Excess weight or smoking

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Abstract

Background: Smoking may cause loss of appetite via endothelial inflammation all over the body.

Methods: Consecutive patients with coronary artery disease (CAD) were studied.

Results: Study included 2,860 cases (1,620 females), totally. Prevalence of CAD was similar in females and males (3.8% versus 4.4%, respectively, p>0.05) with mean ages of 61.5 versus 63.5 years, respectively (p>0.05). Smoking and chronic obstructive pulmonary disease (COPD) were significantly higher in males with CAD (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 CAD (29.7 versus 28.3 kg/m2 and 30.6% versus 23.6%) but differences were non-significant (p>0.05 for both) probably due to small sample sizes of CAD groups. Whereas low density lipoprotein cholesterol (LDL-C) (132.6 versus 115.6 mg/dL, p= 0.008), triglyceride (TG) (250.3 versus 150.1 mg/dL, p= 0.002), hypertension (HT) (58.0% versus 30.9%, p<0.001), and diabetes mellitus (DM) (51.6% versus 38.1%, p<0.05) were all higher in females with CAD, significantly.

Conclusion: Smoking and excess weight may be the major underlying causes of metabolic syndrome in Turkey, and they may cause a similar degree of clinical severity. Smoking and COPD were higher in males with CAD against the higher BMI, WCH, LDL-C, TG, HT, and DM in females terminating with similar prevalence of CAD in both genders. To be able to understand the clear effects of smoking on BMI and its consequences, the next study should be performed on non-smokers in both genders.

Key words: Excess weight, smoking, atherosclerosis, metabolic syndrome, early aging, premature death

Introduction

Due to the prolonged survival of human beings, systemic atherosclerosis may be the main health problem in this century, and its associations with some metabolic disorders and smoking are collected under the title of metabolic syndrome in the literature (1, 2). The syndrome is characterized by a low-grade chronic inflammatory process on vascular endothelium initiated in early years of life (3). The inflammatory process is initiated by some factors including overweight, obesity, smoking, alcohol, and chronic infections and inflammations (4, 5). The syndrome can be slowed down with appropriate non-pharmaceutical approaches including lifestyle changes, diet, and exercise (6). The metabolic syndrome may contain white coat hypertension (WCH), impaired fasting glucose (IFG), impaired glucose tolerance (IGT), hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, overweight, alcohol, and smoking for the development of irreversible consequences including hypertension (HT), type 2 diabetes mellitus (DM), obesity, chronic obstructive pulmonary disease (COPD), cirrhosis, chronic renal disease (CRD), peripheric artery disease (PAD), coronary artery disease (CAD), and stroke (7). In another perspective, the metabolic syndrome may be the most significant disease of human life decreasing its quality and duration at the moment. The syndrome has become increasingly common all over the world, for example 50 million people in the United States have it (8). The syndrome induced accelerated atherosclerotic process all over the body may be the leading cause of early aging and premature death for both genders. For example, CAD is the leading cause of death in developed countries. Although CAD may be equally seen in both genders (7), there may be some gender differences about the risk factors of CAD. We tried to understand whether or not there are some gender differences according to the atherosclerotic risk factors in patients with CAD 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 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 index (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 (9). 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 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 education about proper BP measurement techniques (10). A 24-hour ambulatory blood pressure (ABP) monitoring was not required due to its equal effectiveness with HBP measurements (11). 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 (10). 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 CAD was diagnosed either angiographically or with a history of coronary artery stenting and/or coronary artery bypass graft surgery. Eventually, all cases with CAD were divided into two groups according to gender distribution, and they 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 were 41.7 and 40.8 years, respectively ($p>0.05$). Prevalence of CAD was similar in females and males (3.8% versus 4.4%, respectively, $p>0.05$). Mean ages of the CAD cases were 61.5 versus 63.5 years, respectively ($p>0.05$) (Table 1). Prevalence of smoking was significantly higher in males with CAD (54.5% versus 9.6%, $p<0.001$). Parallel to the higher prevalence of smoking, prevalence of COPD was also higher in males, significantly (18.1% versus 6.4%, $p<0.05$). On the other hand, although the mean weight of males with CAD 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/m2, $p>0.05$), but the difference was statistically non-significant probably due to the small sample sizes of the CAD groups. Similarly, the mean LDL-C and TG values were significantly higher in females with CAD, 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 non-significant probably due to the small sample sizes of the CAD groups again (30.6% versus 23.6%, $p>0.05$). Although the small sample sizes of the CAD groups, prevalence of HT and DM was significantly higher in females with CAD (58.0% versus 30.9%, $p<0.001$ and 51.6% versus 38.1%, $p<0.05$, respectively).

