CIGARETTE SMOKING EFFECTS ON LIPID PROFILE

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خلاصه

Triglyceride (TG), High Denesity lipoprotein, Low density Cholestrol(LDC), Total حاصل کردہ خونی نمونوں سے Triglyceride (TG), Job Cholistrol(TC) کی جانچ کی گئی۔

Abstract

The aim of this study was to determine the dyslipidemias in cigarette smokers of Karachi.

This study was conducted at Federal Urdu University of Arts, Science and Technology (FUUAST) Gulshan-e-Iqbal campus Karachi and Abbassi Shaheed Hospital Karachi from Feb 2015 till Feb 2016. In this study 200 smokers and 200 non-smokers were included between age 18-45 years. Primary outcome measures were total cholesterol (TC), low density lipoprotein (LDL), triglyceride (TG) and high density lipoprotein (HDL).

The mean TC, LDL and TG was $174.60 \pm 24.46 \text{ mg/dl}$, $116.11 \pm 13.0 \text{ mg/dl}$ and $130.77 \pm 19.62 \text{ mg/dL}$ in smokers, respectively. It was significantly increased in smokers as compared to non smokers. While HDL was significantly decreased in smokers (32.51 ± 5.89). The p value for TC between smokers and nonsmokers group is <0.05, while for TG p value is <0.01. For LDL cholesterol p value is statistically significant <0.001. Cigarette smoking is associated with significant lower level of serum HDL and high level of TC, TG and LDL

Introduction

Tobacco smoking is considered as a one of the leading cause of preventable death worldwide. According to WHO (2011) tobacco is responsible for about 5 million deaths each year of which 100,000 deaths occurs in Pakistan. WHO estimates that unless current smoking pattern is reversed, tobacco will be responsible for 10 million deaths per year, by 2020-2030, with 70% occurring in developing countries (Mackay and Erikson, 2002).

According to WHO in 2015, over 1.1 billion people smoked tobacco. In south Asia Pakistan has highest consumption of tobacco (Pak. Today, 2004). The overall tobacco smoking prevalence in Pakistani men is approximately 28.6%. Generally youth and particularly adolescents have this deadly habit with severe physical, psychological and economic burden. In 2000, smoking was practiced by 1.22 billion people, predicted to rise to 1.5

to 1.9 billion by 2025 (Guindon and Boisclair, 2003). Death attributable to tobacco is expected to rise from 1.4% of all deaths in 1990 to 13.3% in 2020.

There are a lot of hazards reported due to tobacco. Smoking adversely affect almost all organs of body and it affects general health of the person (USDHHS, 2015, 2010). Smoking is estimated to cause about 71% of all lung cancer deaths, 42% of chronic respiratory disease and nearly 10% of cardiovascular disease.

Coroary heart disease has been reported as an independent risk factor in smokers, risk of heart disease increases by which mechanism is still unclear. Four reasons have been hypothesized.

1) Increase level of carbon monoxide(Zevin *et al.*, 2001) in blood of smokers may injure the endothelium and speed up the cholesterol level into the artery wall which is the cause of atherosclerosis (Bano *et al.*, 2002)

2) The formation of carboxyhemoglobin creates relative anoxemia in tissue including the myocardium (Sagcan *et al.*, 2003)

3) The nicotine absorbed from cigarette smoke induces cardiac arrhythmias through its pharmacologic action.

4) An another mechanism has been recently suggested that smoking harmfully affect the concentration of lipid in plasma and lipoprotein

However, there are few international studies available about the relationship of tobacco smoking and plasma lipid levels but the results are inconclusive and conflicting. Sangdith (2008) observed high level of plasma cholesterol in smokers than non-smokers. Only a few studies have specifically observed plasma lipid abnormalities in comparison to cigarette smoked per day (Brischetto, 1983).

Although cigarette smoking is very common in Pakistan and it is increasing day by day, very limited local data available on the affect of smoking and lipid abnormalities. So this study is designed to demonstrate detailed profile of plasma lipid and lipoprotein levels according to cigarette smoked per day tobacco usage is also risk factor of many other types of cancers.

Material and Method

The study was conducted at Federal Urdu University of Arts, Science and Technology (FUUAST) Gulshan-e-Iqbal campus Karachi and Abbassi Shaheed Hospital Karachi from Feb 2015 till Feb 2016.

In this study 200 healthy smoker males and 200 healthy nonsmoker males were randomly selected from general public of Karachi.

After obtaining written consent, detailed history and physical examination was done in all subject.

