Effect of Light and Heavy Smoking on High Density Lipoprotein Cholesterol Level

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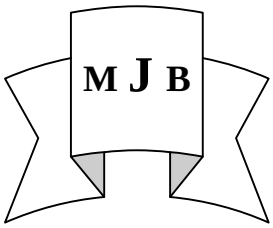

Abstract

Background and purpose: Cigarette smokers have a higher risk of developing several chronic disorders. These include several types of cancer, chronic obstructive pulmonary disease and atherosclerosis which is a chief contributor to the high number of deaths from smoking. Cigarette smoking potentiates the harmful effects of total cholesterol and reduces the cardio-protective properties of high density lipoprotein cholesterol (HDLc), increasing the risk of coronary heart disease(CHD). Decreased serum HDLc is one of the most common lipid disorders in patients with CHD. Existing evidence suggests that every 1 mg/dl decrease in serum HDLc increases the risk of CHD by 2-3%.

HDLc levels are known to be lower in smokers than in nonsmokers. However most studies have not been mentioned whether heavy or light smoking affect HDLc level. The aim of the present study was to evaluate the effect of light and heavy smoking on HDLc level.

Material and method: Sixty apparently healthy participants were included in the study, twenty subjects as control group, twenty light smokers(those who smoke less than ten cigarettes per a day) and twenty heavy smokers (those who smoke ten cigarettes and more per a day).Their mean age ± SD was 35.25±4.38,37.25±4.74,37±3.93 for control, light smokers and heavy smokers groups respectively .The mean of the BMI of the same groups was 23.36±1.7,22.91±1.67 and 23±1.49.HDL-C was measured by colorimetric method.

Results: The mean values of HDLc of control ,light smokers and heavy smokers was 1.43±0.42 mmol/l, 0.75±0.17 mmol/l. and 0.6±0.2 mmol/l groups respectively .There was significant differences in the mean level of HDLc between control group and light smoker and heavy smoker groups($P< 0.05$).However the mean value of HDLc of light smokers was not significantly different from the mean level of heavy smokers($P>0.05$).

Conclusion: Smoking whether light or heavy equally affect HDLc level by deceasing its concentration in the serum and hence increase the risk of CHD.

تأثير التدخين الخفيف والتدخين المفرط على مستوى الكولسترول الحميد

الخلاصة

المدخنين هم الأكثر عرضة للإصابة بالسرطانات على مختلف أنواعها وأمراض الرئة بالإضافة إلى تصلب الشرايين والأخير هو السبب الرئيسي للموت من التدخين.التدخين يؤدي الى زيادة نسبة الكولسترول وتقليل نسبة الكولسترول الحميد وبالتالي يؤدي الى امراض القلب التاجية كون الاخير له صفة الحماية للقلب من إمراض الشرايين.

ان معظم الدراسات تشير إلى أن التدخين يؤدي إلى نقصان مستوى الكولسترول الحميد في حين لم تشير تلك الدراسات إلى أي نوع من التدخين(الخفيف والمفرط) يؤدي إلى ذلك النقصان .

أجريت هذه الدراسة في مدينة بابل في أيلول من العام 2009 لغرض تقييم مستوى الكولسترول الحميد في أمصال 20من المدخنين الذين يدخنون اقل من عشرة سكائر في اليوم الواحد و20من المدخنين الذين يدخنون عشرة سكائر فما فوق بالإضافة إلى 20 شخصا من غير المدخنين كمجموعة مسيطرة .

كان معدل أعمار المجموعات الثلاث 35.25و37.25و37 سنة على التعاقب.كان معدل ا لكولسترول الحميد للمدخنين المفرطين 0.6 مل مول للتر و المدخنين الذين يدخنون اقل من 10 سكائر في اليوم 0.75 مل مول للتر و1.43 مل مول للتر لغير المدخنين.
كان هناك علاقة ذات دلالة عالية(مستوى الاحتمالية اقل من 0.05) بين معدل مستوى الكولسترول الحميد بين غير المدخنين والمدخنين المفرطين والمدخنين الخفيف في حين لم تكن هناك علاقة ذات دلالة عالية(مستوى الاحتمالية أكثر من 0.05) بين المدخنين من كــلا المجموعتين وهذا يدل على أن التدخين الخفيف والمفرط يؤدي إلى انخفاض نسبة الكلسترول الحميد وبشكل متساو.