Table 1: Characteristic features of the study cases

Variables	Males with CAD*	Females with CAD	*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/m²)	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 artery disease †Nonsignificant (p>0.05) ‡Chronic obstructive pulmonary disease §Body mass index ‖Low density lipoprotein cholesterol ¶Triglyceride **White coat hypertension ***Hypertension ****Diabetes mellitus

Discussion

Vascular endothelial inflammation is actually caused by some metabolic risk factors for the development of systemic atherosclerosis, and the symptomatic atherosclerosis is probably the leading cause of early aging and premature death for both genders in human beings. Smoking, alcohol, and excess weight are probably the most common causes of the systemic endothelial damage (12). Definition of the metabolic syndrome or aging syndrome or accelerated endothelial damage syndrome includes metabolic risk factors including overweight, smoking, alcohol, WCH, IFG, IGT, hypertriglyceridemia, hyperbetalipoproteinemia, and dyslipidemia for the development of irreversible endpoints including HT, DM, obesity, COPD, cirrhosis, CRD, PAD, CAD, stroke, early aging, and premature death (13, 14). In a previous study, prevalence 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) (15). On the other hand, prevalence of HT, DM, and CAD always continued to increase by aging without any decrease ($p<0.05$ nearly in all steps) indicating their irreversible properties (15). After development of one of the irreversible consequences, the nonpharmaceutical approaches will provide little benefit to prevent development of the others probably due to cumulative effects of the risk factors on the endothelial system for a long period of time all over the body (13, 14).

Obesity is probably found among one of the irreversible consequences of the metabolic syndrome since after the development of obesity, non-pharmaceutical approaches provide limited success either to heal obesity or to prevent its complications. Overweight and obesity probably lead to a chronic and low-grade inflammatory process on vascular 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 (16). The low-grade chronic inflammation may cause genetic changes on epithelial cells, and the systemic atherosclerosis may decrease clearance of malignant cells by the immune system, effectively (17). For example, effects of excess weight on BP were shown in a study 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) (18), and 52.8% of cases with HT had obesity against 14.5% of cases with NT ($p<0.001$) in another study (19). So the dominant underlying risk factor of the metabolic syndrome appears as weight gaining, which is probably the main cause of insulin resistance, dyslipidemia, IGT, and WCH

by means of a chronic inflammatory process (6). Even prevention of the accelerating trend of weight gaining with diet or exercise, even in the absence of a prominent weight loss, will probably result with resolution of many adverse parameters of the syndrome (20, 21). But according to our opinion, limitation of excess weight as an excessive fat tissue in and around the abdomen under the heading of abdominal obesity is meaningless, instead it should be defined as overweight or obesity by means of BMI since adipocytes function as an endocrine organ that produces a variety of cytokines and hormones anywhere in the body (6). The resulting hyperactivities of sympathetic nervous system and renin-angiotensin-aldosterone system are probably associated with chronic endothelial inflammation, insulin resistance, and an elevated BP. Similarly, the Adult Treatment Panel III reported that although some people were classified as overweight with a large muscular mass, most of them also have excess fat tissue, and excess weight does not only predispose to CAD, stroke, and other endpoints, it also has a high burden of other CAD risk factors including dyslipidemia, HT, and DM (9).

Smoking is a major risk factor for the development of atherosclerotic endpoints such as CAD, PAD, COPD, cirrhosis, CRD, and stroke (17, 22). 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 non-smokers, 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 (23). Evidence revealed an increased energy expenditure during smoking both on rest and light physical activity (24), and nicotine supplied by patch after smoking cessation decreased caloric intake in a dose-related manner (25). According to an animal study, nicotine may lengthen intermeal time and simultaneously decreases amount of meal eaten (26). Additionally, body weight seems to be the highest in former, the lowest in current and medium in never smokers (27). 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 (28). Similarly, although the CAD was detected with similar prevalences in both genders in the previous study (7), prevalence of smoking and COPD were higher in males with CAD against the higher prevalence 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 (29). In other words, smoking is more harmful for women regarding CAD, 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 (22). So smoking is probably a powerful atherosclerotic risk factor with some suppressor effects on appetite.

Smoking-induced weight loss may be related with the smoking-induced endothelial inflammation all over the body, since loss of appetite is one of the major symptoms of inflammations in the body. Physicians can even understand healing of their patients from their returning appetite. Several toxic substances found in cigarette smoke get into the circulation by means of the respiratory system, and they probably cause a subclinical vascular endothelial inflammation until their clearance from the circulation. But due to the continuous smoking habits of the individuals, the clearance process never terminates. So the patients become ill with loss of appetite, subclinically. In another explanation, smoking-induced loss of weight is an indicator of being ill instead of being healthy during smoking (25-27). After smoking cessation, loss of appetite comes back with a prominent weight gaining in the patients but the returned weight is their physiological or 'usual' weights, actually. On the other hand, smoking as a pleasure in life may also show the weakness of volition to control eating in the metabolic syndrome, so it comes with additional weight excess and its complications although there are some inhibitory effects of it on appetite. Similarly, prevalence of HT, DM, and smoking were the highest in the highest TG having group as another significant component of the metabolic syndrome in another study (14).

