Evaluation of lipid profile & BMI in smokers& non-smokers individuals

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الخلاصة

اجريت الدراسة الحالية لتقييم مستويات الدهون والتي تتضمن(المستوى الكلي للكوليستول ومستوى الكوليسترول عالي الكثافة ومستوى الكوليسترول واطئ الكثافة في بلازما الدم المأخوذ من الأشخاص الأصحاء المدخنين وغير المدخنين لتحديد تأثير التدخين على مقياس كتلة الجسم (الوزن). اجريت الدراسة على (105) شخص مدخن و قسموا حسب الوزن إلى ثلاثة مجاميع وهي(وزن طبيعي و وزن فوق الطبيعي ومفرطي السمنه) و (55) شخص غير مدخنين قسموا بنفس الطريقة. استخدمت عدة تشخيصية كاملة لقياس مستوى الدهون. أوضحت نتائج الدراسة وجود فرق معنوي ($P<0.01$) حسب مقياس كتلة الجسم للمدخنين بين المجاميع الوزنية الثلاث ، حيث أظهرت النتائج وجود ارتفاع معنوي في مستوى الكلي للكوليسترول ومستوى الكوليسترول منخفض الكثافة للأشخاص الذين لديهم سمنة مفرطة أكثر من الأشخاص الذين لديهم زيادة في الوزن مقارنتاً بالأشخاص طبيعي الوزن بينما بينت النتائج وجود انخفاض معنوي في مستوى الكوليسترول عالي الكثافة للأشخاص الذين لديهم سمنة مفرطة أكثر من الأشخاص الذين لديهم زيادة في الوزن مقارنتاً بالأشخاص طبيعي الوزن. كما أظهرت النتائج وجود فروقات معنوية ($P<0.01$) بين الأشخاص المدخنين وغير المدخنين .

Abstract

This work was done to evaluate lipid profile (total cholesterol, HDL-cholestrol, LDL-cholestrol) in plasma of healthy smokers /non-smokers and determined the effect of body mass index (BMI) on these two groups. Serum levels of evaluate lipid profile (total cholesterol, HDL-cholestrol, LDL-cholestrol) were measured in (105) smokers subjects grouped in three types (normal weight, over weight and obese) and (55) non-smoker subjects grouped in the same way. Lipid profile were estimated by using the enzymatic colorimetric methods for serum in both smoker and non-smoker subjects.

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The result revealed that BMI for smoker subjects show significant differences($P<0.01$) for three groups (normal weight, over weight and obese) in the total cholesterol levels in serum and LDL-cholestrol were increased significantly in smoker obese subjects more than in smoker over weight subjects and as compared with smokers normal weight; while the serum levels of HDL-cholestrol were decreased significantly in smoker obese subjects more than in smoker over weight subjects as compared with smokers normal weight, also the results showed that there were significant differences between smokers and non- smoker subjects ($P<0.01$) in these three types of groups.

Introduction

Smoking is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease and which is recognized as a major risk factor for the development of ischemic heart disease which may lead to alter the normal plasma lipoprotein pattern and incidence of developing cardiac heart diseases (CHD) which is directly related to the number of cigarette smoked , sudden death is 2-4 times more in heavy smokers than in non smokers[(1)].

Lipid profile is a battery of laboratory studies to help determine the risk factors in coronary artery disease and a lipid profile usually includes total cholesterol, LDL- cholesterol (low-density lipoprotein) which also is called ("bad" cholesterol) and HDL -cholesterol (high-density lipoprotein) which also is called ("good" cholesterol) because it serves a protective function, it sucks up cholesterol found in the bloodstream and returns it to the liver for disposal while LDL - cholesterol carries cholesterol from the liver to the cells that need it so high levels of HDL are desired so that any free cholesterol will be quickly removed from the blood and Proper physical exercise is one way to increase the concentration of HDL- cholesterol [(2,3)] .

Lipoproteins are macromolecules which composed of hydrophobic lipids bound to protein that transport lipids through aqueous plasma (i.e.) Lipoproteins are spherical or discoid

aggregates of lipids and Apo proteins. which are classified into five groups in order of decreasing size and increasing density, these are: chylomicrons, VLDLs (very-low-density lipoproteins), LDLs (low density lipoproteins) and HDLs (high-density lipoproteins). Blood lipid tests should not be performed during stress or acute illness (e.g.) recent myocardial infarction [MI], stroke, pregnancy, trauma and use of certain drugs [4,5].

