Original Article

MJSBH January-June 2013|Vol 12| Issue 1

Assessment of Biochemical Parameters among Smokers and Tobacco Chewers to Ascertain Cardiovascular risk.

Dipendra Kumar Jha[1], Dipendra Raj Pandeya[1], Satrudhan Prasad Gupta[1].

[1]Department of Biochemistry, Nepalese Army Institute of Health Sciences.

ABSTRACT

Introduction: Cigarette smoking is one of the major cause and established risk factor of premature death due to respiratory and cardiovascular illness worldwide. Risk of coronary heart disease is increased by two-to four folds in smoking and tobacco chewing. Smoking and tobacco chewing leads to change in the concentration of serum total cholesterol, triglycerides, LDL, VLDL and HDL. In our present study, the main objective was to assess the blood lipid profile among smokers and tobacco chewers to ascertain cardiovascular risk in Nepal.

Methods: It was a hospital based case control study carried out using data retrieved from the register maintained in the Department of Biochemistry of Institute of Medicine Teaching Hospital, Kathmandu, Nepal between 1st January, 2008 and 31st December, 2009. Of the 150 subjects enrolled in this study, 50 were current smokers, 50 were tobacco chewers and 50 were normal healthy controls. The variables collected were age, gender, total cholesterol, triglycerides, HDL, LDL, VLDL. The One way ANOVA was used to examine the statistical significant difference between groups. Post Hoc test LSD used for the comparison of means of control versus case groups. A p-value of <0.05 (two-tailed) was used to establish statistical significance.

Results: The mean values of serum total cholesterol (257.5±22.6 mg/dl), LDL (186.6±24.0 mg/dl), TG (139.4±39.8 mg/dl) were significantly higher in smokers when compared to controls. In contrast to that HDL (42.9±1.5 mg/dl) was lower when compared to controls (44.8±1.9mg/dl). The mean values for TG (141.5±34.9 mg/dl), total cholesterol (260.3 ±21.2 mg/dl), LDL (188.5±26.0 mg/dl) in tobacco chewers was significantly higher when compared to controls.

Conclusions: The lipid profiles are raised in tobacco chewers and smokers which may lead to higher incidence of cardiovascular disease.

Keywords: cigarette smoker; tobacco chewers; cardiovascular; risk.

INTRODUCTION

Cigarette smoking is one of the major cause and established risk factor of premature death due to respiratory and cardiovascular illness in developing and developed countries[1,2]. Approximately there were 800 million smokers in developed countries as compare to 300 million in developing countries and it was the third leading cause of casualty in the United States[3]. An estimated 443,000 people die of smoking-related diseases in the United States[4]. Nearly four-fifths of estimated 1.1 million smokers live in low or middle-income countries[5]. The number of female smokers is higher in Nepal in comparison to other countries especially from hilly and Himalayan regions in young community[6]. Cigarette smoke is composed of over 4000 chemicals many of which are strong oxidizing agents and chemical carcinogens[7]. Tobacco kills more people than AIDS, alcohol, car crashes, murder, suicides and fires. Cigarette smoking not only causes injury to active but passive smokers also. Risk of coronary heart disease is increased by two-to four folds in smoking

Correspondence:
Dipendra Kumar Jha
Department of Biochemistry, Nepalese Army Institute of Health Sciences,
Kathmandu, Nepal
Email: dipendrakumar@gmail.com

and tobacco chewing[8]. Smoking and tobacco chewing leads to change in the concentration of serum total cholesterol, triglycerides, LDL, VLDL and HDL. The changes in lipid profile leads to atherosclerosis which compounded the risk of getting cardiovascular disease. In our present study, the main objective was to assess the blood lipid profile among smokers and tobacco chewers to ascertain cardiovascular risk.

METHODS

It was a hospital based case control study carried out using data retrieved from the register maintained in the Department of Biochemistry of Institute of Medicine Teaching Hospital, Nepal between 1st January, 2008 and 31st December, 2009. Among 150 subjects enrolled in this study, 50 were current smokers, 50 were tobacco chewers and 50 were normal healthy controls. Questionnaires were used to assess the smokers and tobacco chewers and control for this study. Current smokers and chewers were only included and those who were old smokers and chewers were excluded from this study. The patients receiving lipid lowering agents, those having renal, hepatic, thyroid disorders and patients who were taking non cardiac drugs which affect the lipid profile were excluded from the study.

