



International Journal of
Medicinal Chemistry & Analysis

www.ijmca.com

e ISSN 2249 - 7587

Print ISSN 2249 - 7595

COMPARATIVE STUDY OF LIPIDS PROFILE IN SMOKERS AND DIABETIC SUDANESE PATIENTS

Anas M. Mokhtar and Abdelkarim A. Abdrabo*

Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Al-Neelain University- Sudan.

ABSTRACT

Background: association between dyslipidemia and cardiovascular diseases has been well recognized, smoking and diabetes mellitus are factors alters lipids metabolism. This study was conducted to compare the effect of these factors on lipid profile and it aptitude to progress to CVD in Sudan. Method: lipid profile was studied in 232 volunteer (75 smokers and 75 diabetic patients and 82 normal individual as control) all of them are volunteers from Khartoum state-Sudan. Lipids profile was performed using standard methods. Results: Means of triglyceride, total cholesterol, HDL, LDL and VLDL for smoker were (86±26.8), (120±31.3), (31±8.5), (52.5±9.5), (106±32), and (17±5.3), respectively, and for diabetic were (98±42.9), (154±51.7), (45±12.8), (128±53.8), and (19±8.5), respectively, there are significant differences in lipids levels, P.value <0.05. Compared to control results of triglyceride, total cholesterol, HDL, LDL and VLDL levels = (70±32), (105±23.5), (52.5±9.5), (92.1±24.9), and (20.2±6.4), respectively, diabetic and smokers have significant higher lipids levels, P.value <0.05 except VLDL in diabetic patients. Conclusion: although the levels of lipids were within the normal values, there is significant negative effect of cigarette smoking and diabetes mellitus on lipid metabolism in Sudanese individuals.

Keywords: Smoking, Diabetes Mellitus, Lipids, CVD.

INTRODUCTION

World Health Organization (WHO) statistics show that the global mortality arising due to cardiovascular diseases (CVD) was 17.3 million in 2008 worldwide, it is estimated that CVD is responsible for around 1 in 3 premature deaths in men and 1 in 5 premature deaths in women [1]. The main risk factors for CVD including high blood pressure, smoking, diabetes, poor diet, lack of exercise and being overweight or obese [2]. Diabetes is a prime risk factor for CVD, the pathophysiology of the link between diabetes and CVD is complex and multifactorial [3]. Understanding these profound mechanisms of disease can help clinicians identify and treat CVD in patients with diabetes, as well as help patients prevent these potentially complications. Atherosclerosis is the major threat to the macro vascular for patients with diabetes mellitus [4].

Dyslipidemia is highly correlated with atherosclerosis, and up to 97% of patients with diabetes are dyslipidemic [5]. In addition to the characteristic pattern of increased triglycerides and decreased high-

density lipoprotein (HDL) cholesterol found in the plasma of patients with diabetes, abnormalities are seen in the structure of the lipoprotein particles. In diabetes, the predominant form of low density lipoprotein (LDL) cholesterol is the small, dense form. Small LDL particles are more atherogenic than large LDL particles because they can more easily penetrate and form stronger attachments to the arterial wall, and they are more susceptible to oxidation. Because less cholesterol is carried in the core of small LDL particles than in the core of large particles, subjects with predominantly small LDL particles have higher numbers of particles at comparable LDL cholesterol levels [6].

Other major risk factors for the development of CVD is to smoke cigarette [7], large number of epidemiologic studies claim that in both genders, cigarette smoking generally predisposes to the development of atherosclerosis and also increases in the incidence of myocardial infarction (MI) along with fatal coronary artery disease (CAD) [8]. Whenever a person smokes a cigarette, the chemicals in the smoke, particularly nicotine

and carbon monoxide, damage the cardiovascular system [9]. Nicotine causes both immediate and longer term increases in blood pressure, heart rate, cardiac output and coronary blood flow [10]. Carbon monoxide binds to the hemoglobin, which is normally carries oxygen from the lungs via the blood stream, and therefore reduces the amount of oxygen reaching body tissues [11]. Smoking also makes blood vessels and blood cells sticky, allowing cholesterol and other dangerous fatty material to build up inside them this is called atherosclerosis [12]. This in turn can lead to raised blood pressure and clot formation. Researches has confirmed that smoking damages the blood vessels, a study looked at the arteries of people aged 15 - 34 who had died from accident, suicide or murder and looked for evidence of fatty build up in blood vessels, and measured levels of cholesterol and thiocyanate, a marker for cigarette smoking. They found that any person who had smoked showed more early signs of atherosclerosis than people who had never smoked [13].

