Original Article

Association of Sociodemographic Profile, Dyslipidemias, and Obesity in Smoker, Former Smoker, and Nonsmoker Patients with Coronary Artery Disease

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ABSTRACT

Introduction: In patients with established coronary artery disease (CAD), there are different reports on gender, age, dyslipidemia, and obesity according to smoking behavior. Smoking, obesity, and dyslipidemia are targets in secondary prevention. In this study, we aimed to investigate the sociodemographic differences, lipid profiles, body mass index (BMI), and cigarette smoking status in patients diagnosed with CAD. **Methods:** Patients with records of angiography, smoking behavior, sociodemographic information, lipid levels, and BMI present at the outpatient visits were included in the study. Patients were grouped as active smokers, nonsmokers, and former smokers. Statistical methods were used for comparison of variables and means. **Results:** A total of 235 patients, 167 (71.1%) men and 68 (28.9%) women, were included in the study. Nonsmokers group (31.4%) consisted of mostly women while active (22.6%) and former smoker (46%) groups consisted mostly of men (*P* < 0.0001). The mean age was 60.65 ± 11.55. Age was associated with the smoking status of patients, and nonsmokers consisted of geriatric patients significantly (*P* = 0.001). Educational status was associated with smoking history. Cessation of smoking after CAD diagnosis was achieved in 46% of patients. Active smokers had highest mean triglyceride (TG) values while nonsmokers had highest mean high-density lipoprotein-cholesterol (HDL-C) values. Active smokers had the highest mean TG values while nonsmokers had the highest mean HDL-C values. BMI was higher in nonsmokers than active smokers. **Conclusion:** Gender, age, and educational status are determinants of smoking behavior in patients with CAD. BMI is associated with smoking history. In nonsmokers, values of HDL were highest while TG values were lowest. To prevent reoccurring cardiovascular events, young patients and men are two groups which health professionals need to concentrate in motivating to quit smoking.

KEYWORDS: *Coronary artery disease, dyslipidemias, obesity, smoking cessation*

INTRODUCTION

*A*lthough effects of smoking on cardiovascular disease is quite clear, studies show that patients continue to smoke even after they are diagnosed with coronary artery disease (CAD).[1] In patients with established atherosclerotic CAD, smoking, obesity, and dyslipidemia are targeted risk factors in secondary prevention. Smoking cessation is encouraged by health professionals along with other lifestyle modifications.[2] Smoking and obesity enhance the burden of cardiovascular risk factors including dyslipidemia.[3,4] Thus, the management of serum lipid levels is crucial in patients[2] to reduce the incidence of atherosclerotic events.[2] Cigarette smoking

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increases total cholesterol (TC), triglycerides (TGs), and low-density lipoprotein (LDL) while the cardioprotective high-density lipoprotein (HDL) is decreased.[5] Many researchers worldwide are investigating the mechanisms of effects of smoking on lipid metabolism. Exposure to cigarette smoke may lead to oxidative stress and plasma antioxidant depletion. Thus, HDL particles may undergo changes in structure or composition and cannot protect LDL particles against oxidation. Recently, it has been reported that with smoking, antiaterogenic prostacyclin production decreases which inhibits platelet activation and is also an effective vasodilator.[6]

Associations between smoking behavior and dyslipidemias have been investigated intensely, and there are different reports on the effects of smoking behavior on lipid levels. In a previous meta-analysis, it was reported that in smokers, TC, TG, very LDL (VLDL-C), and LDL-C concentrations were higher while HDL-C levels were lower when compared to nonsmokers.[7] However, in another meta-analysis, it was reported that levels of LDL-C, TC, and TG do not change, but HDL-C may increase with smoking cessation.[8] In a more recent study, in active smokers, LDL-C levels were normal and, in heavy smokers, the levels of TG were significantly high, while HDL-C levels were significantly lower when compared with patients who have cessated smoking or who were nonsmokers.[3]