The amount of body fat is difficult to measure directly, and is usually determined from an indirect measure, the body mass index (BMI) which has been shown to correlate with the amount of body fat in most individuals (notable exceptions are athletes who have large amount of lean muscle mass). The BMI gives a measure of relative weight, adjusted for high, this allows comparisons both within and between populations. The BMI is calculated in both men and women as follows: BMI= (weight in kg) / (high in meters). The healthy range for the BMI is between 19.5 and 25 kg/m² while individuals with BMI ≥25 kg/m² are considered over weight and those with a BMI ≥30 kg/m² are defined as obese [6,7]. Smoking is an important cause of high cholesterol levels and heart disease so smoking affects cholesterol levels by dramatically raising the amount of LDL- cholesterol and decreasing the amount of HDL- cholesterol also increases the fatty lipid levels in the blood stream and accelerates the process of atherosclerosis [4]. Smoking was related to lower HDL-cholesterol (p=0.037) serum level while Total cholesterol, LDL-cholesterol, and the TC/HDL-cholesterol ratio were strongly related with the level of smoking (p=0.006, p=0.008, and p=0.006 respectively). Variant analysis of lipid values stratified by smoking category showed significantly higher triglyceride and lower HDL-cholesterol levels compared with ex- or non-smokers . Although the mean values of total cholesterol, LDL cholesterol, and the TC/HDL ratio were comparable between smokers and ex- or non-smokers, these markers were strongly correlated with the level of smoking[8].

Materials and Methods

This study was conducted in the Diwanyia Teaching Hospital from October 2010 to April 2011. The subjects were chosen in this study were evaluated by a self administrated questionnaire form in semi-structure individual interviews, the participants were matched for body mass index &age.

The subjects divided into two groups (105) healthy smokers & (55) healthy non- smokers (individual who had never smoking) were recruited as control. Only males were included in this study ,as a smoker.

The ages of subjects were (20- 60) years have been examined .The smokers subjects sub classified into three subgroup (35) subjects with normal weight (BMI = 19.5 and 25 kg/m² while (35) individuals with BMI ≥25 kg/m² are considered over weight and (35) individuals with a BMI ≥30 kg/m² are defined as obese . (3.5ml.) of overnight fasting blood samples were taken by venipuncture in tubes containing EDTA tube for lipid analyses. Plasma was obtained by centrifugation for 10 minutes at 4000 rpm &the supernatant plasma was kept at 4 C^0.The estimation of lipid profile are measured to fasting subjects for 12 hour . Plasma total cholesterol concentration , HDL –cholesterol & LDL- cholesterol were measured by enzymes colorimetric methods (Biomerieux kit, France).

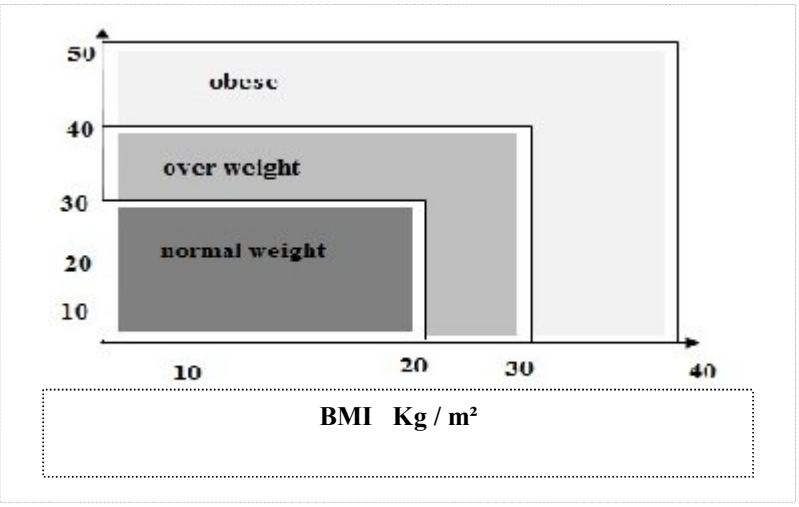
Statistical analysis: The data were collected and analyzed by using SPSS(statistical package for the social sciences) , statistical significance considered when P-value <0.01.

Results and discussion : Combination of Results and discussion

Table(1) Relationship between BMI and Age in smoker male(note : that the smokers smokes same No. of cigarettes daily > 30).

NO. investigation	Normal Weight BMI < 25	Over Weight BMI ≥25 kg/m²	Obese BMI >30 kg/m²
	35	35	35
Age years	27.9 ± 2.1	39.4 ± 3.4	52.3 ± 9
BMI kg/m²	21.3 ± 1.2	29.1 ± 1.4	38.4 ± 3.1

The table show a significant differences between the mean and stander deviation P< 0.01 for the three groups .

