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How is lipid profile and morbidity risk in smokers and nonsmokers?

Abstract

Background: Lipid profile changes are a major risk factor for cardiovascular disease with increasing prevalence in our society due to poor nutritional habits, sedentary, and smoking. Owing to lack of epidemiological data in this area, this study was designed to investigate lipid profile and morbidity risk in smokers and non-smokers in Babol, North of Iran.

Methods: In this study, 204 young men over 20 years old, were selected from the City Health Centers in five areas (North, South, East, West and Central) by cluster-sampling method and were referred to Musa- AL -Reza Clinic. After filling the registration form and a questionnaire about their medical history, blood samples were taken in fasting condition. After serum preparation, lipid profile tests were performed using standard spectrophotometric methods in the laboratory of biochemistry. Statistical analysis was done using *t*-test.

Results: Forty three patients (21.1%) were smokers and 161 (78.9%) were nonsmokers. The frequencies of male smokers with total cholesterol index in morbidity range were significantly higher than nonsmokers (39.5% VS. 25.4%, $p < 0.05$). Such circumstances were particularly more pronounced in LDL-C index and the number of male smokers was more both in moderate and high morbidity levels of LDL-C (14% VS. 10.5% and 23.2% VS. 16.8% respectively, $p < 0.05$).

Conclusion: Smokers had impaired lipid profiles and are at increased risk of cardiovascular diseases. Designing and implementing health programs seem to be necessary for changing adverse eating habits and reducing population smoking behavior.

Key words: Lipid profile, Smoking, Risk factor, Cardiovascular diseases, Babol.

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Cigarette contains dangerous chemicals and aggressive free radicals that can cause oxidation of biochemical compounds and vital macromolecules, cellular and tissue damages with many underlying diseases such as cardiovascular and pulmonary diseases, lung, blood and mouth cancers (1,2). There is 0.3 to 3.3 billion chemicals with more than 4000 composition in each milliliter of cigarette smoke, of which 43 types are known as carcinogenic compounds. Based on recent studies, each cigarette smoke contains 10^{15} free radicals in the gas phase and 10^{14} radicals in Bitumen phase that are potentially capable of invading body's biochemical macromolecules, causing lipid, protein and DNA peroxidation, leading to cellular and tissue damages which can be the etiology of many smoking-related diseases and complications (3-5). Smoking directly involves nearly 350,000 men and 80,000 women deaths annually due to early cardiovascular events. WHO has recently introduced smoking as the fourth global health threat (6). Cigarette causes remarkable stress for vessels and reduces coronary blood flow and oxygen delivery to the heart by impairing myocardial perforation. Mechanisms which are involved in increasing the risk of cardiovascular diseases in smokers include increased activity of platelets and leukocytes, destructive effects on lipids, high blood pressure and insulin resistance (7,8).

Reactive Oxygen Species (ROS) cause vascular endothelial damage through various ways including direct cellular damage, indirect effects on lipids and other compounds peroxidation and decreased production of nitric oxide.

Various studies have shown the association between endothelial dysfunction in peripheral artery dilation and atherosclerosis with cigarette smoking (6, 9-11). Mahjoub et al. showed that the amount of some biochemical compounds were different in smokers' and nonsmokers' blood and saliva. The amount of Triglyceride (TG), cholesterol and Low Density Lipoprotein (LDL-C) is also significantly more in acute and stable stages of myocardial infarction while the amount of High density lipoprotein (HDL-C) is less compared to control. Moreover, lipid profile status gradually shifts to normal; from acute to stable phase of the disease, but does not ever reach the normal level. Mahjoub et al. also showed that free radicals produced by different sources can play a major role in destroying erythrocyte membranes, other cells and tissues through peroxidation of biochemical compounds, particularly lipids and proteins (12-14).

Various studies indicated that lipoproteins concentration can be obviously changed in stressful situations such as acute myocardial infarction, surgery and infections. Stressful situations lead to production of acute phase proteins that cause impairment in lipoproteins production (15,16). Increase in lipoproteins and their lipid contents, particularly cholesterol level raise the risk of cardiovascular disease and atherosclerosis (17). Stamler et al., in three cohort studies, showed mortality rate due to cardiovascular diseases (CVD) 34 to 42 percent. They found a strong relationship between mortality rate and high levels of cholesterol; and death from CVD was 2.1 to 2.87 fold in people with high cholesterol level (18).

In a study by Holvoet et al. on 63 patients with acute myocardial infarction and unstable angina through angiography, 35 patients had stable angina having been identified by angiography, 28 patients had heart transplants and coronary artery disease (CAD) and 79 had heart transplant without CAD. It has been recognized that oxidized LDL level was significantly more in patients with CAD compared to those without it (19). In a 10-year period study done by Sharrelt et al. on 12339 patients age ranging from 45 to 64 years, it has become clear that the ideal amount of LDL-C is less than 100 mg/dl both in men and women; and the increase more than 118 mg/dl has been associated

with increased risk of chronic heart disease (CHD) rate (1.42% in men and 1.37% in women). Increase in HDL-C more than 40 mg/dl in men and 51 mg/dl in women; reduce the rate of CHD to 0.64% and 0.69% in men and women respectively. TG is a risk factor for CHD only in women and its increase up to 115 mg/dl can raise CHD risk to 31.1% (20).