In a cohort study, the risk for recurrent cardiovascular events in patients with known CAD, has been found to increase most with presence of diabetes mellitus, followed by increased LDL/HDL ratio and smoking.[9] It was reported that body mass index (BMI), increased alcohol consumption, and age could affect the levels of serum lipid levels regardless of smoking status. However, smoking status was the most effective independent risk factor for dyslipidemia.[3]

It is well known that smoking behavior influences BMI and was found that current smokers have lower BMI values than nonsmokers and female former smokers had higher BMI values;[10] following an acute coronary event, weight gain has been observed in patients on smoking cessation.[11] Despite a history of acute coronary syndrome (ACS), some patients continue to have the risk factors of smoking and being obese.[12] In a multicenter study, the highest risk for ACS was attributed to young male current smokers, whereas old women had the lowest risk.[13]

There are limited studies on how obesity and dyslipidemia are affected by smoking behavior during the follow-up of patients with documented CAD and on the influence of sociodemographic factors such as age,

gender, and educational status on the smoking behavior in this population. Here, we aimed to investigate the association between lipid profiles, BMI, age, and gender or educational status in current smokers, nonsmokers, and former smokers who have been diagnosed with CAD angiographically.

METHODS

Study subjects and data collection

This is a retrospective study depending on records of patients diagnosed as CAD (≥50% stenosis of the coronary arteries detected angiographically). Patients admitted to our hospital for their routine outpatient visits after angiography who had information about their smoking behavior in addition to their sociodemographic and clinical data were included in the study. Patients were grouped according to gender, age, education status, lipid profiles, and BMI. Comparisons were made between groups according to the smoking behavior.

Lipid profile and body mass index

Fasting values of HDL-C <40 mg/dL in men and HDL-C <50 mg/dL in women were considered low and TC ≥200 mg/dL, LDL-C ≥100 mg/dL, and TGs ≥150 mg/dL were considered high.[14] Patients with BMI ≥30 were considered obese.

Assessment of smoking behavior

Smoking status at the time of outpatient visits was obtained. Patients who reported that they have been smoking in the past 6 months were grouped as "active smokers." Patients who have never smoked were grouped as "nonsmokers," while patients who have been on smoking cessation for at least 6 months were "former smokers." Active smokers and former smokers were grouped together as "patients with a smoking history" and were compared with nonsmokers. Nonsmokers and former smokers were also grouped together and compared with active smokers. The average number of cigarettes (1 package = 20 cigarettes) consumed per person was also recorded.

Statistical analysis

The descriptive and analytical statistical analyses were performed using the SPSS (Statistical Package for the Social Sciences, Chicago, IL, USA) version 21.0. Descriptive statistics were given in terms of means, medians where necessary and as standard deviations, counts, and percentages. Student's *t*-test, one-way ANOVA and Mann–Whitney U-test, and Kruskal–Wallis test were used when necessary. Chi-square test was used to compare proportions in different groups. $P < 0.05$ was considered to show a statistically significant result. Correlation coefficient (r) was accepted as follows: 0–0.24 weak, 0.25–

0.49 medium, 0.50–0.74 strong, and 0.75–1.00 very strong. Correlation coefficients and their significance between variables were calculated by Spearman's rho test and Pearson test. Exclusion criteria were the presence of renal, liver, and thyroid diseases, having TG values >400 mg/dl, and using steroids. The results were presented within 95% confidence intervals and *P*. The study was approved by the Research Ethics Committee of the hospital.

RESULTS

Sociodemographic factors: Age, gender, and educational status

Records of 235 patients, 168 (71.5%) men and 67 (28.5%) women, with smoking status information were gathered. Data for lipid profile were present in 202 (86%) patients while data for BMI at the time of the outpatient visit were obtained in 152 (64.7%) patients. Smoking behavior according to different characteristics of patients is shown in Table 1.

Forty-six percent (*n* = 108) of patients cessated smoking after they were diagnosed with CAD, 31.4% (*n* = 74) never smoked and 22.6% (*n* = 53) were active smokers. Patients with a smoking history (former and active smokers) (*n* = 161; 68.5%) had a mean cigarette consumption of 38.06 ± 33.07 packages/year.