In the past evaluation of cardiac problems in the laboratory has been used primarily to assess cardiac disease such as heart attacks or acute myocardial infarctions (AMI) and offer body chemistry information to aid supportive cardiac therapy. Laboratory information was used to document the occurrence of an AMI and endorse a treatment regimen, such as 24-hour nursing care in the coronary care unit. more recently research has shown that risk factors can also be predictive, use of laboratory results still supports diagnosis of AMI but also assesses risk for future cardiac disease through analysis of body chemistry metabolites, such as total cholesterol, HDL, and LDL, risk factor assessment enables health-care professionals to educate the patient and to institute activities that will reduce risk for a cardiac disease.

This study was conducted to investigate the effect of smoking and diabetes on lipid profile and ability to progress to CVD in our local population.

MATERIAL AND METHODS

A total number of 232 volunteer was introduced in the study, (75 known diabetic patient, 75 cigarette smoker and 82 control non-smoker non diabetic individual), age range of diabetic was (21-52) year (M±SD= 36±7.6), for smoker (29-52)) year (M±SD=

40±6.4) and for control (18-66) year (M±SD= 42±12.3) and the duration of diabetes (Mean ±SD=6.7±4.6) year and the duration of smoking (M±SD= 12.4±6.2) year and they smoke about (11±3.2) cigarette/day, all volunteer participant were from Khartoum state, any individual complaining from thyroid dysfunction, renal disease, liver disease, hypertension and overweight or history of other disease affect lipids profile results were excluded from the study. From each participant, 5.0 ml of venous blood were collected using heparin containing container, blood was then centrifuged for 3-5 min immediately and plasma was separated for estimation of total cholesterol, triglyceride, HDL using (Mindry-BS-380China). LDL- was calculated using the equation (LDL = Total cholesterol – HDL + TG/5) and VLDL was calculated using the equation (VLDL =TG/5) [14].

Statistical evaluation was performed using SPSS (SPSS for windows version 17) to assess significant difference using T-test and correlations between lipid profile and the duration of smoking and diabetes were Assessed using bivariate correlations. P<0.05 was considered statistically significant

RESULTS

The table -1 shows comparison between lipid profile of cigarette smokers (M±SD in mg/dl) of triglyceride, total cholesterol, HDL, LDL, VLDL (86±26.8), (120±31.3), (31±8.5), (52.5±9.5), (106±32), and (17±5.3), respectively, for non-smokers were (70±32), (105±23.5), (52.5±9.5), (92.1±24.9), (20.2±6.4), respectively, there were significant differences P.values <0.05.

Table-2 shows comparison between lipid profile of diabetic and control, (M±SD in mg/dl) in diabetic patients triglyceride, total cholesterol, HDL, LDL, VLDL results were (98±42.9), (154±51.7), (45±12.8), (128±53.8), (19±8.5), respectively, there were significant differences in all lipids level except VLDL, P.value <0.05.

Table-3 shows comparison between lipid profile of diabetic patient and the smoker, lipids profile in diabetic were significantly higher compared to smokers P.value <0.05.

Table 1. Lipid profile in Control and Smoker

Parameter	Group 157		P. value
	Control(n=82)Mean ± SD	Smoker(n=75) Mean ± SD	
Triglyceride	70±32	86±26.8	0.003
Total cholesterol	105±23.5	120±31.3	0.000
HDL	52.5±9.5	31±8.5	0.000
LDL	92.1±24.9	106±32	0.002
VLDL	20.2±6.4	17±5.3	0.003

Table 2. Lipid profile in Control and Diabetic

Parameter	Group 157		P. value
	Control(n=82) Mean \pm SD	Diabetic(n=75) Mean \pm SD	
Triglyceride	70 \pm 32	98 \pm 42.9	0.005
Total cholesterol	105 \pm 23.5	154 \pm 51.7	0.007
HDL	52.5 \pm 9.5	45 \pm 12.8	0.000
LDL	92.1 \pm 24.9	128 \pm 53.8	0.000
VLDL	20.2 \pm 6.4	19 \pm 8.5	0.667

Table 3. Lipid profile in Diabetic and Smoker

Parameter	Group 150		P. value
	Diabetic(n=75)Mean \pm SD	Smoker(n=75)Mean \pm SD	
Triglyceride	98 \pm 42.9	86 \pm 26.8	0.049
Total cholesterol	154 \pm 51.7	120 \pm 31.3	0.000
HDL	45 \pm 12.8	31 \pm 8.5	0.000
LDL	128 \pm 53.8	106 \pm 32	0.003
VLDL	19 \pm 8.5	17 \pm 5.3	0.049

