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Lipid Profile among Sudanese Cigarette Smokers in Khartoum State, Sudan

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Abstract: It has proven that high level of LDL-C and low levels of HDL-cholesterol predispose individuals to the risk of coronary heart disease. Cigarette smoke contains toxicants that can disrupt normal metabolic process and cause dyslipidemia. The present study was undertaken to evaluate lipids profile among healthy Sudanese smoker in Khartoum state and to compare it with healthy non-smoker in the fasting state. This was across-sectional studies conducted during the period from May to October 2011. Fifty male smoker and fifty male non-smoker were recruited for the study after obtaining written informed consent. Both groups were age and weight matched. The serum levels of total cholesterol, low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and triglycerides were assayed for each group using standard biochemical methods. The serum levels of total cholesterol, LDL-C and triglycerides were significantly raised in smoker group when compared to non-smoker group (P<0.05). Where's the serum level of HDL-C were significantly reduced in the smoker group (P<0.01). There was a significantly strong positive correlation between the serum levels of total cholesterol, triglyceride and number of cigarette smoked per day and a significant moderate positive correlation between the serum levels of LDL-C and the number of cigarettes smoked per day. These finding indicate that smoking produce adverse effects on lipid profile, therefor increasing the risk of coronary heart disease.

Keywords: lipids profile, coronary heart disease, total cholesterol, HDL-C, LDL-C, triglyceride

1. Introduction

Worldwide the effect of smoking are estimated to kill 3 million per year. This contrast with 0.2million in 1950 and projections for 2050 of 10 million (1). Smoking in different form is a major risk factor for atherosclerosis and coronary heart disease (7,14). In Pakistan it is estimated that 365 men and 9% women used forms of tobacco on regular bases and average age of onset is 18 years in males and 24 years for females (3). there is dose response relationship between the number of cigarettes smoked and cardiovascular morbidity and mortality (4,15) dyslipidemia associated with cigarette smoking was reported by various workers (10,12). There is overwhelming evidence from cohort studies that high plasma cholesterol concentration is associated with increased risk of Coronary Heart Disease(CHD) and that decreasing plasma cholesterol concentration decrease the risk of coronary heart disease it has been estimated that each 1% increase in plasma cholesterol concentration is associated with a 2.7% increase in risk (6).

Cigarette smoking hypertension, hyperlipidemia, obesity, and physical inactivity have long been recognized as environmental risk factors for CHD (3).

Since dietary and environmental factors influence lipid profile, the lipid profile of Sudanese cigarette smoker in Khartoum state has been determined to see if variation occur in smokers.

2. Materials and Methods

50 healthy non-obese male smokers in age group of 20-40 years were recruited for the study after obtaining written informed consent (test group). 50 non-obese male non-smokers, age and weight matched were selected as control (control group). Diuretics a detailed physical examination of

the subjects of both groups was done, fasting venues blood sample were collected from subjects by vein puncture into plain sample container. The blood was allowed to clot the sample was spent at 3000 rpm for 3 min and the serum was collected for analysis. Total cholesterol, triglyceride, LDL and HDL- cholesterol were estimated using the enzymatic end point kit methods

Inclusion criteria: male cigarette smokers

Exclusion criteria - Those excluded from the study person up using alcohol, X-EX smoker with diabetes mellitus, hypertension, renal disease, hepatic impairment, endocrine disorder and obesity and on drugs like beta blockers, lipid lowering drugs, and thiazide diuretics

Study was conducted under the following parameters:

- a. History taking
- b. clinical examination

3. Observations and Results

Table 1: Mean total cholesterol, HDL-C, LDL-C and triglycerides for Smokers and non-Non-smoker

Lipid Profile	Smokers	Non-Smokers	P value	
Values (mg/dL)	(Mean+SD)	(Mean+SD)		
	n = 50	n= 50		
total cholesterol	176.7±18.1	150.8±21.4	<0.05*	
HDL-Cholesterol	36.5±8.8	56.8±12	<0.01**	
LDL-Cholesterol	123.4±18	77.7±16	<0.01**	
Triglycerides	178.6±23.8	132.3±22	<0.01**	
*Significant, **Highly significant				

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Table 1: Pearson's correlation coefficients (r) for serum lipid profile and number of cigarette

Parameters	Correlation coefficients		P value	
total cholesterol	0.78		0.041	
HDL-Cholesterol	ol 0.00		0.931	
LDL-Cholesterol 0.59			0.047	
Triglycerides	0.75		0.035	
P value < 0.05 is considered significant				

