A COMPARATIVE STUDY OF LIPID PROFILE AND BODY MASS INDEX IN CORONARY HEART DISEASE PATIENTS AND HEALTHY SMOKERS SUDANESE SUBJECTS

Salah A. M. Ali
Dept. of Biotechnology, faculty of Science and Technology, Omdurman Islamic University **SUDAN**
Salahelgafary@gmail.com

Awatif ME Omran
Dept. of Biochemistrty, College of Science ,Tabouk University **SUADI ARABIA**
Awatify2006@gmail.com

Fatima A. B. Abdalla
Dept. of Biochemistrty, faculty of Medicine , Omdurman Islamic University **SUDAN**

ABSTRACT

The present study was carried out at the department of biotechnology-Omdurman Islamic University with cooperation with department of Clinical chemistry- Sudan Centre for Heart Diseases as a comparative study of Lipid Profile and Body Mass Index (BMI) in Coronary Heart Disease (CHD) Patients (N=50) and healthy smokers (N=50). From the results, when compared CHD Patients and smokers it found that, there is no significant difference between the two groups in lipid profile levels (Triglycerides (TG) (141.98+9 28.77 mg/dl) , (153.34+53.27 mg/dl) respectively and High Density lipoprotein (HDL) (46.00+6.37 mg/dl) , (47.98+17.73 mg/dl) respectively, Cholesterol (TC) (242.14+51.7 mg/dl) , (237.26+44.37 mg/dl) , Low Density lipoprotein (LDL) (168.66+56.64 mg/dl) , (157.82+43.8 mg/dl) respectively) (P.value >0.05) , but there was decrease in lipid profile levels in smokers when compared to CHD patients . On the other hand, the BMI in CHD patients was lower than Smokers ,with a significant difference between the two groups (24.9+2.4 kg/m^2) and (26.4+2.96 kg/m^2) respectively)(p.value <0.05). The results showed that, cigarettes smoking influences the normal Lipid Profile levels and BMI , hence causing dyslipidemia that may lead to atherosclerosis and increase the risk of CHD in smokers.

Keywords: Lipid Profile, Body Mass Index, Coronary Heart Disease, Smokers.

INTRODUCTION

Coronary heart disease (CHD) has been estimated to become the leading cause of death in developing countries by 2020. WHO.(1999) . More than 80% of global CHD burden occurs in low income countries but the knowledge regarding important risk factors is derived largely from developed countries. Obesity is defined as having an excess of body weight. A world Health Organization (WHO) release defined obesity as a chronic disease increasing globally replacing traditional health concerns Javed *et al* (2014). It is directly related to cardiovascular problems and children whose parents are cardiovascular patients tend to have higher weight in the childhood and develop obesity as adults Klatzkin *et al*(2015).

There is geographic variability in the prevalence of CHD risk factors. Yusuf *et al* (2004) and it is important to use the local data when discussing the relation between a disease and its risk factors. It is still unclear to what extent the CHD risk factors known in the western civilizations are applicable in Pakistan. One possible reason is the high prevalence of CHD risk factors like hypertension, obesity and dyslipidemia in Pakistan. Awan ZA *et al* (2011). Considering obesity as

CHD risk factor, it has been shown that increased abdominal obesity, insulin resistance and type 2 diabetes (T2DM) are more prevalent in South Asians and are important CHD risk factors .Raji A *et al* (2001). As CHD can be a sequel of obesity, the biochemical pathways involved in the development of obesity have also been investigated to play a role in the development of CHD. Flegal KM, *et al* (2008).

Blood lipid levels are modifiable risk factors for atherosclerosis and CHD. Being hydrophobic in nature, cholesterol, cholesterol esters, triglycerides and phospholipids are transported to the other tissues in the form of lipoproteins. Major classes of lipoproteins are chylomicrons (CM), low density lipoproteins (LDL) and high density lipoproteins (HDL), named by the site of their assembly and type of lipid and apo protein they have. Chen J, *et al* (2009).

Excess fatty acids (FA) in the liver are converted into triacyglycerols which along with phospholipids, free and esterified cholesterol are packaged into very low density lipoprotein (VLDL) along with a variety of apo proteins.

While travelling through the peripheral tissues, triacyglycerol content is hydrolysed with the help of lipoprotein lipase (LPL) into FA and VLDL remnants. Maxfield FR, *et al* (2005). VLDL remnants through further hydrolysis of triglyceride contents give rise to intermediate density lipoproteins (IDL) and LDL. LDL having apoB100 apo protein component is the major cholesterol carrier in peripheral circulation. Packard C. *et al* (2003). Elevated plasma levels of these non HDL lipoproteins are major CHD risk factors. Cohen JC , *et al* (2006).