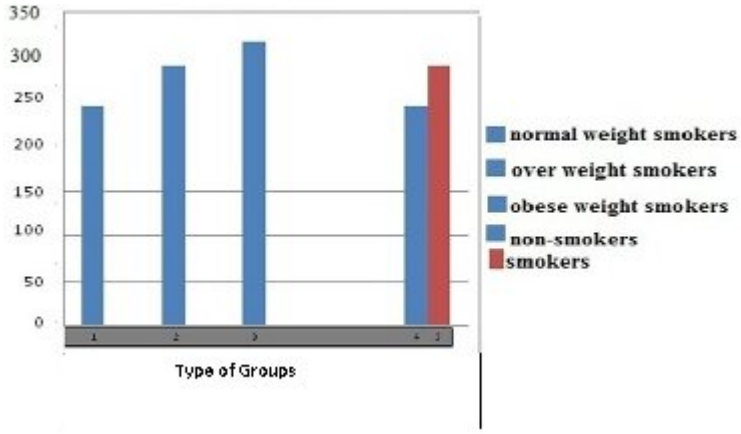


Figure(1):Relationship between BMI and Age in smoker male (note : that the smokers smokes same No. of cigarettes daily > 30)

Table(2):The mean &SD of lipid profile concentration in three groups of smoker .(note : that the smokers smokes same No. of cigarettes daily > 30)

	Normal Weight	Over Weight	Obese
T-cholesterol Mg/100m	245 ± 5	288.8 ± 10	317 ± 12
LDL- cholesterol Mg/100m	130.3 ± 3.5	198.8 ± 11.5	224 ± 10.4
HDL- cholesterol Mg/100m	48.5 ± 1.4	33.4 ± 1.5	25.6 ± 1.3

The table show a significant differences in the results between the three groups were significant P< 0.01.

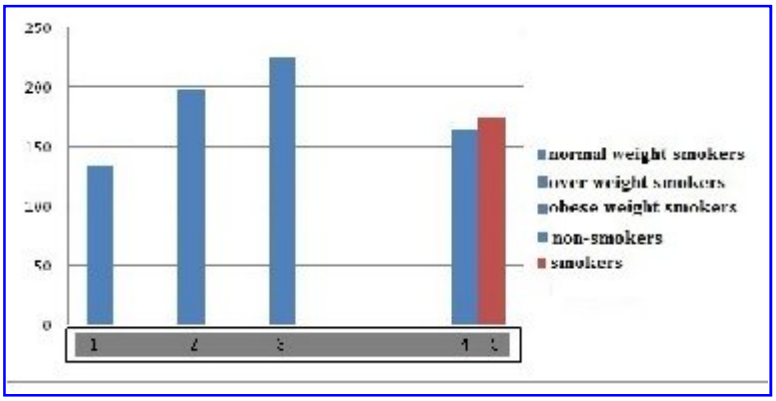


Figure(2): Relationship between T-cholesterol of smokers (three groups) &non-smokers

Table(3):The mean &SD of lipid profile concentration in three groups of non-smoker

	Normal Weight	Over Weight	Obese
T-cholesterol Mg/100m	224 ± 1.5	265.3 ± 8	292 ± 9.5
LDL- cholesterol Mg/100m	112 ± 1.8	169 ± 8	201 ± 8.5
LDL- cholesterol Mg/100m	50.5 ± 1.4	44.3 ± 1.5	38.6 ± 1.5

The table show that the differences between the three groups were significant P< 0.01 .



Figure(3): Relationship between LDL of all groups smokers and non-smokers, T-cholesterol of smokers (three groups) &non-smokers

Table (4):Relationship between T-cholesterol , LDL & HDL in smokers & non-smokers male .

	smokers	Non-smokers
NO. Investigated	105	55
T-cholesterol Mg/100m	281.5 ± 9.5	245.3 ± 6.25
LDL- cholesterol Mg/100m	185.235 ± 8.5	170.15 ± 4.5
HDL- cholesterol Mg/100m	35.5 ± 1.4	44.34 ± 1.4

There were a significant differences between the smokers and non-smokers male P< 0.01.

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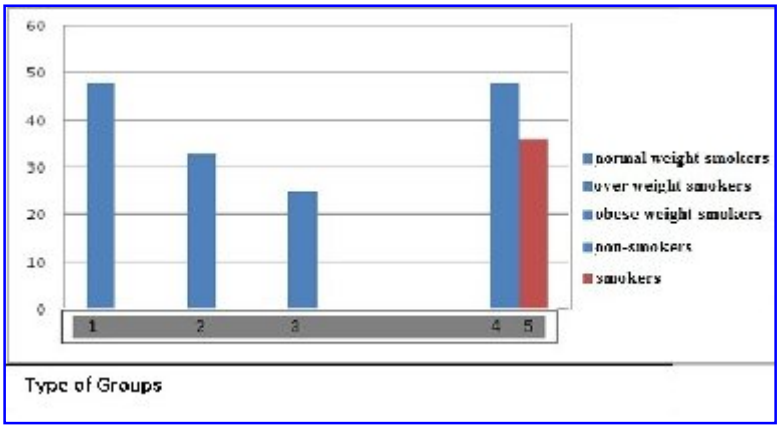

Figures(4):Relationship between T-cholesterol , LDL & HDL in smokers & non-smokers male

Discussion

It has been reported that incidence of coronary heart disease is directly related to number of cigarettes smoked & nicotine has a considerable influence on increasing the lipid levels in blood [(1)].