Table 1 shows significantly raised means of serum total cholesterol, LDL-C, and triglyceride of cigarette smoker when compare with the non- smokers, were as the means of serum HDL is significantly reduced in the smokers. Table 2 shows significantly, strong positive correlation between the serum levels of total cholesterol, triglycerides and the number of cigarettes smoke per day and also a significant- moderate positive correlation with the serum levels of LDL-C and non-correlation with the serum level of HDL-C

4. Discussion

The mean of serum total cholesterol, triglyceride and LDL-C were significantly were raised in the smokers group when compare to nonsmoker, these observations are in tune with findings of other workers (10,12). The mean of serum levels of HDL-C was significantly reduced in the smokers group, thus cigarette smoking substantially increased the risk of coronary heart disease and ischemic stroke (5,8,11). Various mechanisms leading to lipid alteration by smoking, these are: (a) nicotine stimulate sympatric adrenal system leading to increase secretion of catecholamine resulting in increased lipolysis and increased concentration of plasma free fatty acid (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglyceride along with very low density lipoprotein (VLDL) in the blood stream (16), (b) fall in estrogen levels occur due to smoking which further leads to decrease HDL-C its interesting to note in this study that there is a strong positive correlation between the serum levels of total cholesterol - triglyceride and the number of cigarette smoke per day. Smoking alter the lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with the number of cigarette smoked (8)

5. Conclusion

The serum anti estrogenic HDL-C levels are significantly low in cigarette smokers in respective of the number of cigarettes smoked per day. The serum levels of total cholesterol, low density LDL-cholesterol and triglyceride are significantly raised in cigarette smokers as compared to non-smokers and therefor rising the cardiovascular diseases. Increasing the number of cigarette smoked per day also increased the risk.

References

- [1] Betteridge DJ, Morrell JM. Clinicians' guide to Lipids and Coronary Heart Disease. London: Chapman Hall Medical; 1988;126-130.
- [2] Brischetto CS, Conner WE, Connor SL, Matarazzo JD. Plasma lipid and lipoprotein pro.les of cigarette smokers from randomly selected families: Enhancement of

- hyperlipidemia and depression of high density lipoprotein. AmJ Cardiol 1983; 52: 675.
- [3] 3-Fick H, Van Antropen VL, Richards GA. Increased levels of antibiotics to cardiolipids and oxidized LDL-Cholestrol are increase associated with plasma vitamin C status in cigarette smokers 1996;124: 75-81
- [4] Kannel WB. Update on the risk of cigarette smoking in coronary artery disease. *Am Heart J* 1981; 101: 319-28.
- [5] Krishna Swami S, Richard J, Prasad NK et al. Association between cigarette smoking and coronary artery disease in patients in India. How quantitative is it? An assessment by selective coronary arteriography. *Intern J Cardiol* 1991; 31: 305-12.
- [6] Law MR, Wald NJ, Thompson SG. By how much and how quickly dose reduction in serum cholesterol concentration lower the rick of ischemic heart disease. Br.Med. J 1994; 308: 1933-40.
- [7] Mc Gill HC. Cardiovascular pathology of smoking. *Am Heart J* 1988; 115: 250-7.
- [8] Michael A, Jonas, John AO et al. Statement on smoking and cardiovascular disease for health care professionals. AHA Medical/Scientific statement. Circulation 1992; 86: 1644-9.
- [9] MJOS OD. Lipid effects of smoking. Am Heart J 1988;115: 272-5.
- [10] Muscat JE, Harris RE *et al.* Cigarette smoking and plasma cholesterol. *Am Heart J* 1991; 121: 141-7.
- [11] Rosenson RS. Low level of HDL-cholesterol (Hypoalphalipoproteinemia). An approach to management. *Arch Intern Med* 1993; 153(13): 1528-40.
- [12] Rustogi R, Shrivastva SSI *et al.* Lipid profile in smokers. *JAPI* 1989; 37 (12): 764-7.
- [13] Simons LA, Simons J, Jones AS. The interaction of body weight, age, cigarette smoking and hormone usage with blood pressure and plasma lipids in an Australian community. *Aus NZ J Med* 1984; 14: 215-21.
- [14] Wilhelmsen L. Coronary heart disease. Epidemiology of smoking & intervention studies of smoking. *Am Heart J* 1988; 115: 242-7.
- [15] Wynder EL, Harris *et al.* Population screening for plasma cholesterol. Community based results from Connecticut. *Am Heart J* 1989; 117: 649-56.
- [16] Berliner J., Navab M., Fogelman AM. et al. Atherosclerosis: Basic mechanism, oxidation, inflammation and genetics. Circulation 1986; 5(supp):5-10.

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