DISCUSSION

According to national cholesterol education program (NCEP) the lipid level evaluation based on concentration (total cholesterol below 200 mg/dl is desirable, 200-239 mg/dl is borderline high, 240 mg/dl and above high) (LDL cholesterol below 100 mg/dl optimal, 100-129 mg/dl near optimal, 130-159 mg/dl borderline high, 160-189 mg/dl high, 190 mg/dl and above very high) (HDL <40 mg/dl is low, >59 mg/dl desirable) (triglycerides below 150 mg/dl desirable, 150-199 mg/dl borderline high, 200-499 mg/dl high, 500 mg/dl and above very high), (VLDL < 30 mg/dl). In this study the results of diabetic are within desirable range HDL is near the low limit, LDL is in near optimal range. These finding is in agreement with Yadav, BK et al study which was done in Nepal 2005 [15]. Also Gordon L et al., 2010 confirmed

that dyslipidemia exists in type 2 diabetic population of their study [16]. Although the mean values of total cholesterol, LDL and TG obtained in smokers in our study are within normal values, but they are significantly higher than control group, this results is in accordance with Batic-Mujanovic O et al., 2008, who reported that smokers had significantly higher serum total cholesterol (P=0.01), triglyceride (P=0.002) and LDL level (P=0.03) and significantly lower HDL level (P=0.003) comparing with nonsmokers [17].

CONCLUSION

The results of this study can be concluded to that diabetic individuals have higher levels of lipids profile compared to control and smokers. Both factors have significant negative effect on lipids metabolism.

REFERENCES

1. Khan I, Farhan M, Ramesh S, Thiagarajan P. Effects of Smoking on Serum Lipid Levels in Nascent Young Indian Smokers. *International Journal of Pharma Sciences and Research*, 3, 2012, 463-466.
2. May AL, Kuklina EV, Yoon PW. Prevalence of Cardiovascular Disease Risk Factors Among US Adolescents (1999-2008). *Pediatrics*, 129, 2012, 1035-1041.
3. Dokken BB. The Pathophysiology of Cardiovascular Disease and Diabetes, Beyond Blood Pressure and Lipids. *Diabetes Spectrum*, 21, 2008, 160-165.
4. Garber AJ. Attenuating Cardiovascular Risk factors in Patients with Type 2 Diabetes. *Am Fam Phys*, 62, 2000, 2633-2642.
5. Ahmed SM, Clasen ME, Donnelly JF. Management of Dyslipidemia in Adults. *Am Fam Physician*, 57, 1998, 2192-2204
6. Grundy SM. Small LDL, Atherogenic Dyslipidemia, and the Metabolic Syndrome. *Circulation*, 95, 1997, 1-4.
7. Akbari MZ, Bhatti MS, Shakoor M. lipid profile in smoking. *JAMC*, 2000, 12, 19-21.
8. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease. *J Am Coll Cardiol*, 43, 2004, 1731-1737.
9. Zevin S, Saunders S, Gourlay SG, Peyton Jacob P, Benowitz NL. Cardiovascular effects of carbon monoxide and cigarette smoking. *J Am Coll Cardiol*, 38, 2001, 1633-1638.
10. Jolma CD, Samson RA, Klewer SE, Donnerstein RL, Goldberg SJ. Acute cardiac effects of nicotine in healthy young adults. *Echocardiography*, 19, 2002, 443
11. Sangone A, Lawrence T, balcerzak SP. The effect of smoking in tissue oxygen. *Blood journal*, 41, 1973, 845-851
12. Pittilo R M. Cigarette smoking, endothelial injury and cardiovascular disease. *Int J Exp Pathol.*, 81, 2000, 219-230.

13. McGill HC, McMahan CA, Malcom GT, Oalman MC, Strong JP. Effects of Serum Lipoproteins and Smoking in Young Men and Women. Atherosclerosis. *Thrombosis and vascular biology*, 17, 1997, 95-106.
14. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without the use of the preparative ultracentrifuge. *Clin Chem.*, 18, 1972, 499-502.
15. Yadav BK., Bade AR, Singh J, Jha B. Comparative study of lipid profile in smokers, tobacco chewers and diabetic patients. *Journal of Institute of Medicine*, 27, 2005, 38-41.
16. Gordon L, Ragoobirsingh D, Morrison EY, Choo-Kang E, McGrowder D, Martorell E. Lipid profile of type 2 diabetic and hypertensive patients in the jamaican population. *J Lab Physicians*, 2(1), 2010, 25-30.
17. Batic-Mujanovic O, Beganlic A, Salihefendic N, Pranjic N, Kusljugic Z. Influence of smoking on serum lipid and lipoprotein levels among family medicine patients. *Med Arh.*, 62(5-6), 2008, 264-7.