ± 6.7 (19.0-48.6)	ns
Mean LDL-C‖ (mg/dL)	*115.6 ± 38.5 (43-192)*	*132.6 ± 47.3 (10-232)*	*0.008*
Mean TG¶ (mg/dL)	*150.1 ± 113.4 (53-594)*	*250.3 ± 233.9 (81-1380)*	*0.002*
Prevalence of WCH**	23.6% (13)	30.6% (19)	ns
*Prevalence of HT****	*30.9% (17)*	*58.0% (36)*	*<0.001*
*Prevalence of DM*****	*38.1% (21)*	*51.6% (32)*	*<0.05*

*Coronary heart disease †Nonsignificant (p>0.05) ‡Chronic obstructive pulmonary disease §Body mass index ‖Low density lipoprotein cholesterol ¶Triglyceride **White coat hypertension ***Hypertension ****Diabetes mellitus

Table 1: Characteristic features of the study cases.

Discussion

Metabolic syndrome is actually exaggerated by metabolic risk factors for the development of systemic atherosclerosis, and the symptomatic atherosclerosis is probably the leading cause of death for both sexes in human being. Smoking and excess weight are probably the most significant accelerating factors of the metabolic syndrome [10]. Definition of the syndrome include both reversible metabolic risk factors including overweight, smoking, WCH, IFG, IGT, hypertriglyceridemia, hyperbetalipoproteinemia, and dyslipidemia and final diseases including aging, obesity, COPD, hepatic cirrhosis, CRF, HT, DM, CHD, PAD, and stroke [11,12]. In a previous study [13], prevalences of hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, IGT, and WCH had a parallel fashion to excess weight by increasing until the seventh decade of life and decreasing afterwards, significantly (p<0.05 nearly in all steps). On the other hand, prevalences of HT, DM, and CHD always continued to increase by aging without any decrease (p<0.05 nearly in all steps) indicating their irreversible properties [13]. After development of one of the terminal diseases, the nonpharmaceutical approaches will provide little benefit to prevent development of the others, probably due to cumulative effects of the metabolic risk factors on endothelial system for a long period of time [11,12]. Obesity is probably found among one of the terminal accelerating diseases of the syndrome since after the development of obesity, pharmaceutical and nonpharmaceutical approaches will provide little benefit either to heal obesity or to prevent its complications.

Excess weight probably leads to a chronic and low-grade inflammatory process on the endothelial system, and risk of death from all causes including cardiovascular diseases and cancers increases parallel to the range of weight excess in all age groups [14]. The excess weight induced increased risk of cancers may either be related with the chronic inflammatory process or systemic atherosclerosis. The low-grade chronic inflammation may cause genetic changes on epithelial cells of the organs, and the systemic atherosclerosis may decrease clearence of malignant cells by the immune system, effectively. Effects of body weight on BP were shown in a study [15] that the prevalence of sustained normotension (NT) was significantly higher in the underweight (80.3%) than the normal weight (64.0%) and overweight cases (31.5%, p<0.05 for both), and 55.1% of cases with HT had obesity against 26.6% of cases with NT (p<0.001) in another study [16]. So the dominant underlying risk factor of the metabolic syndrome appears as an already existing excess weight or a trend towards excess weight, which is probably the main cause of insulin resistance, dyslipidemia, IGT, and WCH via the chronic inflammatory process [4]. Even prevention of the accelerating trend of weight gain with diet or exercise, even in the absence of a prominent weight loss, will probably result with resolution of many parameters of the syndrome [17,18]. But according to our opinion, limitation of excess weight as an excessive fat tissue in and around abdomen under the heading of abdominal obesity is meaningless, instead it should be defined as overweight or obesity via BMI, since adipocytes function as an endocrine organ

that produces a variety of cytokines and hormones anywhere in the body [4]. The resulting hyperactivity of sympathetic nervous system and renin-angiotensin-aldosterone system is probably associated with chronic endothelial inflammation, insulin resistance, and an elevated BP. Similarly, the Adult Treatment Panel III reported [7] that although some people classified as overweight with a large muscular mass, most of them also have excess fat tissue, and excess weight not only predispose to CHD, stroke, and numerous other conditions, but also has a high burden of other CHD risk factors including dyslipidemia, HT, and type 2 DM.

It is already known that smoking is a major risk factor for the development of symptomatic atherosclerosis such as CHD, PAD, and probably COPD [19]. Its atherosclerotic effects are the most obvious in Buerger's disease (thromboangiitis obliterans). It is an obliterative disease characterized by inflammatory changes in small and medium-sized arteries and veins, and it has not been documented in nonsmokers, implicating cigarette smoking as a primary etiologic factor. Although the known strong atherosclerotic effects of smoking, some studies reported that smoking in humans and nicotine administration in animals are associated with a decreased body weight [20]. Evidence revealed an increased energy expenditure while smoking, both during rest and light physical activity [21], and nicotine supplied by patch after smoking cessation decreased caloric intake in a dose-related manner [22]. According to an animal study, nicotine may lengthen intermeal time and simultaneously decrease amount of meal eaten [23]. Additionally, body weight seems to be the highest in former, the lowest in current and medium in never smokers [24]. In another study, there was a relationship between overweight and smoking in men [25]. Smoking may be associated with postcessation weight gain, but evidence suggests that risk of weight gain is the highest during the first year after quitting and declines over the years [26]. Similarly, although the CHD were detected with similar prevalences in both sexes in the present study, prevalences of smoking and COPD were higher in males against the higher prevalences of BMI, WCH, LDL-C, TG, HT and DM in females as the other atherosclerotic risk factors. This result may indicate both the strong atherosclerotic and weight decreasing roles of smoking. Similarly, the incidence of a myocardial infarction is increased sixfold in women and threefold in men who smoke at least 20 cigarettes per day compared to the never smoked cases [27]. In other words, smoking is more harmful for women with CHD probably due to the associated higher BMI and its consequences in women. Similar to our results, the proportion of smokers is consistently higher in men in the literature [19]. So smoking is probably a powerful atherosclerotic risk factor with some suppressor effects on appetite. On the other hand, smoking, as a pleasure in life, may also show the weakness of volition of the individuals to control eating in the metabolic syndrome, so it comes with additional excess weight and its complications although its some inhibitory effects on appetite in front. Similarly, prevalences of HT, DM, and smoking were the highest in the highest TG having group as a significant component of the metabolic syndrome in another study [12].

As a conclusion, metabolic syndrome is a systemic atherosclerotic process exaggerated by some metabolic disorders. Smoking and excess weight may be the major triggering causes of the syndrome, and they come with similar degree of clinical severity in front. Smoking and COPD were higher in males with CHD against the higher BMI, WCH, LDL-C, TG, HT, and DM in females, resulting with similar prevalences of CHD in both sexes.

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