Smoking is one of the leading causes of preventable death globally. In the United States, about 500,000 deaths per year are attributed to smoking-related diseases and a recent study estimated that as much as 1/3 of Chinese male population will have significantly shortened life spans due to smoking. Male and female smokers lose an average of 13.2 and 14.5 years of life, respectively. At least half of all lifelong smokers die earlier as a result of smoking.(Sander *et al*.,2009, Matthew Hilton.,2006)

There are many ingredients in tobacco smoke that cause many diseases in the body including, infection, cancers and heart disease. Metha *et al.,* (2008) and Zhong *etal*(2008) and every 6 minutes one person die In the world due to smoking Mathers CD and loncar 2006. Cigarette contains over 4,000 chemicals, 200 of them are poisons ,contains over 80 cause cancer include (CO, free radicals, nicotine, and tar) Abel *et al*(2005) and Carel *et al*(1985).

Most dangerous component are tar and Carbon monoxide. Tar contains chemicals carcinogens which deposits in lung. Also smoke produce CO which binds to Hb retard carrying of oxygen and lead to hypoxia , lung cancer, kidney cancer, heart disease and stroke .Payam *et al*(2005) . Cigarette smoke affects many organs. The liver is one of those Organs that might be affected by smoking despite the fact that there is no direct contact between liver and smoking Sanger *et al.,* (2008). Liver is vital organ of vertebrates play many roles in the body such as: metabolism with numerous function including: regulation of glycogen storage, de composition of RBCS, plasma protein synthesis, hormone production and eliminate toxins from the body .To assess the liver function there are many tests which can be conducted inside clinical laboratory. Some of these tests are, serum total protein, albumin, Alanine Aminotransferase (ALT), Aspartate

Aminotransferase (AST) and bilirubin Kalaed *et al.*,(2014).The determination functions of liver must be done carefully and precise because these parameters can affected by external factor such as: environmental factor . Some recent studies were conducted on effect of cigarette smoking on albumin, liver enzymes (AST, ALT and ALP), and bilirubin in males' smoker.

Many studies showed that effect of cigarette smoked in LFT caused significant increase in (AST, ALT and ALP)also significant decrease in serum albumin and serum total bilirubin Nathwani *et al.*,(2005) According to the American Heart Association about 22% of adults with diabetes smoke, even though U.S. research indicates that the most harmful effect of smoking is linked to a significantly higher risk of developing Type 2 diabetes. In fact, the University of Lausanne (Lausanne, Switzerland) analyzed several studies involving more than one million patients and discovered that one of the risks of smoking is a 44% higher chance of developing Type 2 diabetes compared with non-smokers. In addition, the risk increases with the average number of cigarettes smoked daily. Smoking has been identified as a possible risk factor for insulin resistance (a precursor for diabetes). Also been shown to deteriorate glucose and lipid metabolism which may lead to the onset of type 2 diabetes.(Fagard,2009) There is also some evidence which suggests that smoking increases diabetes risk through a body mass index independent mechanism.(Cullen *etal*., 2009, Nagaya.,2008)

Numerous cross-sectional studies indicate that body weight, or body mass index (BMI; in kg/m2), is lower in cigarette smokers than in nonsmokers. In the World Health Organization Monitoring Cardiac Disease surveys, BMI was lower in smokers than in nonsmokers in 20 (men) and 30 (women) of the 42 populations, and there was no population in which smokers had a higher BMI than did nonsmokers.(Molarius *etal*.,1997)

Smoking's effect on body weight could lead to weight loss by increasing the metabolic rate, decreasing metabolic efficiency, or decreasing caloric absorption (reduction in appetite), all of which are associated with tobacco use. The metabolic effect of smoking could explain the lower body weight found in smokers.(Dallosso *etal*.,1984).

Few studies have evaluated the chronic metabolic effects of smoking, and the results have conflicted. After 30 day of smoking cessation, the resting metabolic rate in female quitters was shown to be 16% lower than it had been when they were smoking, and an increase in body weight was attributable to a decrease in resting metabolic rate and an increase in caloric intake.(Moffatt RJ.,1991)

Physical activity increases metabolic rate and may help to control body weight, but smokers tend to be less physically active than nonsmokers.(Klesges RC *etal*.,1990)

It has been observed that many lipid/lipoprotein abnormalities are prevalent in obesity and heart problems, collectively termed as dyslipidemia, however, these dyslipidemias are often hyperlipidemia where in majority of lipids are shifted towards the upper limits of range or higher than the range. Owing to the recent modernization of the lifestyle and availability of transportation means combined with a unique ethnicity have resulted in the high prevalence of metabolic disorders in Sudan like the rest of the world. This not only affects the daily activities, work performance and social interactions but also poses a huge burden on healthcare.