Inclusion criteria

All males between 18-45 years with a BMI less than 28 on an average dietwere divided into 4 groups.

a) Non-smokers: subjects who have never smoked or those who left smoking atleast 5 years before the present study b) Mild smokers: 1-15 cigarettes/ day for at least 5 years or more.

c) Moderate smokers: 15-20 cigarettes /day for atleast 5 years or more.

d) Heavy smokers: more than 20 cigarettes/ day for at least 5yearsor more.

Exclusion criteria

Subjects having history of diabetes mellitus, nephrotic syndrome, alcoholism, hypertension were excluded.

• Subjects on HMG COA reductase inhibitors, fibric acid derivatives, nicotinic acid, beta-blockers, diuretics. **Outcome measures:** All variables were seen in smokers and no-smokers

- TC
- TG
- HDL
- LDL

After overnight fasting following laboratory investigations were done in all subjects serum TC, serum HDL, serum LDL, serum TG.

Total cholesterol and triglyceride level estimations are carried out using enzymatic kit method. HDL-C is estimated by precipitation of non HDLlipoprotein and estimation done using supernatant.

LDL-Cholesterol was calculated from the formula:

LDL-C = TC-HDL TG/2.2 [mg/dL]

Fresh, clear, un-hemolyzed lithium heparinized 5-10 mL of serum was used as specimen.

Data was analyzed on SPSS version 16. t test was used between two groups for significant comparison.

Results

In this study 200 male smokers and 200 male nonsmokers between ages 18-45 years were included. In present study 11% of subjects were between age group 18-22 years, 40% were between 23-33 years and 51% were between 34-44 years. Out of 200 smoker subjects 84 (42%) smokes less than 10 cigarettes per day, 88 (44%) were moderate smokers while 28 (14%) were heavy smokers.

In this study mean TC in non-smokers was 138.12 ± 27.56 , in smokers 174.60 ± 24.46 and p value was <0.5. Mean TG for non-smoker was 102.87 ± 25.24 while in smokers it was 130.77 ± 19.62 and p value was <0.01. In this study mean LDL for non-smokers was 74.94 ± 24.37 , in smokers 116 ± 13.0 and p value was <0.001. In our study mean HDL for non-smokers was 44.84 ± 7.65 , in smokers 32.51 ± 5.89 and p value was <0.01. The relationship of plasma lipid abnormalities in nonsmoker and smoker in regard of cigarette smoked per day is shown in Fig. 1.



Fig.1. Lipid Profile.

Discussion

In current study we analyzed that TC,TG, LDL all were significantly increased and HDL was significantly decreased in accordance to the number of cigarettes smoked per day. The lipid profile abnormalities are more pronounced in heavy smoker than in mild smokers. The similar observation was reported by Devaranavadgi *et al.*, (2012), Meenakshi Sundaram *et al.*, (2010) and Imamura *et al.*, (1996). Study done by Brishetto *et al.*, (1983) demonstrated decrease in TC but there was no significant decrease in HDL-C, and there was no significant increase in LDL-C and TG in heavy smokers. This finding is contrary to present study.

Several studies have shown an association between smoking and altered serum lipid profile but many of these have lacked enough statistical power to establish firm association.

In our study total cholesterol for non smokers was 138.12 ± 24.56 mg/dL while for smokers was 174.60 ± 24.46 mg/dL and p value of two group is <0.05 which is similarly reported by Sinah *et al.*, (1995) and Neki *et al.*, 2002). In our study mean TGs in non-smokers was 102.87+30.24 mg/dl and in smoker group it was 130.77 + 19.62 mg/dL

(p<0.001). This result is consistently reported by Craig *et al.*, (1989), Neki *et al.*, (2002), Jaleel *et al.*, (2007), Ayaori *et al.*, (2000), Rastogi *et al.*, (1989). In contrast to our study, study done by Sinha *et al.*, (1995) demonstrated mean TG level in smokers was 170.8 \pm 59.7mg/dL (p<0.01).

In our study mean LDL cholesterol was 74.94 ± 24.37 mg/dl in non smokers while in smokers it was 161.11 ± 13 mg/dl (p value is <0.001) similarly reported by Craig *et al.*, (1989) and Rastogi *et al.*, (1989) But study done by Sinha et al showed less significant results with mean LDL C in smoker group was 100.2 ± 31.0 mg/dL and p value was <0.05. Another study done by Neki *et al.*, (2002) also showed less significant results for LDL C, with mean LDL C in smokers was 103.7 ± 29.16 mg/dL and non-smokers was 87.0 ± 17.80 and p value was <0.05.

Mean HDL cholesterol in non-smokers was $32.51 \pm 5.89 \text{ mg/dL}$ while in smoker it was $44.84 \pm 7.65 \text{ mg/dL}$ (p <0.01). It is comparable with the studies done by Adedeji and Etukud (2007), Neki *et al.*, (2002) but Jaleel *et al.*, (2007)

reported mean HDL C in smokers was 0.77 ± 0.12 mg/dl and in non-smokers was 0.96 ± 0.10 with less significant p value <0.05.