Patients were 30–85 years old. The mean age was 60.29 ± 11.55. Majority of our patients were aged over 50 years (81.3%).

When patients were grouped according to ages, there was a significant difference between their smoking behavior (*P* = 0.001) [Table 2]. The former smoker group was aged between 51 and 64 years while the active smoker group had younger individuals (*P* = 0.001). The nonsmoker group had the highest mean age and was comprised geriatric patients (*P* = 0.001) [Figure 1].

In the study group, men (71.5%) were more frequent compared with women (28.5%). Smoking status of patients was different between genders (*P* < 0.0001). Women were more frequent in the nonsmokers group while men were more in the group with a smoking history (*P* < 0.0001). Men were significantly more frequently presented in the former smoker group (*P* < 0.0001).

Primary school graduates made the largest group of our study (68.3%). There was a significant difference between the educational and smoking status of participants (*P* < 0.0001).

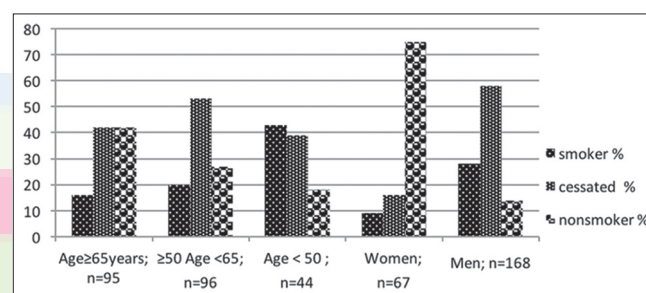The patients who had primary school education constituted the majority of all groups according to

Figure 1: Gender and age characteristics of patients according to smoking behavior, *n* = 235, 100%

Characteristics of patients	*n*/total *n** (%)	Active smoker/total *n** (%)	Former smoker/total *n* * (%)	Nonsmoker/Total *n** (%)
Men	168/235 (71.5)	47/53 (88.7)	97/108 (89.81)	24/74 (32.43)
Women	67/235 (28.5)	6/53 (11.3)	11/108 (10.19)	50/74 (67.57)
Age (years)				
30-50	44/235 (18.7)	19/53 (35.85)	17/108 (15.74)	8/74 (10.81)
51-64	96/235 (40.9)	19/53 (35.85)	51/108 (47.22)	26/74 (35.14)
≥65	95/235 (40.4)	15/53 (28.30)	40/108 (37.04)	40/74 (54.05)
Graduate of secondary school	29/235 (12.3)	7/53 (13.21)	15/108 (13.89)	7/74 (9.46)
Graduate of primary school	159/235 (67.7)	42/53 (79.2)	81/108 (75.00)	36/74 (48.65)
Primary school education <3 years	18/235 (7.6)	3/53 (5.7)	2/108 (1.85)	13/74 (17.57)
Illiterate	29/235 (12.3)	1/53 (1.89)	10/108 (9.26)	18/74 (24.32)
BMI				
≥30	67/152 (44.1)	4/17 (23.5)	38/85 (44.70)	25/50 (50.00)
<30	85/152 (55.9)	13/17 (76.5)	47/85 (55.30)	25/50 (50.00)
Triglyceride ≥150	103/202 (50.9)	26/42 (61.9)	48/98 (48.98)	29/62 (46.77)
Total cholesterol ≥200	83/202 (41.1)	15/42 (35.7)	42/98 (42.85)	26/62 (41.93)
HDL-C <40; <50 (men; women)	75/202 (62.0)	17/42 (40.5)	34/98 (34.69)	24/62 (38.70)
LDL-C ≥100	124/202 (37.1)	23/42 (54.7)	66/98 (67.34)	35/62 (56.45)

*Data which we could retrieve from the records of the patients. The number of patients (*n*) is different for different characteristics.
BMI=Body mass index; LDL-C=Low-density lipoprotein-cholesterol; HDL-C=High-density lipoprotein-cholesterol