Keeping in view the importance of lipid traits in the development of nutritional disorders, the current study was conducted to investigate the lipid profile patterns in the smokers and CHD patients in Sudanese subjects.

METHODOLOGY

Design of the study:

This study was conducted in the department of clinical chemistry -Sudan centre for heart diseases in 2017. Among 50 Sudanese male voluntary heavy cigarette smokers 50(non-diabetics) and 50 Coronary heart disease male patients (non-smokers and non-diabetics) their age between 20-80 years.

Two groups were collected with the same range of age for statistical comparison.
The clinical data, medical history and other relevant information were collected from subjects by personal interview through the designed study questionnaire.

Samples and data collection

Under a septic condition, about 5 ml of venous blood were collected from each volunteer by vein puncture (after overnight fasting 10-12 hours) and were divided into two, one placed in fluoride oxalate and the other in heparinized containers, blood was centrifuged at 3000 rpm for 10 minutes to settle all the formed elements and separate serum which kept in epindorfe tubes in refrigerator at 2-8 c^0until further analysis.

Lipid Profile Measurement

Plasma total cholesterol, HDL cholesterol and triglycerides (TG) were estimated in all subjects by kit methods on spectrophotometer. (Biosystems.,2011) Commercially available test kits, products of BioMed Biotechnologies Egyptian were used and with the manufacturer's instructions strictly adhered to. Plasma LDL Cholesterol was calculated from the results of lipid profiles by using the following formula.

$$\text{LDL Cholesterol(mg/dl)} = \text{Total Cholesterol} - \frac{\text{Triglycerides}}{5} - \text{HDL Cholesterol}$$

Body Mass Index Measurement

The height and weight of each participant were measured in order to calculate BMI. BMI was calculated as body weight in kilograms divided by the square of height in meters (kg/m^2). Following the National Institutes of Health in Clinical Guidelines .Flegal KM,*et al*(2008) .

Statistical analysis

All data were analyzed by using statistical analysis available software (SPSS) program version 20 Unpaired t-test were applied to test the significance of variance ($p<0.05$) of the parameters under study between the two groups.

RESULTS

The present study comprises lipid profile and BMI of 50 CHD patients (100% male) with 50 healthy smokers (100% male). Their Age range between 20-80 year. This study aims to compare the lipid profile and BMI of both CHD patients and smokers.

Data was collected in pre-designed validated Performa and results were tabulated in Table 1 to 3 and the following Figure .

Table (1) Shows the Descriptive Statistics of Coronary heart disease patients

	N	Minimum	Maximum	Mean	Std. Deviation
Age /year	50	22	78	41.96	16.778
Weight /kg	50	53	95	77.28	10.321
Height cm	50	143	190	175.06	10.076
Body Mass Index kg/m2	50	18.3	29.3	24.932	2.4226
Triglycerids mg/dl	50	44	300	153.34	53.26
Total Cholesterol mg/dl	50	176	403	242.14	51.696
HDL Cholesterol mg/dl	50	30	54	46.00	6.373
LDL Cholesterol mg/dl	50	96	341	168.66	56.937

Table (2) Shows the Descriptive Statistics of smokers

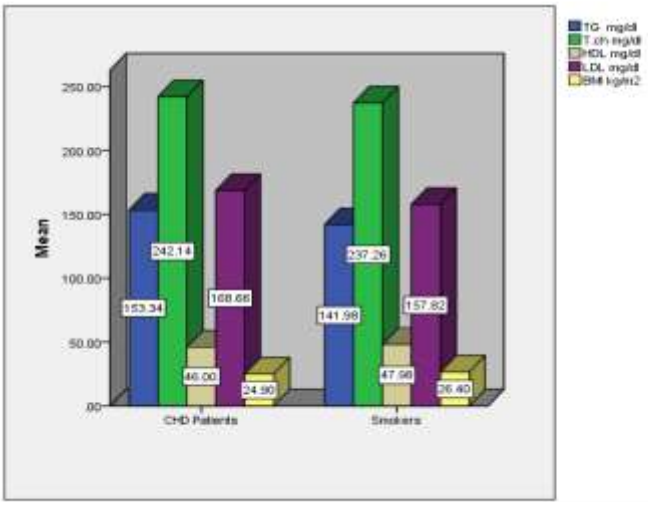
	N	Minimum	Maximum	Mean	Std. Deviation
Age /year	50	23	80	51.84	18.237
Weight /kg	50	53	104	83.02	12.138
Height cm	50	158	197	177.62	9.227
Body Mass Index kg/m2	50	18.4	32.4	26.350	2.9585
Triglycerids mg/dl	50	97	294	141.98	28.77
Total Cholesterol mg/dl	50	137	326	237.26	44.371
HDL Cholesterol mg/dl	50	24	127	47.98	17.731
LDL Cholesterol mg/dl	50	58	257	157.82	43.809

Table(3) shows the (Mean ±SD) for quantitative variables of CHD Patients and Smokers.