Introduction

Cigarette smoking are social habits of lifestyle that are closely related to a variety of diseases. Smoking is a major risk factor for atherosclerotic cerebro- and cardiovascular diseases. Various pathophysiological mechanisms, including injury of the vascular endothelium and lipid peroxidation, have been known to be involved in facilitation of the atherosclerotic progression due to smoking [1,2].Smoking affects the blood lipid profile: serum HDL and LDL cholesterol concentrations have been reported to be lower and higher, respectively, in smokers than in non-smokers [3,4] .Thus, smoking may exert its atherogenic effects through blood lipid levels [5,6].

High density lipoprotein (HDL) is a plasma lipoprotein transporting 30.0% phospholipids, 25.0% cholesterol and 5.0% triacylglycerol. Its high density is due to 40.0% proteins. The cholesterol associated with it is unutilized excess cholesterol from tissues. This cholesterol is returned to liver. The liver converts it into bile acids, bile salts, and cholesteryl esters. The two bile salts formed in the liver are sodium glycocholate and sodium taurocholate. These compounds are secreted by liver into bile and through bile they reach the small intestines. The returning of the scavenged cholesterol to liver from peripheral tissues by HDL is known as "Reverse cholesterol transport"[7].

Pre-β-HDL cholesterol; rich in apoprotein A-1 is synthesized by liver and intestinal mucosal cells and released into circulation. Plasma lecithin-cholesterol acyl transferase (LCAT) converts free cholesterol in pre-β-HDL to cholesteryl ester, resulting in the formation of α-HDL cholesterol. LCAT catalyzes the freely reversible reaction which transfers the fatty acid at C2 in lecithin to cholesterol. It is α-HDL which takes part in the reverse cholesterol transport. HDLc inhibits the oxidation of low density lipoprotein cholesterol (LDLc) and reduce the risk of thrombosis by inhibiting platelet aggregation[8].

A higher level of HDLc is an index of safety from the risk of coronary heart Disease (CHD).This is due to the fact that unutilized excess cholesterol which is potentially harmful, is scavenged from the tissues by HDL preventing its deposition and plaque formation[9]. Low plasma HDLc is a risk factor for CHD particularly in the male[10,11]. Experimental studies in laboratory animals and epidemiological studies in population groups have shown that raising the level of HDLc in plasma may retard the development of atherosclerosis[12].

Effect of life style modification on HDLc in plasma are many such as Exercise; walking seems to have no significant effect in increasing HDLc in plasma[13]. Regular aerobic exercises increase HDLc in plasma up to 9.0% in sedentary people[14] . Exercise is believed to increase HDLc by stimulating the production of pre-β-HDLc and increasing reverse cholesterol transport[15]. Cigarette smoking is associated with lowered HDLc and LCAT activity[16,17].After cessation of smoking HDLc is observed to

increase[18].Moderate alcohol intake is good as it raises the plasma HDLc by increasing esterified fraction of cholesterol in plasma[19]. Both LDLc and HDLc in plasma are reduced with low intake of fat. A fat rich in polyunsaturated fatty acids has a beneficial effect in raising plasma HDLc[20].

Medication such as Niacin, Fibrates and Statins have been found to increase HDLc in plasma. These medications also lower LDLc and TG in plasma. Niacin is most effective in raising plasma HDLc. However, the side effects of niacin like cutaneous flushing and dyspepsia have to be taken care of. Fibrate therapy has resulted in an increase of plasma HDLc by 10-25%[21].Gemfibrozil has significantly reduced the risk of coronary heart disease. Statins (Simvastatin,Atorvastatin) have been found to elevate plasma HDLc by raising apolipoprotein A-1 synthesis[22].