Table 2: Mean values of age, body mass index, and lipid profile of patients and smoking behavior

	Smoking behavior	n*	Mean±SD	95% CI for mean		P
				Lower bound	Upper bound	
Age (years)	Smoker	53	56.17±12.80	52.64	59.70	0.001
	Former smoker	108	60.69±10.34	58.71	62.66	
	Nonsmoker	74	63.92±11.37	61.24	66.59	
	Total patients	235	60.65±11.55	59.16	62.15	
Triglycerides	Smoker	42	190.15±93.00	160.79	219.50	0.627
	Former smoker	98	173.96±103.86	153.14	194.79	
	Nonsmoker	62	172.86±93.03	149.43	196.29	
	Total patients	202	176.90±98.19	163.28	190.53	
Total cholesterol	Smoker	42	190.33±42.49	177.09	203.58	0.945
	Former smoker	98	192.88±46.66	183.48	202.29	
	Nonsmoker	62	193.29±50.49	180.46	206.11	
	Total patients	202	192.47±46.84	185.96	198.99	
HDL-C	Smoker	42	43.674±10.18	40.50	46.84	0.158
	Former smoker	98	43.249±8.82	41.46	45.03	
	Nonsmoker	62	46.265±11.07	43.45	49.07	
	Total patients	202	44.273±9.89	42.89	45.65	
LDL-C	Smoker	42	109.19±36.67	97.76	120.62	0.484
	Former smoker	98	117.48±38.53	109.75	125.20	
	Nonsmoker	62	113.19±39.71	103.10	123.28	
	Total patients	202	114.44±38.47	109.10	119.78	
BMI	Smoker	17	26.72±5.00	24.06	29.38	0.014
	Former smoker	85	30.14±4.65	29.12	31.17	
	Nonsmoker	50	31.11±5.81	29.39	32.84	
	Total patients	152	30.07±5.21	29.21	30.93	

*Data which we could retrieve from the records of the patients. SD=Standard deviation; CI=Confidence interval; BMI=Body mass index; LDL-C=Low-density lipoprotein-cholesterol; HDL-C=High-density lipoprotein-cholesterol

smoking behavior. Moreover, patients who were illiterate or had primary school education <3 years were more frequent in the nonsmoker group than patients who were active and former smokers [Table 1].

Lipid levels

Although patients with TC >200 mg/dL were more in the nonsmoker group, there were no significant differences between smoking behavior of patients who had high and low values of TC ($P = 0.724$). Patients with normal levels of HDL-C and low levels of HDL-C were not different among smoking behavior groups ($P = 0.829$) although we observed that patients who had normal HDL-C (65.3%) levels were more in the former smoker group than other patients [Table 1].

According to LDL-C ≥100 and LDL-C <100 mg/dL and TG ≥150 and <150 mg/dL, no differences with smoking behavior were found ($P = 0.278$, $P = 0.183$; respectively) [Table 1].

Nevertheless, among active smokers, patients with values of TG ≥150 mg/dL (61.9%) and patients with values of HDL-C <40 mg/dL in men and HDL-C <50 mg/dL in women (40.5%) were more frequent than they are in the other groups and

patients with values of TC ≥200 mg/dL were less frequent (35.7%) than they are in the other groups. Patients with LDL-C ≥100 mg/dL (67.3%) were more frequent in the former smoker group [Table 1].

The mean values of lipid profile were not associated with smoking status groups as shown in Tables 2 and 3.

Patients who had at least one of the lipid abnormalities were 94.5% among all the patients, and there was no significant difference between groups ($P = 0.093$).

Body mass index

Among patients with BMI ≥30 kg/m², nonsmokers and former smokers were more frequent than active smokers ($P = 0.006$), while in nonobese patients, active smokers were more frequent ($P = 0.024$), Table 2. Furthermore, in nonsmokers, the mean value of BMI was the highest ($P = 0.014$). Smoker and nonsmoker patients have significant differences in the mean values of BMI [Table 3].