The variables collected were age, gender, total cholesterol, triglycerides, HDL, LDL, and VLDL. Estimation of total cholesterol and triglycerides was done by CHOD-PAP[9] and GPO-PAP method respectively. Estimation of high density lipoproteins was done by kinetic enzymatic method[10]. The values of LDL and VLDL were obtained by the Friedewald formula[11]. All these laboratory parameters were analyzed using Human reagent kits and semi auto analyzer (Humalyser 3500, Germany).

Preceding the study, approval for the study was obtained from the institutional research ethical committee. Analysis was done using descriptive statistics and testing of hypothesis. The data was analyzed using Excel 2003, R 2.8.0, Statistical Package for the Social Sciences (SPSS) for Windows Version 16.0 (SPSS Inc; Chicago, IL, USA) and the EPI Info 3.5.1 Windows Version. The One way ANOVA was used to examine the statistical significant difference between groups. Post Hoc test LSD used for the comparison of means of control versus case groups. A p-value of <0.05 (two-tailed) was used to establish statistical significance.

RESULTS

The mean values of serum total cholesterol (257.5±22.6 mg/dl), LDL (186.6±24.0 mg/dl), TG (139.4±39.8 mg/dl) were significantly higher in smokers when compared to controls. In contrast to that HDL (42.9±1.5 mg/dl) was lower when compared to controls (44.8±1.9) mg/dl) (Table 1).

The mean values for TG (141.5±34.9 mg/dl), total cholesterol (260.3 ±21.2 mg/dl), LDL (188.5±26.0 mg/dl) in tobacco chewers was significantly higher when compared to controls. There was also significant difference in HDL level for tobacco chewers in comparison to controls.

Table 1. Comparison of lipid profile between smokers and controls.

Variables	Controls	Smokers	P - value
TC (mg/dl)	173.2±32.6 (169.5-176.9)	257.5±22.6 (254.9-260.1)	0.0001
TG (mg/dl)	115.0±25.12 (112.2-117.9)	139.4±39.8 (134.9-144.0)	0.0001
HDL (mg/dl)	44.8±1.9 (43.6-45.1)	42.9±1.5 (42.8-43.1)	0.0001
LDL (mg/dl)	104.4±25.4 (101.5-107.2)	186.6±24.0 (183.9-189.4)	0.0001
VLDL (mg/dl)	23.0±4.85 (22.4-23.5)	27.9±7.9 (27.1-28.8)	0.0001

Table 2: comparison of lipid profile between tobacco chewers and controls.

Variables	Controls	Tobacco chewers	P - value
TC (mg/dl)	173.2±32.6 (169.5-176.9)	260.3 ±21.2 (256.5-264.2)	0.0001
TG (mg/dl)	115.0±25.12 (112.2-117.9)	141.5±34.9 (135.1-147.8)	0.0001
HDL (mg/dl)	44.8±1.9 (43.6-45.1)	43.6±1.4 (43.3-43.8)	0.0001
LDL (mg/dl)	104.4±25.4 (101.5-107.2)	188.5±26.0 (183.8-193.2)	0.0001
VLDL (mg/dl)	23.0±4.85 (22.4-23.5)	28.24±6.9 (26.98-29.5)	0.0001