Variables	Coronary Heart Disease N=50	Smokers N=50	P.value
TG(mg/dl)	153.34±53.27$^{n.s}$	141.98±28.77$^{n.s}$	0.188
T.ch(mg/dl)	242.14±51.7 $^{n.s}$	237.26±44.37 $^{n.s}$	0.614
HDL(mg/dl)	46.00±6.37$^{n.s}$	47.98±17.73$^{n.s}$	0.459
LDL(mg/dl)	168.66±56.94 $^{n.s}$	157.82±43.8 $^{n.s}$	0.289
BMI(kg/m^2)	24.9±2.4 *	26.4±2.96 *	0.010

n.s=No significanct difference >0.05
*=Significant at 0.05 level of probability <0.05
**=Significant at 0.05 level of probability <0.01

Figure Shows the Comparison between Lipid Profile and BMI of CHD Patients and Smokers



DISCUSSION

Abnormalities in the lipid profile , specifically hypertriglyceridemia and low level of HDL-C have been shown to be strong predisposing issue to many diseases including obesity , diabetes and cardiovascular diseases.

In this study, the mean values of lipid profile was higher in CHD patients than smokers, except HDL-C was low (Triglycerides levels in CHD patients and smokers was (153.3 ± 53.27) mg/dl and (141.9± 28.77)mg/dl respectively.

There was no significant difference in the mean values of s. triglycerides between the two groups, but there was slightly decreasing in CHD patients (p.value >0.05).

Also it was observed that HDL-cholesterol level was increased in smokers when compared with CHD patients. The mean HDL-C in CHD patients was (46.0 ± 6.37) mg/dl and (47.98 ± 17.73) mg/dl in smokers respectively (p value >0.05).

In other studies it has been estimated that the risk of CVD decreases by 2 to 3% for every 1 mg/dL increase in HDL-C. Turner R, Millns *et al* (1998). Despite some controversy, elevated levels of triglycerides, fasting as well as nonfasting, also appear to be an independent risk factor for CHD. Eberly LE *et al* (2003), Bansal S, *et al* (2007). Evidence from epidemiologic studies suggests that the co-occurrence of low HDL-C and elevated triglyceride levels is a strong risk factor for CHD. Jeppesen J, *et al* (1997) , Assmann G, *et al* (1992).

The mean serum total cholesterol in CHD patients was (242.14 ± 51.7) mg/dl while in smokers was (237.26 ± 44.37) mg/dl. There was no significant difference in the mean values of total cholesterol between the two groups but there is slightly increasing in CHD patients (p value >0.05).

On the other hand, it was also observed that LDL-cholesterol level was decreased in smokers when compared with CHD patients. The mean LDL-C CHD patients was (168.66 ± 56.94) mg/dl and (157.82 ± 43.8) mg/dl in smokers respectively (p.value >0.05).

These findings are in accordance with the studies shown that, whether an increased level of small dense LDL represents an independent risk factor remains somewhat controversial, but it is clearly associated with an increase in CHD risk . Sacks FM, *et al* (2003).

serum cholesterol has been shown to be an established CHD risk factor in European people. Stamler J, *et al* (2000) as well as Asians . Chen Z, Peto *et al* (1991) Elevated levels of plasma LDL-C are major CHD risk factors as therapy with LDL-C drugs have reduced CHD risk and the reduction is proportion to decrease in LDL-C levels . Unit ES. *et al* (2005), de Lemos J, *et al* (2010).

As expected, already established CHD risk factors had a high prevalence in the patients in the current study as well. It was clear that lipid profile of patients was more atherogenic and patients exhibited significantly higher prevalence of other CHD risk factors when compared to smokers .

Also from the results, it was also observed that BMI was increased in smokers when compared with CHD patients. The mean the mean of BMI was (26.4±2.96) kg/m^2 and (24.9±2.4) kg/m^2 in smokers respectively (p value <0.05).

The findings are in accordance with another Pakistani study and the studies carried out in other developed countries . Kwiterovich PO *et al* (1993), Lamarche B, *et al* (1995) .

The study had limitation of small sample size. The sample size of hundreds is not sufficient to rule out any minor gender related differences. Despite these limitations, the study gives basic information about dyslipidemia in Smokers and CHD population in Sudan, and can be helpful in advanced research about lipid profile and establishing a correlation between lipid profile parameters and dyslipidemia, CHD and the complications associated with cigarettes smoking in Sudan population.

CONCLUSIONS

Tobacco smoking is associated with dyslipidaemia (Increase LDL-C and decrease HDL-C levels), these findings in Smokers may lead to increase morbidity and mortality through increasing the risk of coronary heart diseases

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