Correlations

BMI levels correlated weakly with levels of TC and TG ($r = 0.181, 0.238$; $P = 0.044, 0.007$). A weak negative correlation was present between packages/year and HDL-C ($r = -0.154$; $P = 0.028$).

Table 3: Comparison of age, lipid profile, and body mass index of patients with different groups of smoking behavior

	Mean±SD (n*)		P	Mean±SD (n*)		P
	Smoker	Nonsmoker + former		Patients with a smoking history	Nonsmoker	
Age (years)	56.17±12.80 (53)	61.98±10.85 (182)	0.001	59.20±11.37 (161)	63.92±11.37 (74)	0.101
Triglycerides	190.15±93.00 (42)	173.53±99.47 (160)	0.159	178.74±100.72 (140)	172.86±93.03 (62)	0.729
Cholesterol	190.33±42.49 (42)	193.04±48.03 (160)	0.740	192.11±45.3 (140)	193.29±50.49 (62)	0.870
HDL-C	43.67±10.18 (42)	44.43±9.84 (160)	0.660	43.3±9.22 (140)	46.26±11.07 (62)	0.056
LDL-C	109.19±36.67 (42)	115.82±38.92 (160)	0.322	114.99±38.04 (140)	113.19±39.71 (62)	0.760
BMI	26.72±5.00 (17)	30.49±5.10 (135)	0.006	29.58±4.85 (102)	31.11±5.8 (50)	0.04

*Data which we could retrieve from the records of the patients. SD=Standard deviation; BMI=Body mass index; LDL-C=Low-density lipoprotein-cholesterol; HDL-C=High-density lipoprotein-cholesterol

Lipid levels correlated with each other significantly as follows:

TG and TC ($r = 0.463$, $P < 0.0001$); TG and LDL ($r = 0.184$, $P = 0.009$); LDL and TC ($r = 0.899$, $P < 0.0001$); LDL and HDL ($r = 0.250$, $P = 0.001$); and HDL and TK ($r = 0.382$; $P < 0.0001$).

DISCUSSION

Sociodemographic factors: Age, gender, and educational status

In this study, we have found that age was associated with smoking status of patients. Nonsmokers group consisted of geriatric patients mostly, where in active smoker group, there were more younger patients [Table 1].

This may be explained as that aging people are more aware about the negative effects of long-term smoking. Thus, younger patients tend to continue smoking while elderly patients are willing to cessate smoking. In the daily medical practice, smoking cessation encouragement by health professionals is especially important in younger people.

In our study, nonsmokers were more among women while active smokers and former smokers were mostly men. Among the geriatric patients who have never smoked, 65% were women. One of the explanations may be that when our patients were younger, smoking was not common among women, so they did not develop the smoking habit. In recent decades, prevalence of smoking in women has been increasing due to tobacco epidemic as described in literature.[15,16] In our country in a review of 31 studies, it was reported that smoking frequency in men was between 27.5% and 63.8% and it was 8.4%–27.8% in women.[17]

Men who continue smoking after the diagnosis of CAD were 20% among all patients. In a recent study evaluating the gender-related differences in controlling risk factors in patients with CAD, men were less frequent nonsmokers and their attempts to cessate smoking were lesser when compared with women.[18,19]

Educational status plays an important role in decision to cessate smoking. Educational level of patients with a history of ACS was found to be inversely associated with active smoking in studies.[20,21] The educational status and smoking history of patients were highly significant in our study. Patients who were illiterate and had primary school education <3 years were more frequent in the nonsmoker group, which consisted mostly of women (65.4%), whereas patients who were graduates of primary school were among active and former smokers, which consisted mostly of men [Table 1].