Alcohol may also cause severe endothelial damage not only in the vasculature of gastrointestinal tract instead all over the body (30, 31). It may have similar adverse effects with smoking on vascular endothelium with different severity in different organs (17). In other words, alcohol causes COPD and smoking causes cirrhosis, too. Both of them affect both arterial and venous endothelial cells all over the body. Buerger's disease alone is clear evidence to show the strong atherosclerotic effects of smoking since this disease has not been shown in the absence of smoking in the literature. Similarly, the alcoholic cirrhosis alone is clear evidence to show strong atherosclerotic effects of alcohol. The effects of smoking and alcohol on vascular endothelium terminate with a symptomatic atherosclerosis, end-organ insufficiencies, early aging, and premature death, therefore they must be included among the parameters of the metabolic syndrome. We did not study the effects of alcohol due to its low prevalence in Turkey in the present study.

Male gender alone may also be a significant factor for the accelerated atherosclerotic process since females live longer than males all over the world (32). Concern to protect his family is a feature of male sex in human beings and even in animal kinds. The feature probably comes from testosterone. You rarely see females fighting with each other for a male but you can easily see some fighting males for a female in human beings and especially in animal species. You can see soldiers or coalmine workers in males but rarely in females. Males use their physical force more in daily life. The dominant physical role of male sex is also seen during sexual activities. The overuse of body probably

causes an accelerated atherosclerosis and a shortened lifespan in males. The shortened survival of male gender was even shown in the sickle cell patients although they have significantly shorter mean life expectancy with the current health services (33). Due to some antidepressive properties of smoking and alcohol, they are also more common in males all over the world which may also indicate presence of some additional stresses on the male gender in society. But the longer lifespan of females cannot be explained by the strong atherosclerotic effects of smoking and alcohol alone. Effects of testosterone may also be important in the shortened survival of males. So the dominant role of male gender, smoking, and alcohol put them into the accelerated atherosclerotic process whereas excess weight is the major problem in females regarding the accelerated aging process. In other words, overuse of the body in males and underuse of the body in females may accelerate the endothelial inflammation and damage, atherosclerosis, early aging, and premature death. Avoidance of smoking, alcohol, and excess weight are essential in protection from the metabolic syndrome. On the other hand, the term of regular exercise should be replaced with daily and essential activities in the protection of females since they actually need a lifestyle change instead of exercise. Avoidance of animal-rich diet, walking as much as possible, avoidance of elevators, eating fruit with its peel even to escape chronic constipation, drinking tea, finding daily responsibilities, finding new aims to live, and not using taxis should be the lifestyles of people under the risk of syndrome.

As a conclusion, metabolic syndrome or aging syndrome or accelerated endothelial damage syndrome is a systemic and inflammatory atherosclerotic process. Smoking and excess weight may be the major underlying causes of the syndrome in Turkey, and they may cause similar degrees of clinical severity. Smoking and COPD were higher in males with CAD against the higher BMI, WCH, LDL-C, TG, HT, and DM in females terminating with a similar prevalence of CAD in both genders. To be able to understand the clear effects of smoking on BMI and its consequences, the next study should be performed on non-smokers in both genders.