Aim of the Study

HDLc levels are known to be lower in smokers than in nonsmokers. However most studies have not been mentioned whether heavy or light smoking affect HDLc level. The aim of the present study was to evaluate the effect of light and heavy smoking on HDLc level.

Material and Methods

The study was conducted in Babylon city during September 2009. The study included 60 apparently healthy male subjects who are similar in terms of age and physical activity. Twenty heavy smokers(those who smoke 10 cigarettes and more per a day). Twenty light smokers(those who smoke less than 10 cigarettes per a day). Another 20 subject who are non smokers included in this study as a control group. The duration of smoking of the first two group was between five and ten years.

All subjects were required to meet certain criteria. None of the subjects drank alcohol or taking medication. None of them were obese (<25kg/m^2).All subjects fasted for 14-16 hours before their blood was drawn to measure the serum HDL. Body weight and height were measured and the BMI was calculated as weight/height^2 (kg/m^2).

HDL-C concentrations were measured by enzymatic techniques in the biochemistry laboratory of the medical college. HDLc levels were measured after precipitation with phoshotungstic acid and magnesium. Kits for the HDLc assays were from Biolabo (France).

Statistical analysis: SPSS (version 10) program was used for statistical analysis. The results were represented through mean and standard deviation. F-test was used to compare between means & to study the significance of the difference. P value < 0.05 was considered to be statistically significant.

Results

A total of sixty apparently healthy male subjects were studied, twenty heavy smokers,twenty light smokers in addition to twenty non smokers were enrolled for comparative assessment. Their mean age ±SD was 37±3.93 ,37.25±4.74,35.25±4.38.(table 1).The BMI of the same groups was23±1.49 ,22.91±1.67 and 23.36±1.7 respectively.(table1)

Table 1 Numbers ,age and body mass index in heavy smokers, light smokers and non smokers groups

Subject characteristics	Heavy smokers	Light smokers	Non smokers
Number	20	20	20
Age(mean±SD)years	37±3.93	37.25±4.74	35.25±4.38
BMI(mean±SD)kg/m^2	23±1.49	22.91±1.67	23.36±1.7

There was no significant differences in terms of age and BMI between all three groups(P value >0.05).

The mean value of HDLc of non smokers was 1.43±0.42 mmol/l which was significantly different(P value <0.05) from mean value of HDLc of heavy smokers(0.6±0.2 mmol/l) and light smokers group(0.75±0.17 mmol/l.).However ,there was no significant differences in the mean value of HDLc between light and heavy smoker groups(P value >0.05).(table2)

Table 2 Mean value of serum level of HDLc of non smokers, light smokers and heavy smokers groups

Group	Mean value of HDLc ±SD(mmol/l)
Non smokers	1.43±0.42
Light smokers	0.75±0.17
Heavy smokers	0.6±0.2

Discussion

It is well known that HDLc is inversely correlated with the risk of atherosclerosis and coronary heart disease .Similarly, it is well known that smoking affect HDLc by deceasing its level in the serum, therefore this study was carried out to shade a light on which type of smoking heavy or light decrease HDLc level.

The protective effect of HDL has been related to their role in the cholesterol reverse transport (from peripheral tissues to the liver) and to their ability to inhibit oxidation of low-density lipoproteins (LDL) and biological membranes [8,9]. Smoking affects the blood lipid profile: serum HDL and LDL cholesterol concentrations have been reported to be lower and higher, respectively, in smokers than in non-smokers .Thus, smoking may exert its atherogenic effects through blood lipid levels [3-6].

It has been reported that the safest level of HDLc to be protective against CHD is more than 1.17 mmol/l[7,9,10,11]. In the present study, the mean value of HDLc of control group was 1.43mmol/l.

Furthermore,in the present study HDLc was low in smokers compared with control group and it is identical with most of the studies[4,17,18]. However its level was low in both groups of smokers whether light or heavy.

Conclusion

Smoking whether light or heavy equally affect HDLc level by deceasing its concentration in the serum and hence increase the risk of CHD.

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