In another study, in which smoking trends were evaluated at the three different time points, the prevalence of smoking was found to be associated with educational levels of participants and it was lower in higher educated women compared with lower educated women;[22] these results were different from our results. However, if we take education status of our participants as an indicator of socioeconomic status,[15,22] our patients and especially women had lower education and socioeconomic status. Smoking is not a habit adopted by women, so in our patient group, even if the women had lower education status, they were likely to avoid smoking. It has also been reported that tobacco epidemic spreads among women had much later influence than in men.[15]

Smoking behavior after coronary artery disease diagnosis

In our study, we have observed that although patients have been diagnosed with CAD, 22.6% of them were active smokers, and this may be due to their lack of awareness about the consequences of smoking. Although the risks of smoking after an acute cardiac event are known by the patients, it is reported that up to 20% of patients continue to smoke after discharge and are unsuccessful to quit smoking[1] and patients continue smoking after cardiac ischemic event.[19] In a recent study,[11] it was demonstrated that the smoking relapse rate was 59% at 1st year after ACS.

Similarly, about 46% of our patients have quitted smoking after they have been diagnosed with CAD

whom we called former smokers. This may be the result of encouragements about smoking cessation.

Interestingly, active smokers were younger and had less comorbidities such as hypertension (HT) and diabetes mellitus (DM) compared with nonsmokers, and they may have been less alert about cardiovascular prevention. Our patients with HT and DM were more frequent nonsmokers (31.4%), were relatively older, and had a prior diagnosis of CAD compared with younger group.

Both in primary and secondary prevention, smoking cessation is important because smoking affects already established risk factors for vascular diseases such as HT, DM, and dyslipidemias. As a part of the treatment procedure, health professionals should take action in smoking cessation therapies and support patients with vascular diseases in smoking cessation.[23]

Lipid profiles of the patients and smoking behavior

In our study, we have not found any differences between the frequencies of patients with higher values of lipids and with normal values of lipids according to their smoking behavior, but we observed that patients with normal HDL-C and LDL-C values were more in the nonsmoker group than the patients with a smoking history group; in fact, HDL-C values were the highest in nonsmokers and TG mean values were the lowest in nonsmokers compared with smoker group. This finding is compatible with literature.

Gepner *et al*. observed that HDL-C levels increased significantly in patients after smoking cessation for 1 year, especially in women, when compared with active smokers; however, levels of TC, LDL-C, and TG did not change significantly.[24] It is clear that especially, levels of HDL-C increase in patients who do not smoke, and in a recent meta-analysis, it was reported that there was a rise in HDL-C following smoking cessation.[25]

Genetic and environmental factors should also be considered when investigating the association between smoking and lipid profile. In China, in a study population aged over 90 years, smokers had TC levels lower than nonsmokers, but no differences were observed between TG, LDL-C, and HDL-C levels of patients according to smoking status.[26]

In this study, as smoking dosage increased, HDL-C levels decreased. There are reports about smoking dosage and lipid profile. Earlier, it is reported that as smoking dosage increases, cholesterol, TG, VLDL-C, and LDL-C levels increase progressively and HDL-C levels decrease progressively.[7]

Body mass index of the patients and smoking status

In the active smokers group, nonobese patients were more frequently presented than in obese. Furthermore, in nonsmokers group, the mean value of BMI was the highest [Table 2].

In literature, the relationship between BMI and smoking status has been investigated widely. Gepner *et al*. found that when compared with active smokers, former smokers had more weight gain and increase in BMI.[24] After quitting smoking, patients gain weight; however, smoking cessation was found to be associated with decreased cardiovascular disease rate despite weight gain following cessation.[27] One year after myocardial infarction, in patients who attempted to quit smoking, abstainers gained more weight than patients who had relapses.[28]

Clair *et al*. have observed that recent quitters gained more weight than long-term quitters and nonsmokers and smokers regardless of the presence of DM.[27]

In multicenter studies at different times, frequency of smoking and obesity along with the high lipid values was compared in patients with a history of ACS, and it has been reported that the ratio of patients with the presence of mentioned risk factors was still high.[20] Thus, despite the lipid-lowering therapy, goals of recommended lipid values cannot be reached. In fact, nonadherence to medications has been reported to be common in chronic conditions such as HT, hyperlipidemia, and diabetes which contribute to cardiovascular disease.[29]

Preventive cardiology aims to eliminate the continuing risk factors such as smoking, obesity, and dyslipidemia for an effective lifestyle change. Follow-up programs in outpatient clinics for smoking cessation and obesity may motivate these patients.