Many studies have shown that smoking introduces many toxic substances like nicotine, tar, and free radicals into the blood stream, free radicals combine with LDL cholesterol particles causing them to more easily burrow into the artery-cell walls clogging the arteries and hastening the atherosclerotic process, The remaining toxic substances don't belong in the blood stream and most likely have a bad effect on the arterial system so since HDL levels are depressed in smokers, cholesterol is free to build up in the arteries so smoking starts a vicious cycle preventing a person from doing the kinds of activities that will help increase their HDL levels [(10)].

Lipoprotein(a) concentrations in serum were not correlated with other well-recognized risk factors for early myocardial infarction such as Apo lipoproteins A and B, LDL cholesterol, and HDL cholesterol. Lp(a) concentrations were not influenced by age[(11)].

The habit of smoking cigarettes exerts various influences on lipid metabolism. It is well proved that smokers show lower concentrations of HDL- cholesterol but have higher concentration LDL- cholesterol. The groups of all smokers, light smokers, and heavy smokers, were compared with the nonsmokers groups, there were no significant differences between the mean concentrations of Lp(a) in each group[(12)] .

The serum total cholesterol and LDL-C were significantly higher in smokers ($p < 0.05$) whereas serum HDL-C was significantly lower ($P < 0.05$) in the fed state ,the total serum cholesterol level was increased by 10.4 mg/dL in smokers whereas the increase was 4.8 mg/dL in nonsmokers. There was less rise of HDL-C(1.9 mg/dL) in smokers as compared to that in nonsmokers (3.4 mg/dL) and in LDL-cholesterol (1.8 mg/dL) in smokers compared to nonsmokers (3.4 mg/dL) in fed state[(13)].

A total of children were cigarette smokers. Age, height, and body mass index (BMI) were not significantly different between

the children with and those without family history(FH). Children with FH had severely increased LDL-C and decreased HDL-C levels compared with non affected siblings . LDL-C highly significant increasing(P<0.001)but HDL-C highly significant decreasing (P<0.001)[14] .

We found no significant difference in mean serum cholesterol and LDL-C while there were significant difference in mean serum HDL-C according to the BMI[15].

Plasma levels of fasting cholesterol, lipoprotein cholesterol and malondialdehyde were estimated. In smokers the levels of total cholesterol and LDL cholesterol were significantly elevated when compared with the controls[16]. Cigarette smoking is associated with increases in plasma triglycerides and decreases in plasma high density-lipoprotein-cholesterol concentration. These changes not only increase risk of coronary heart disease, smokers in particular had decreased α-tocopherol levels when compared with nonsmokers. Smokers had also lower HDL cholesterol (HDL-C) contents, but this difference was statistically significant only for males[17].

BMI and smoking were highly significant related to concentration of LDL (p=0.002), total cholesterol / HDL (p=0.001) and inversely related to concentration of HDL (p<0.01)(13).

Cigarette smoking and elevated levels of serum total cholesterol are the second and third most common causes of death in the world, respectively, largely due to their major etiologic role in CHD and ischemic stroke, the risk for CHD events in smokers compared with nonsmokers increased significantly by 16% for every 1.06 mmol/l (40.99 mg/dl) increase in total cholesterol and by 39% for every 0.40 mmol/l (15.47 mg/dl) decrease in HDL cholesterol. However, this varied considerably by gender and region[18].

The smokers had no significant between total cholesterol, and LDL cholesterol compared with non-smokers[19]. In smokers the levels of total cholesterol and LDL- cholesterol were significantly elevated when compared with non smokers while

HDL- cholesterol is not significantly different between the two groups[20].

The mean value of HDL- cholesterol are significantly lower in smokers than in non- smokers[21].

Serum TC, LDL-C, and VLDL, were significantly higher in smokers ($p < 0.05$) as compared to non-smokers serum HDL-C was significantly lower in smokers ($p < 0.01$) as compared to non-smokers[22].

Also smoking introduces many toxic substances like nicotine, tar &free radicals into the blood stream, free radicals combine with LDL-C particles causing them to more easily burrow into the artery cell walls clogging the arteries &hastening the atherosclerotic process, these changes may contribute to the atherogenic potential of cigarette smoking also free radicals present in cigarette smoke promote the oxidation of proteins and lipids such as the methionine residue at the reactive site of antitrypsin is highly oxidized in smokers, which greatly decreases the protease inhibitor activity of antitrypsin [23].

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