Rosengren et al. showed the role of serum cholesterol in the long-term prognosis on 7100 patients with a history of myocardial infarction. They found that the risk of mortality is considerably high in coronary disease with very high levels of cholesterol; and the use of lipid-lowering therapy has been useful in high risk groups (21). In a cross-sectional study by Craig et al. on 180 patients (above 20 years) in the hospital, it was found that screening adults for prevention of CHD could be measured by total cholesterol HDL-C in non-fasting state (22).

Considering the low ages for developing cardiovascular disease, the importance of abnormal lipid profile and smoking in its incidence, particularly in the presence of risk factors together, and lack of information in this area, this study has been done to identify the lipid profile status and morbidity risk in male smokers and nonsmokers in the Babol, northern Iran.

Methods

This cross-sectional descriptive-analytic study was performed in a two-year period (2004-2005). Announcement of this study was done through the Babol Health Department sent to all the City Health Centers. All volunteers over 20 years referred to the place where the study would be implemented; then they entered to the study after filling out the questionnaire containing demographic information, nutrition, smoking and family history of cardiovascular disease and diabetes. Blood sampling was conducted in Musa al-Reza Health Center and biochemical tests in the biochemistry laboratory of Babol University of Medical Sciences. Samples were centrifuged by CLEMENTS 2000 apparatus (Australia) and obtained serum was maintained in eppendorph microtubes in a -20 °C freezer.

All lipid profile-related experiments were performed in enzymatic method using Pars Azmoon kits. All the samples light absorption were read at specified wavelengths by spectrophotometer CECIL 1020 (UK) and considered-compounds concentration was calculated. In samples with

Triglyceride concentrations more than 400 mg dl, LDL-C was measured directly and for less than this amount, it was calculated using the Friedewald formula respectively (23).

$$\text{LDL-C (mg/dl)} = \text{Total Cholesterol} - \text{HDL-C} - [\text{Triglyceride}/5]$$

Normal ranges of lipids and lipoproteins and their morbidity were defined using reliable references. In adults, the ideal level of total cholesterol was determined, less than 200 mg/dl; moderate and high morbidity risks were considered 200-239 mg/dl and 240 mg/dl or more respectively. Triglyceride ideal level was determined less than 200 mg/dl; 200-239 mg/dl and 400 mg/dl or more were considered as moderate and high morbidity risks respectively. Ideal level for LDL-C was determined 130 mg/dl; moderate and high morbidity risks were considered 130-159 mg/dl and 160 mg/dl or more respectively. In addition, the amount of HDL-C was determined in two levels: less than 35 mg/dl (risk-causing) and more than that (normal) (24,25).

The study data were analyzed using the statistical software SPSS T-test for comparing means (p-value less than 0.05 was considered significant).

Results

The study population included 204 men over 20 years, 43 patients (21.1%) smokers and 161 patients (78.9%) nonsmokers. Mean cholesterol level in smokers and nonsmokers groups was 231.9±75.1 and 221.3±58.6 mg/dl respectively. More than fifty-one of the male nonsmokers and 37.2% of the smokers had normal level of cholesterol. There are no significant differences between mean cholesterol values in smokers and nonsmokers, but higher percentage of male smokers were in high morbidity level of cholesterol, which was significant (p<0.05) (Table 1).

Mean TG was 196.23±112.8 in nonsmokers and 202.19±136.5 mg/dl in smokers, which was not statistically significant; but a higher percentage of male smokers were within the range of high morbidity risk levels of TG that was significant (p<0.05) (Table 1). Mean of LDL-C was 126.8±75 and 118.39±62 mg/dl in smoker and nonsmoker groups respectively was not statistically significant; but a higher percentage of male smokers were within moderate and high morbidity risk levels of LDL-C compared to nonsmokers, which was significant (p<0.05) (Table 1).

Mean of HDL-C was 62.8±27.2 in nonsmokers and 66.5±31.4 mg/dl in smokers which was not statistically

significant but a higher percentage of male smokers were within the low morbidity risk levels of HDL-C compared to smokers, which was significant (p<0.05) (Table 1).