Limitations of the study

Smoking behavior and disease history was based on self-reports of the patients, so some information may be incorrect. We assumed that patients were adherent to their medication. Since this was a retrospective study on records of patients, some data such as BMI records were missing. Furthermore, physical activity levels which may affect lipid levels and BMI as well as the economic profiles of the patients which may affect attitudes were not present.

CONCLUSION

As health professionals, after patients are diagnosed with CAD, we need to urge patients to make necessary lifestyle modifications such as cessating smoking to prevent acute cardiac events. In our study, we found

that nonsmokers are more among women while active smokers and former smokers are mostly men. Young and male patients are two groups which health professionals need to focus in motivating to quit smoking. We found no significant association between smoking status and lipid profile. Instead, we found a significant association between BMI and smoking status. Active smokers had higher TG and lower HDL values, TG levels were low, and HDL levels were high in nonsmokers. Severity of coronary angiography and smoking status in CAD may be evaluated in larger groups as longitudinal studies.

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Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Choi YJ, Park JS, Kim U, Lee SH, Son JW, Shin DG, *et al.* Changes in smoking behavior and adherence to preventive guidelines among smokers after a heart attack. J Geriatr Cardiol 2013;10:146-50.
2. Takahashi E, Moriyama K, Yamakado M. Status of lipid management using lifestyle modification in Japanese adults: An analysis of the 2009 Japan Society of Ningen Dock database. Intern Med 2013;52:1681-6.
3. Tan XJ, Jiao GP, Ren YJ, Gao XR, Ding Y, Wang XR, *et al.* Relationship between smoking and dyslipidemia in Western Chinese elderly males. J Clin Lab Anal 2008;22:159-63.
4. Wang S, Xu L, Jonas JB, You QS, Wang YX, Yang H, *et al.* Prevalence and associated factors of dyslipidemia in the adult Chinese population. PLoS One 2011;6:e17326.
5. Chelland Campbell S, Moffatt RJ, Stamford BA. Smoking and smoking cessation – The relationship between cardiovascular disease and lipoprotein metabolism: A review. Atherosclerosis 2008;201:225-35.
6. Sotoda Y, Hirooka S, Orita H, Wakabayashi I. Recent knowledge of smoking and peripheral arterial disease in lower extremities. Nihon Eiseigaku Zasshi 2015;70:211-9.
7. Craig WY, Palomaki GE, Haddow JE. Cigarette smoking and serum lipid and lipoprotein concentrations: An analysis of published data. BMJ 1989;298:784-8.
8. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: A meta-analysis. Prev Med 2003;37:283-90.
9. Zengin E, Bickel C, Schnabel RB, Zeller T, Lackner KJ, Rupprecht HJ, *et al.* Risk factors of coronary artery disease in secondary prevention – Results from the AtheroGene – Study. PLoS One 2015;10:e0131434.
10. Plurphanswat N, Rodu B. The association of smoking and demographic characteristics on body mass index and obesity among adults in the U.S 1999-2012. BMC Obes 2014;1:18.
11. Dehghani P, Habib B, Windle SB, Roy N, Old W, Grondin FR, *et al.* Smokers and postcessation weight gain after acute coronary syndrome. J Am Heart Assoc 2017;6. pii: e004785.
12. Notara V, Panagiotakos DB, Kogias Y, Stravopodis P, Antonoulas A, Zombolos S, *et al.* The impact of educational status on 10-year (2004-2014) cardiovascular disease prognosis