References

1. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005; 365(9468): 1415-1428.
2. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation 2004; 109(3): 433-438.
3. Tonkin AM. The metabolic syndrome(s)? Curr Atheroscler Rep 2004; 6(3): 165-166.
4. Haidar, Soeatmadji DW. Effects of high-carbohydrate and high fat diet on formation of foam cells and expression of TNF-alpha in Rattus novergicus. Acta Med Indones 2007; 39(3): 119-123.
5. Rudijanto A. The role of vascular smooth muscle cells on the pathogenesis of atherosclerosis. Acta Med Indones 2007; 39(2): 86-93.
6. Franklin SS, Barboza MG, Pio JR, Wong ND. Blood pressure categories, hypertensive subtypes, and the metabolic syndrome. J Hypertens 2006; 24(10): 2009-2016.
7. Helvaci MR, Kaya H, Gundogdu M. Gender differences in coronary heart disease in Turkey. Pak J Med Sci 2012; 28(1): 40-44.
8. Clark LT, El-Atat F. Metabolic Syndrome in African Americans: implications for preventing coronary heart disease. Clin Cardiol 2007; 30(4): 161-164.
9. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation 2002; 17:106(25): 3143-3421.
10. O'Brien E, Asmar R, Beilin L, Imai Y, Mallion JM, Mancia G, et al. European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. J Hypertens 2003; 21(5): 821-848.
11. Helvaci MR, Seyhanli M. What a high prevalence of white coat hypertension in society! Intern Med 2006; 45(10): 671-674.
12. Hunt KJ, Resendez RG, Williams K, Haffner SM, Stern MP. National Cholesterol Education Program versus World Health Organization metabolic syndrome in relation to all-cause and cardiovascular mortality in the San Antonio Heart Study. Circulation 2004; 110(10): 1251-1257.
13. Helvaci MR, Kaya H, Sevinc A, Camci C. Body weight and white coat hypertension. Pak J Med Sci 2009; 25(6): 6: 916-921.
14. Helvaci MR, Kaya H, Gundogdu M. Association of increased triglyceride levels in metabolic syndrome with coronary artery disease. Pak J Med Sci 2010; 26(3): 667-672.
15. Helvaci MR, Kaya H, Seyhanli M, Yalcin A. White coat hypertension in definition of metabolic syndrome. Int Heart J 2008; 49(4): 449-457.
16. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med 1999; 341(15): 1097-1105.
17. Helvaci MR, Aydin Y, Gundogdu M. Smoking induced atherosclerosis in cancers. HealthMED 2012; 6(11): 3744-3749.
18. Helvaci MR, Kaya H, Yalcin A, Kuvandik G. Prevalence of white coat hypertension in underweight and overweight subjects. Int Heart J 2007; 48(5): 605-613.
19. Helvaci MR, Kaya H, Duru M, Yalcin A. What is the relationship between white coat hypertension and dyslipidemia? Int Heart J 2008; 49(1): 87-93.
20. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi T, Azizi F. Beneficial effects of a Dietary Approaches to Stop Hypertension eating plan on features of the metabolic syndrome. Diabetes Care 2005; 28(12): 2823-2831.
21. Helvaci MR, Kaya H, Borazan A, Ozer C, Seyhanli M, Yalcin A. Metformin and parameters of physical health. Intern Med 2008; 47(8): 697-703.

22. Fodor JG, Tzerovska R, Dorner T, Rieder A. Do we diagnose and treat coronary heart disease differently in men and women? Wien Med Wochenschr 2004; 154(17-18): 423-425.
23. Grunberg NE, Greenwood MR, Collins F, Epstein LH, Hatsukami D, Niaura R, et al. National working conference on smoking and body weight. Task Force 1: Mechanisms relevant to the relations between cigarette smoking and body weight. Health Psychol 1992; 11: 4-9.
24. Walker JF, Collins LC, Rowell PP, Goldsmith LJ, Moffatt RJ, Stamford BA. The effect of smoking on energy expenditure and plasma catecholamine and nicotine levels during light physical activity. Nicotine Tob Res 1999; 1(4): 365-370.
25. Hughes JR, Hatsukami DK. Effects of three doses of transdermal nicotine on post-cessation eating, hunger and weight. J Subst Abuse 1997; 9: 151-159.
26. Miyata G, Meguid MM, Varma M, Fetissov SO, Kim HJ. Nicotine alters the usual reciprocity between meal size and meal number in female rat. Physiol Behav 2001; 74(1-2): 169-176.
27. Laaksonen M, Rahkonen O, Prattala R. Smoking status and relative weight by educational level in Finland, 1978-1995. Prev Med 1998; 27(3): 431-437.
28. Froom P, Melamed S, Benbassat J. Smoking cessation and weight gain. J Fam Pract 1998; 46(6): 460-464.
29. Prescott E, Hippe M, Schnohr P, Hein HO, Vestbo J. Smoking and risk of myocardial infarction in women and men: longitudinal population study. BMJ 1998; 316(7137): 1043-1047.
30. Schoepf D, Heun R. Alcohol dependence and physical comorbidity: Increased prevalence but reduced relevance of individual comorbidities for hospital-based mortality during a 12.5-year observation period in general hospital admissions in urban North-West England. Eur Psychiatry 2015; 30(4): 459-468.
31. Singh G, Zhang W, Kuo YF, Sharma G. Association of Psychological Disorders With 30-Day Readmission Rates in Patients With COPD. Chest 2016; 149(4): 905-915.
32. Mathers CD, Sadana R, Salomon JA, Murray CJ, Lopez AD. Healthy life expectancy in 191 countries, 1999. Lancet 2001; 357(9269): 1685-1691.
33. Helvaci MR, Gokce C, Davran R, Akkucuk S, Ugur M, Oruc C. Mortal quintet of sickle cell diseases. Int J Clin Exp Med 2015; 8(7): 11442-11448.