Proceeding S.Z.P.G.M.I. vol: 17(2): pp. 71-73, 2003.

Study of Erythrocyte Sedimentation Rate (ESR) Values and Serum Lipids in Young Cigarette Smokers

Mehrun Nisa, Samina Zaman

Department of Physiology and Department of Pathology, King Edward Medical College, Lahore,

SUMMARY

The aim of this study was to find out the association between cigarette smoking and erythrocyte sedimentation rate (ESR) values and serum lipid profile in young smokers and compare it with age and weight matched non-smokers. The subjects consisted of 50 healthy cigarette smokers and 50 healthy, age and weight matched non-obese, non-smokers who served as controls. Subject in both groups were 25 -35 years of age. They had no history of diabetes mellitus, hypertension, hepatic or renal disease, obesity, alcohol abuse and were not taking any medicines like β-blockers, diuretics or lipid lowering agents. Results showed significantly higher ESR values, serum total cholesterol (S.TC), low density lipoprotein cholesterol (LDL-C) and mean serum triglyceride (TG) levels, in smokers as compared to non-smokers. Mean serum high density lipoprotein cholesterol (HDL-C) was significantly lower in smokers as compared to non smokers.

Conclusion: smoking produces rise in ESR values and adverse effects on Lipid profile, thus increasing the risk of atherosclerosis.

Keywords: Cigarette Smoking, Erythrocyte sedimentation rate values, Serum total cholesterol.

INTRODUCTION

Cigarette smoking is a powerful risk factor for atherosclerosis and coronary heart disease.[1,2] There is direct relationship between number of cigarettes smoked and cardiovascular morbidity and mortality.[3,4] Rise in S.TC, LDL-C, TG and fall in antiatherogenic cholesterol (HDL-C) have been reported by many authors. Mechanism by which cigarette smoke may initiate or accelerate atherosclerosis and its complication are:

a. Nicotine stimulates catecholamines resulting in lipolysis and increased concentration of plasma free fatty acids (FFAs) which further results in increased secretion of hepatic FFAs and triglycerides along with very low density lipoprotein cholesterol VLDL-C in blood.[9,10]
b. Fall in estrogen due to smoking leads to decreased HDL-C.
c. Hyperinsulinaemia in smokers leads to increased cholesterol, LDL-C, VLDL-C and TG due to decreased activity of lipoprotein lipase.[12,13]
d. Cigarette smoke induces endothelial damage by producing free radicals such as nitric oxide and hydrogen peroxide. This oxidative stress promotes a systemic acute phase reaction thus increasing inflammatory cytokines, C-reactive protein, fibrinogen, blood cell count whole blood viscosity and rouleaux formation. Eventually this leads to rise in ESR values.[14,15,16]

AIMS AND OBJECTIVES

The aim of the study was to find out difference in the serum lipid profile and ESR values between young smokers and non-smokers in the fasting state.

MATERIALS AND METHODS

Fifty healthy male smokers in the age group of 25-35 years were recruited for the study after obtaining written informed consents (Group-I). 50 healthy non- obese, non smokers, age and weight matched male subjects were selected from hospital staff and attendants and were regarded as control (Group-II). Exclusion criteria was alcoholics, ex-smokers, diabetics, hypertensives with renal or hepatic failure, endocrine disorders, obesity or on drugs like β -blockers, lipid lowering agents and thiazide diuretics. A detailed physical examination of both groups was done. After overnight fasting they were subjected to investigations including blood glucose, lipid profile (S.TC TG, LDL-C, HDL-C) and blood urea.

For determination of ESR the modified Westergrens method was used by drawing non-fasting venous blood samples into EDTA with subjects in supine position with only short term venous occlusion.[17]

RESULTS

Most of the smokers (34) had been smoking for a mean duration of 8 years as shown in Table 1. Anybody smoking for at least one cigarette per day and had smoked for at least one year was considered as active and chronic smoker.

Table 1: Distribution of smokers in relation to duration and number of cigarettes smoked (n=50)

Smokers		Duration of	Mean
No.	%	smoking (Years)	(Years)
10	20	1-5	3
34	68	6-10	8
6	12	11-15	13

The serum lipid concentration in smokers and nonsmoker revealed significant difference (Table 2). The mean total serum cholesterol level in non smoker was 159.8 (±19.26) mg/dL and in smoker it was much higher, that is, 180±25.1 mg/dL. The mean Triglyceride level was 130.1±30.61 and 175±65.56 mg/dL in non smokers and smokers respectively; again showing gross difference. There was no significant difference in the value of high density lipids. The mean HDL-C in non smokers was 46.65±5.18 mg/dL and in smokers it was 43.5±6.12 mg/dL. For low density lipid difference was significant. The mean LDL-C for non smoker and smoker was 85±17.79 mg/dL and 105.2±21.16 mg/dL respectively.

The mean ESR value for smoker, i.e; 8 mm/ Hr was much higher than non smoker i.e; 3.6 mm/Hr (Table 2).

Most of the smokers (34) had been smoking for a mean duration of 8 years as shown in Table 1.