Table 1: Data obtained in smoker and non-smoker in that studied subjects

| Subjects | Male non-smokers N(%) | Male smokers N(%) | Pvalue |
|--------------------------|--------------------------|----------------------|--------|
| Total Cholesterol | | | |
| Normal level | 83(51.6) | 16(37.2) | NS |
| Moderate morbidity risk | 37(23) | 10(23.3) | NS |
| High morbidity risk | 41(25.4) | 17(39.5) | p<0.05 |
| Total triglycerid | | | |
| Normal level | 104(64.6) | 26(60.5) | NS |
| Moderate morbidity risk | 46(28.6) | 12(27.9) | NS |
| High morbidity risk | 11(6.8) | 5(11.6) | p<0.05 |
| Total LDL-c | | | |
| Normal level | 11(72.7) | 27(62.8) | Ns |
| Moderate morbidity risk | 17(10.5) | 6(14) | p<0.05 |
| High morbidity risk | 27(16.8) | 10(23.2) | p<0.05 |
| HDL-C | | | |
| Normal level | 154(95.7) | 40(93) | NS |
| Low level | 7(4.3) | 3(7) | p<0.05 |

Discussion

Based on the results of this study, smokers are within a higher level of lipid profile morbidity. This means that they do not only have greater risk for cardiovascular disease because of smoking, but also are in the range of moderate and high morbidity risk with regard to their lipid profile status compared to nonsmokers.

In a similar study done to evaluate cardiovascular risk factors in urban population of Tehran, average total serum cholesterol level was 210 mg/dl in women and 202 mg/dl in men. 23.6 % and 45.4 % of adults had high and normal levels of cholesterol respectively and 31% were in borderline (26). According to a report by National Health Center of America from 1988 to 1994, 17.5 % of men and 20% of the 20-74 age bracket of women had total cholesterol level of more than 240 mg/dl (high morbidity range) (24). In another study, from 1999 to 2000, comparison between cholesterol concentrations of 4148 men and women over 20 years and 15179 men and women over 20 years from 1994 to 1988

showed that the mean concentration of cholesterol has decreased from 205 mg/dl during 1994-1998 to 203 mg/dl during 1999-2000 in America's population (27). In our study, 6.8% of male nonsmokers and 11.6 % of the smokers population were within the range of high morbidity level of triglycerides (400 mg/dl and higher). While based on the results of Tehran Lipid and Glucose Study (TLGS), 5.3% of males and 3.4 % of women had more than 400 mg/dl level of triglyceride (26). Thus, this percentage is higher in our study which could be due to genetic differences and particularly nutritional habits especially in the use of more greasy foods in Babol; although this percentage is not clear in Tehran study with regard to cigarette smoking.

In our study, 72.7 % of male non smokers and 62.8 % of smokers were within normal ranges of LDL-C while in Tehran, the lipid and glucose study was 49.5 % in total population. Furthermore, individuals with moderate and high morbidity risk were fewer in our study compared to those surveyed in Tehran study (26). Prevalence of LDL-C \geq 160 mg/dl (high morbidity risk level) was moderately higher in this study than the values obtained in Canada and India, but lower compared to the amounts reported in Saudi Arabia (28-30). Such differences in lipid profile status could be due to genetic differences and particularly nutritional habits in different populations.

In our study, a higher percentage of people were within the normal ranges of HDL-C compared to Tehran population. Thus, unlike TG status, other indicators of lipid profile are more appropriate in Babol than Tehran (26). Like residents of other developing countries, the Iranian people are facing rapid changes in their lifestyle due to increased access to food and increased consumption of high fatty foods and the reduced physical activity. In the near future, these changes may lead to higher prevalence of disability and deaths caused by CVD (31). According to USPSTF (The US Preventive Services Task Force), doctors are recommended to routinely measure lipoproteins in men over 35 and women over 45 years in order to screen people with lipid abnormalities and treatment of those who are at increased risk of CHD (32). USPSTF also advises physicians to routinely assess lipoproteins in 20-35 year-old men and 20-45 year-old women in diagnosing lipid disorders in people with other risk factors for CHD. The institute does not have specific recommendations about lipoprotein assessment to screen lipid disorders in younger people (20-35 year-old men and 20-45 year-old women) without

presence of other risk factors for CHD (32). USPSTF also recommends HDL-C and total cholesterol measurements to screen for lipid disorders, which can be both done in fasting and non fasting condition. It should be noted however that triglyceride evaluation could not be an appropriate method in screening lipid disorders. Although measuring HDL-C and total cholesterol is more sensitive to assess CHD risk, total cholesterol estimation alone can be used in the screening test if the laboratories can not accurately measure HDL-C. In spite of being useful in evaluation and assessment, LDL-C measurement is costly and requires fasting samples. In patients with screening results showing high risk, lipoproteins analysis including triglycerides and LDL-C measurement (in fasting state) may be necessary and beneficial to select the appropriate treatment (32). Reasonable time for lipids and lipoproteins routine assessment is done every five years (33). On this basis, it is better to evaluate lipids and lipoproteins level in a shorter period of time to people with near-to-high levels and at longer periods for those with frequently low or normal levels. According to our study, a significant percentage of the population studied, particularly the smokers, had impaired lipid profiles and increased risk of cardiovascular disease; this information seems to be necessary to design an appropriate policy for changing poor eating habits and providing public health as well as identifying more patients at critical risks. In addition, an interventional approach is strongly advised to promote health education and health programs.

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