and all-cause mortality among acute coronary syndrome patients in the Greek Acute Coronary Syndrome (GREECS) Longitudinal Study. J Prev Med Public Health 2016;49:220-9.
13. Teo KK, Ounpuu S, Hawken S, Pandey MR, Valentin V, Hunt D, *et al.* Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: A case-control study. Lancet 2006;368:647-58.
14. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 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;106:3143-421.
15. Pärna K, Pürjer ML, Ringmets I, Tekkel M. Educational differences in cigarette smoking among adult population in Estonia, 1990-2010: Does the trend fit the model of tobacco epidemic? BMC Public Health 2014;14:709.
16. Jeong YA, Jeong MH, Jeong HC, Ahn Y, Kim YJ, Kim CJ, *et al.* Impact of smoking on clinical outcomes in female patients with acute myocardial infarction. Korean Circ J 2015;45:22-7.
17. Doğanay S, Sözmen K, Kalaça S, Ünal B. How has the prevalence of smoking changed in Turkey? Turkish Journal of Public Health 2012;10:93-115
18. De Smedt D, De Bacquer D, De Sutter J, Dallongeville J, Gevaert S, De Backer G, *et al.* The gender gap in risk factor control: Effects of age and education on the control of cardiovascular risk factors in male and female coronary patients. The EUROASPIRE IV study by the European Society of Cardiology. Int J Cardiol 2016;209:284-90.
19. Vogiatzis I, Tsikrika E, Sachpekidis V, Pittas S, Kotsani A. Factors affecting smoking resumption after acute coronary syndromes. Hellenic J Cardiol 2010;51:294-300.
20. Kotseva K, Wood D, De Backer G, De Bacquer D, Pyörälä K, Keil U, *et al.* Cardiovascular prevention guidelines in daily practice: A comparison of EUROASPIRE I, II, and III surveys in eight European countries. Lancet 2009;373:929-40.
21. Tang L, Patao C, Chuang J, Wong ND. Cardiovascular risk factor control and adherence to recommended lifestyle and medical therapies in persons with coronary heart disease (from the National Health and Nutrition Examination Survey 2007-2010). Am J Cardiol 2013;112:1126-32.
22. Mahley RW, Can S, Ozbayrakçi S, Bersot TP, Tanir S, Palaoğlu KE, *et al.* Modulation of high-density lipoproteins in a population in Istanbul, Turkey, with low levels of high-density lipoproteins. Am J Cardiol 2005;96:547-55.
23. Katsiki N, Papadopoulou SK, Fachantidou AI, Mikhailidis DP. Smoking and vascular risk: Are all forms of smoking harmful to all types of vascular disease? Public Health 2013;127:435-41.
24. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH, *et al.* Effects of smoking and smoking cessation on lipids and lipoproteins: Outcomes from a randomized clinical trial. Am Heart J 2011;161:145-51.
25. Kar D, Gillies C, Zaccardi F, Webb D, Seidu S, Tesfaye S, *et al.* Relationship of cardiometabolic parameters in non-smokers, current smokers, and quitters in diabetes: A systematic review and meta-analysis. Cardiovasc Diabetol 2016;15:158.
26. Yan-Ling Z, Dong-Qing Z, Chang-Quan H, Bi-Rong D. Cigarette smoking and its association with serum lipid/lipoprotein among chinese nonagenarians/centenarians. Lipids Health Dis 2012;11:94.
27. Clair C, Rigotti NA, Porneala B, Fox CS, D'Agostino RB, Pencina MJ, *et al.* Association of smoking cessation and weight change with cardiovascular disease among adults with and

without diabetes. JAMA 2013;309:1014-21.

28. Grandi SM, Filion KB, Gervais A, Joseph L, O'Loughlin J, Paradis G, *et al*. Weight change in patients attempting to quit smoking post-myocardial infarction. Am J Med 2014;127:641-90.

29. Fischer MA, Stedman MR, Lii J, Vogeli C, Shrank WH, Brookhart MA, *et al*. Primary medication non-adherence: Analysis of 195,930 electronic prescriptions. J Gen Intern Med 2010;25:284-90.