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EFFECTS OF SMOKING ON SERUM LIPID PROFILE IN PATIENTS WITH MYOCARDIAL INFARCTION

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SUMMARY

Several studies have shown that smoking lowers high density lipoprotein levels, resulting in an increased risk of coronary heart disease (CHD). Mortality from coronary heart disease is substantially higher in cigarette smokers than in nonsmokers.

In this study, serum lipid profile was studied prospectively in healthy adults and in A group of patients with acute or old myocardial infarction. Serum lipid levels were measured enzymatically.

Serum HDL cholesterol levels of smoking group was found lower than nonsmoking group with statistical significance (p=0.007).

Key words: Serum lipids, smoking, myocard infarction.

ÖZET

Pek çok çalışmada sigara kullananların düşük HDL-kolesterol düzeylerine sahip olduğu ve bunun koroner kalp hastalığı (KKH) riskinin artmasıyla sonuçlandığı gösterilmiştir. KKH'dan ölümler sigara içenlerde içmeyenlere oranla önemli derecede yüksektir.

Bu çalışmada 78 sağlıklı ve 48 myokard infarktüsü tanısı konmuş hastada serum lipid profilleri saptanmış ve sigara kullanımının serum lipid profili dağılımına etkisi incelenmiştir. Serum lipid düzeyleri enzimatik metodlarla tayin edilmiştir.

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Sigara kullanan grupta HDL kolesterol düzeyleri sigara kullanmayan gruba göre istatistiksel olarak anlamlı derecede düşük bulundu (p=0.007).

INTRODUCTION

Cigarette smoking is believed to cause harmful cardiovascular and atherogenic effects resulting from changes in lipid metabolism (1).

The atherogenic process is influenced by the levels of different plasma lipoproteins, and epidemyologic studies have shown a positive relationship between increased levels of total cholesterol and coronary heart disease (CHD). Cholesterol occurs in serum as a component of lipoproteins; approximately 13% is found in very low density lipoprotein, 70% in low density lipoprotein and 17% in high density lipoprotein: this distribution varies individually (1,2).

RESULT AND DISCUSSION

Comparison of smoking in control and myocardial infarction groups is shown in Table 1 and effects of smoking on serum lipid profile are shown in Table 2.

Table 1: Comparison of smoking of groups

SMOKING / GROUPS	SMOKING	NONSMOKING	TOTAL
CONTROL	n = 42 (% 53.8)	n = 36 (% 46.2)	n = 78 (% 45.1)
MYOCARD INFARCT	n = 37 (% 77.1)	n = 11 (% 22.9)	n = 48 (% 27.7)

N: Number of individuals, ($X^2 = 7.15$, $p<0.05$).

Table 2: Effects of smoking on serum lipid porfile

GROUP	Total cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL-cholesterol (mg/dl)	LDL-cholesterol (mg/dl)
Smoking (n=79)	205.53±41.09	153.16±52.43	35.88±8.89	133.36±44.59
Nonsmoking (n=47)	200.22±47.91	143.36±3.69	41.23±12.63	132.53±41.18
P Value	0.484	0.457	0.007	0.911

n: Number of individuals, Values are mean±SD.

Serum HDL cholesterol levels of smoking group was found lower than nonsmoking group ($p = 0.007$). Whereas serum total and LDL cholesterol and triglyceride levels were not different between smoking and nonsmoking groups ($p>0.05$).

There is evidence that CHD is more strongly related to the distribution of serum cholesterol among the different lipoprotein classes than to total serum cholesterol concentration. An increase in serum low density lipoprotein cholesterol is associated with increased HDL cholesterol levels in serum are associated with decreased risk of atherosclerosis. Recent studies have shown that plasma HDL cholesterol levels tend to lower in smokers than in nonsmokers. The greater risk to smokers of CHD development may result HDL-lowering effect of cigarette smoking (6).

EXPERIMENTAL

Serum lipid profile has been studied in 48 patients (mean age: 53.16±12.02). There was no significant difference between patient with CHD and control subjects in age) with severe coronary vascular disease documented angiography. Angiographic inclusion criteria were: more than 50% stenosis of at least one major coronary vessel due to atherosclerosis and vascular event, defined as myocardial infarction percutaneous transluminal coronary angioplasty or coronary artery bypass grafting. Patients were included irrespective of concomitant risk factors for atherosclerosis such as smoking, arterial hypertension, hyperlipidemia, increased body mass index and diabetes mellitus. There were 36 nonsmoking, 4 diabetic and 6 hypertension patients with CHD. The patients who suffered from hypertension and had a higher cholesterol level than 200 mg/dl were included.

Healthy persons (mean age: 51.95±19.46) without any symptoms for cardiovascular disease were selected for the control groups. None of these individuals had a history of a vascular event.

Blood samples were drawn in plain tubes after the subjects had fasted overnight. The samples were centrifuged for 10 min. at 1500xg at room temperature and serum was removed. Unless immediate analysis, serum was frozen at -20°C.

Serum total cholesterol (TC) levels were measured enzimatically (3). Serum HDL-C was measured following precipitation of apolipoprotein B-containing lipoproteins with phosphotungstic acid and magnesium ions (4). Serum triglyceride levels were measured enzimatically (5). LDL-C concentrations were calculated by using the Friedewald formula (4).

Statistical analyses were made using SPSS version 5.1.

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