DISCUSSION

It has been accounted that prevalence of cardiovascular disease is unswervingly linked to number of cigarettes smoked and tobacco chewed. Risk of unexpected casualty raises by 3-6 times in smokers than in non smokers. The present study illustrated that the significant higher levels of total cholesterol 257.5±22.6 mg/dl in smokers as compared to that of controls 173.2±32.6 mg/dl. Our results concurred with the findings of Friedman et al[12]. In contrast to that HDL levels showed statistically significant decrease in smokers 42.9±1.5

mg/dl as compared to controls 44.8±1.9 mg/dl. These results are in conformity with those of Criqui and his collegues[13]. The cigarette smoking has been related with altered total cholesterol, triglycerides levels, reduced fibrinolysis, augmented fibrinogen levels and variation in endothelial and platelet functions, which can lead to atherosclerosis. Similarly in tobacco chewers, levels of total cholesterol 260.3±21.2 mg/dl and triglycerides 141.5±34.9 mg/dl were significantly higher when compared to the levels of total cholesterol 173.2±32.6 mg/dl and triglycerides115.0±25.12 mg/dl in controls respectively. Nicotine which is main constituents of tobacco has a substantial influence on elevating the lipid levels in blood[14]. Nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA), which further results in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL in the blood stream[15].

CONCLUSIONS

The lipid profile is deranged in smokers and tobacco chewers when compared to control which may be the reason for higher incidence of cardiovascular disease.

REFERENCES

1. *Tertemiz KC, Kömüs N, Ellidokuz H, Sevinç C, Cımrın AH. Mortality and factors affecting mortality in chronic obstructive pulmonary disease. Tuberk Toraks. 2012;60(2):114-22.*
2. *Gellert C, Schöttker B, Brenner H. Smoking and all-cause mortality in older people: systematic review and meta-analysis. Arch Intern Med. 2012;172(11):837-44.*
3. *Wewers ME, Bailey WC, Carlsen KH, Eisner MD, Folan P, Heath J, et al. American Thoracic Society Subcommittee on Tobacco Control Initiatives. An official American Thoracic Society workshop report: tobacco control initiatives within the American Thoracic Society. Proc Am Thorac Soc. 2010;7(1):1-7.*
4. *Kahende JW, Loomis BR, Adhikari B, Marshall L. A review of economic evaluations of tobacco control programs. Int J Environ Res Public Health. 2009;6(1):51-68.*
5. *Sreeramareddy CT, Ramakrishnareddy N, Harsha Kumar H, Sathian B, Arokiasamy JT. Prevalence, distribution and correlates of tobacco smoking and chewing in Nepal: a secondary data analysis of Nepal Demographic and Health Survey-2006. Subst Abuse Treat Prev Policy. 2011;6:33.*
6. *Aryal UR, Lohani SP. Perceived risk of cigarette smoking among college students. J Nepal Health Res Counc. 2011;9(2):176-80.*
7. *Canales L, Chen J, Kelty E, Musah S, Webb C, Pisano MM, et al. Developmental cigarette smoke exposure: liver proteome profile alterations in low birth weight pups. Toxicology. 2012;300(1-2):1-11.*
8. *Lakier JB. Smoking and cardiovascular disease. Am J Med. 1992;93(1A):8S-12S.*
9. *Trinder P. Determination of serum cholesterol by enzymatic colorimetric method. Ann ClinBiochem. 1969;6:24-7.*
10. *Moshides S. Kinetic Enzymatic Method for Automated Determination of HDL Cholesterol in Plasma. J Clin Chem Clin Biochem. 1987;25(9):583-7.*
11. *Warnick GR, Knopp RH, Fitzpatrick V, Branson L. Estimating low-density lipoprotein cholesterol by the Friedewald equation is adequate for classifying patients on the basis of nationally recommended cutpoints. Clin Chem.1990;36(1):15-9.*
12. *Friedman GD, Dale LG, Ury HK. Mortality in middle-aged smokers & non-smolers. N Engl J Med. 1979;300(5):213-7.*
13. *Criqui MH, Wallace RB, Heiss G, Mishkel M, Schonfeld G, Jones GT. Cigarette smoking and plasma high-density lipoprotein cholesterol. The Lipid Research Clinics Program Prevalence Study. Circulation. 1980;62:70-6.*
14. *Rose EJ. Nicotine and nonnicotine factors in cigarette addiction. Psychopharmacology (Berl). 2006;184(3-4):274-85.*
15. *Markus H, Wolfgang K. Nicotine and sympathetic neurotransmission. Cardiovasc Drugs Ther. 1997;10(6):657-65.*