Table 2: Lipid profile and ESR in non-smokers and smokers.

Lipid Profile values mg/dl	Non-smokers (n-50)	Smokers (n = 50)	P value
Mean TC	159.8±19.26	180±25.10	< 0.05
Mean TG	130.10±30.61	175±65.56	< 0.01
Mean LDL-C	85±17.79	105.2±21.16	< 0.05
Mean HDL-C	46.65±5.18	43.5±6.12	< 0.01
Mean ESR	3.66±1.59	8.025±2.66	<0.001

P value < 0.05 significant

DISCUSSION

The mean total serum cholesterol in non-smokers was 159.8±19.26 mg/dl while it was significant higher (P<0.05) in smokers i.e. 180±25.10 mg/dl. These observation are similar to the findings of others.[6,7,9] The mean serum triglyceride level in non-smokers and smokers were 130.10±30.61 mg/dl and 175±65.56 mg/dl respectively. These findings are consistent with those observed by Wynder et al.[4] and Rustogi et al.[7] The mean LDL –C in non-smokers and smokers was 85±17.79 mg/dl and 105.2±21.16 mg/dl respectively showing significant rise in smokers similar to Rustogi et al.[7]

The mean HDL-C in non smokers was 46.65±5.18 mg /dl and in smokers 43.5±6.12 (P value < 0.01). This findings is consistent with the study of Rosenson[18] who reported that there is a fall in HDL-C level by 3-5 mg/dl in smokers.

The erythrocyte sedimentation rate (ESR) values given in Table 2 show a significant rise of ESR (P value < 0.001) in smokers as compared to

non-smokers indicating a strong association of markers of systemic inflammation with smoking. similar results are shown by Bermudez et al.[15]

CONCLUSION

The serum antiatherogenic HDL-C level is significantly low in cigarette smokers irrespective of the No. of cigarettes smoked. While levels of total cholesterol, TG and LDL-C are significantly increased. Also there is significant rise in ESR value in smoker indicating widespread vascular inflammation.

REFERENCE

1. Wilhelmsen L. Coronary heart disease. Epidemiology of smoking and intervention studies of smoking. Am Heart J 1988; 115: 242-7.
2. Mc Gill HC. Cardiovascular pathology of smoking. Am Heart J 1988; 115: 250-7
3. Kannel WB. Update on the risk of cigarette smoking in coronary artery disease. Am Heart J 1981; 101: 319-28.
4. Wynder EL, Haris et al. Population screening for plasma cholesterol. Community based results from Connecticut. Am Heart J 198; 56.
5. Carison LA, Bottiger LE, Ahfeldt PE. Risk factors for myocardial infarction in the Stockholm prospective study. A 14 years follow up on the role of plasma triglycerides and cholesterol. Acta med Scand 1979;206: 315-60.
6. Mjos OD. Lipid effects of smoking. Am Heart J 1988; 115: 272-5.
7. Rustogi R, Shrivastva SSI, et al. Lipid Profile is smokers. JAPI 1989; 37(12): 764-7.
8. Austin MA. Plasma triglycerides and coronary heart disease. Arterio Throm 1991; 11: 2-14.
9. Muscat JE, Harris RE, et al. Cigarette smoking and plasma cholesterol. Am Heart J 1991; 121: 141-7.
10. Simons LA, Simons J, Jones AS. The interaction of body weight, age, cigarette smoking and hormone usage with blood pressure lipids in an Australian Community. Aus NZ J med 1984; 14: 215-21.
11. Benouwitz HL. Pharmacologic aspects of cigarette smoking and nicotin addiction. New Engl Med. 1998; 319: 318-30.
12. Stalder M, Pometta B, Suenram A. Relationship between plasma insulin levels and HDL cholesterol in Health men. Diabetologia 1998; 72:212-14.
13. Reaven GM. Role of Insulin resistance in human disease. Diabetes 1988; 37: 595-607.
14. Tappia PS, Troughton KL, Langley Evans Sc et al. Cigarette smoking influences cytokine production and antioxidant defe Sci. 1995; 88: 485-89.
15. Bermudez EA, Rifai N, Buring JE, et al. Relation between markers of systemic vascular inflammation and smoking in wom Cardiol. 2002; 89: 1117-19.
16. De Matt MPM, Pietersma A, Kofflard M et al. Association of plasma fibrinogen level with coronary artery disease, smoki inflammatory markers. Atherosclerosis. 1996; 121: 185-191.
17. Piva E, Fassina P, Plebani M. Determination of length of sedimentation reaction in non-anticoagulated blood. Clin. Chem Lab. Med. 2002 July; 40 (7): 713-7.
18. Ronsenson Rs. Low level of HDL cholesterol (Hypoalphalipoproteinemia. An approach to management. Arch intern med. 1993; 15: 40-7.

The Authors:

Mehrun Nisa,
Assistant Professor
Department of Physiology,
King Edward Medical College,
Lahore,

Samina Zaman
Department of Pathology,
King Edward Medical College,
Lahore.