Conclusion

Cigarette smoking is associated with dyslipidemia with significant decrease in HDL-C and increase in TC, TG, and LDL-C. These abnormalities are directly proportional to the number of cigarette smoked per day.

Refrences

- Adedeji, O.A. and Etukudo, M.H. (2006). Lipid profile of cigarette smokers in Calabar municipality. *Pakistan Journal of Nutrition*. 5(3); 237-238.
- Ayaori, M., Hisada, T., Suzukawa, M., Yoshida, H., Nishiwaki, M., Ito, T. and Nakamura, H. (2000). Plasma levels and redox status of ascorbic acid and levels of lipid peroxidation products in active and passive smokers. *Environmental Health Perspective*. 10892, 105.
- Barua, R.S., Ambrose, J.A., Reynolds, E.L.J., DeVoe, M.C., Zervas, J.G., and Saha, D.C. (2002). Heavy and light cigarette smokers have similar dysfunction of endothelial vasoregularity activity An in vivo and in the vitro and in vitro correlation. *Journal of American College of Cardiology*. 39(11); 1758-1763.
- Brischetto, C.S., Connor, W.E., Connor, S.I., and Matarazzo, J.D. (1983). Plasma lipid and lipoprotein profiles of cigarette smokers from randomly selected families: enhancement of hyperlipidemia and depression of high density lipoprotein. *The American Journal of Cardiology*. 52(7); 675-680.Craig, W.Y., Palomaki, G.E., and Haddow, J.E. (1989). Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. BMJ. 298 (6676); 784.
- Devaranavadgi, B. B., Aski, B.S., Kashinath, R.T., and Hundekari, I.A. (2012). Effect of cigarette smoking on blood lipid- A study in Belgaum, Northern Karnataka, India. *Global Journal of Medical Research*. 126(6).
- Guindon, G.E. and Boisclair, D. Past. (2003). Current and future trends in tobacco use.
- Imamura, H., Tanaka, K., Hirae, C., Futagami, T., Yoshimura, Y., Uchida, K., and Kobata, D. (1996). Relationship of cigarette smoking to blood pressure and serum lipids and lipoproteins in men. *Clinical and Experimental Pharmacology Physiology*. 23(5); 397-402.
- Jaleel, A., Jaleel, F., Majeed, R., and Alam, E. (2007). Leptin and blood lipid level in smokers and ex smokers. WASJ. 2(4); 348-352.
- Mackay, J. and Eriksen, M.P. (2002). The Tobacco Atlas. World Health Organization.Meenakshisundaram, R., Rajendiran, C., and Thirumalaikolundu subramanian, P. (2010). Lipid and lipoprotein profiles among middle aged male smoker: a study from southern India. TOBINDUC DIS. 8 (11).
- Neki, N.S. (2002). Lipid profile in chronic smokers. A clinical study. Journal of Immunology and Clinical Microbiology. 3(1); 51-54.
- "Pakistan's tobacco consumption: The most in South Asia!" Pakistan Today. Retrieved 17 June 2014.
- Rastogi, R., Shrivastava, S.S., Mehrota, T.N., Singh, V.S. and Gupta, M.K. (1989). Lipid profile in smokers. Journal of Association of Physicians of India. 37(12); 764.
- Saengdith P. Effects of cigarette smoking on serum lipid among priestsin Bangkok. *Journal of Medical Association Thai*. 2008: 91(1); S41-4.
- Sagcan, A., Akin, M., Omay, B., Nalbantgil, S., and Sekuri, C. (2003). In-vitro- Response of platelet Aggregation Induced by Various Agonists in Chronic Smoking Coronary Artery Disease Patients. *Journal of Clinical and Basic Cardiology*. 6 (1); 55-57.
- Sinha, A.K., Misra, G.C., and Patel, D.K. (1995). Effect of cigarette smoking on lipid profile in the young. *Journal* of Association of Physicians of India. 43(3); 185-188.
- U.S. Department of Health and Human Services (2010). How Tobacco Smoke Causes Disease: What It Means to You. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. [accessed 2015 Oct 5].
- U.S. Department of Health and Human Services. The. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2014 [accessed 2015 Oct 5].

World Health Organization (WHO, 2011). Global Status Report on Non-Communicable Diseases. Geneva.

Zevin, S., Saunders, S., Gourlay, S.G., Jacob, P. and Benowitz, N.L. (2001). Cardiovascular effects of carbon monoxide and cigarette smoking. *Journal of the American College of Cardiology*. 38(6); 1633-1638