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Original research article :

The Comparative study of Homocysteine level and its association with lipid profile in smoker and Non smoker healthy adults of Udaipur city

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ABSTRACT

Background: Smoking is strongly and independently associated with cardiovascular disease and is the biggest single avoidable health habit contributing to chronic diseases in the western world.
Objectives: The objective of this study is to find out the correlation of lipid profile & Homocysteine and their comparison in smoker and non-smoker.

Methodology: Total 300 patients were include in this study, in which patients ranging within age 20-40 years, they were further categorized according to use of tobacco smoking. Group A- This group consist of tobacco smoker patients between ages 20- 40 years. (n=150).Group B-This group consist of tobacco non-smoker patients between ages 20-40 years. (n=150). 10 ml blood was drawn through vein puncture. From all collected blood samples serum lipid pofile, Liver function test and Homocysteine levels measured. All collected data were analysed statistically to calculate p value to see the difference of significance.
Results: The Mean concentration of Total cholesterol in smoker group was 205.31 ±39.47 while that of Non smoker control group 189.69 ±36.81 and the difference among them found to be highly significant .The Mean concentration of S.HDL in smoker group was 35.3±7.89 and 42.56±39.85 in Nonsmoker group respectively and the difference among them found to be highly significant. similar to that S.LDL concentration in soker group was 128 ±30.57 and in non smoker group 115.31 ±26.27 and difference among them found to be highly significant . Serum Triglyceride concentration in case group was 170.10±25.60while that of control group 147.21 ± 67.36 and p value is <0.001 so the difference among them is also significant.The Mean concentration of S.homocysteine in smoker

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group was 27.32±17.41 and 6.66±3.09 in control group and the difference among them found to be highly significant.

Conclusion: In conclusion, smoking produces adverse effects on lipid profile and homocysteine, thus increasing the cardiovascular disease risk. Further studies are needed to establish that smoking-related alterations have influences on the atherosclerotic lesions of smokers.

Keywords: Smoker, Homocystein, Lipid Profile,CVD

INTRODUCTION

In developing countries like India percentage of population indulged in smoking has been increasing day by day causing morbidity and mortality in old as well as young population by putting smokers at higher risk of atherosclerosis leading to cardiovascular and cerebrovascular accidents. Many mechanisms have been considered regarding altered lipid profile in chronic smokers. Smoking is associated with increased homocysteine level in blood which causes oxidative modification in LDL cholesterol and decrease in HDL cholesterol.[1,2]

Clinical, case–control and cohort studies indicate a graded relationship between an increasing level of plasma homocysteine and risk of cardiovascular disease[3,4]. Suggestions that the relationship may be causal have been tempered by the observation that the relationship may be stronger in case–control observation than in the methodologically stronger cohort studies[5] although the appropriateness of combining a small number of heterogeneous cohort studies has been questioned[6]. It is clear that the very high homocysteine concentrations seen with inborn errors of the enzymes involved in homocysteine metabolism do cause a primary thrombotic disorder. However, whether the moderate hyperhomocysteinaemia, more commonly seen in association with cardiovascular disease, is a cause or an effect of the disease awaits the outcome of ongoing placebo-controlled interventional trials with homocysteine lowering vitamin Smoking is strongly and independently associated with cardiovascular disease and is the biggest single avoidable health habit contributing to chronic disease in the Western world. Up to 50% of avoidable deaths in the industrialized world have been attributed to smoking, half of which are cardiovascular[7,8].

Cigarette smoking is known to be associated with a raised plasma homocysteine level[9]. Smokers also tend to have lower levels of the B-vitamins, folate, vitamin B6 and vitamin B12[10,11], all of which affect homocysteine levels by acting as co-factors (vitamins B6 and B12) or co-substrate (folate) for the enzymes controlling homocysteine metabolism[12,13]. Despite these observations, little information is available on the effect of homocysteine on the risk of cardiovascular disease in smokers, apart from a single report from our group

METHODS

This cross sectional study was conducted under the central laboratory of Biochemistry Department of RNT medical college & hospital, Udaipur. Samples were collected from general OPD and medical students in RNT medical hospital.

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Study design and criteria- Total 300 patients were include in this study, in which patients ranging within age 20-40years, they were further be categorized according to use of tobacco smoking.

Group A- This group consist of tobacco smoker patients (case) between ages 20- 40 years. (n=150).Group B-This group consist of tobacco non-smoker patients (control) between ages 20-40 years. (n=150)

Smoking questionnaire :A questionnaire was conducted on every participant by face-to-face interview, to obtain their smoking status by asking whether they smoked or not. If the answer was "yes", further information was needed to provide on the duration of smoking and the number of cigarette smoked per day. "Smoking" was defined by answers of having smoked more than 100 cigarettes in participant's lifetime, almost having smoked at least 3 to 4 consecutive months or having smoked one year or more altogether.

The proposed study was done as per pre laid Performa. All participants were questioned and the information was noted on the printed Performa.

Inclusion criteria- Patients aged between 20-40 years. A detailed family history of enrolled candidates. Tobacco smoker patients do not suffer from any other disease.The patients was diagnose based on clinical examination, laboratory investigations and other test.

In this study the BMI (BMI= Weight in Kg/ height in m^2), lifestyle, area, socioeconomic status and diet, religion and tobacco smoking habit of the enrolled participants was noted. The adults were obtain after applying the exclusion criteria.

Exclusion criteria- Pathophysiological status- Renal failure, congestive heart, heart disease, chronic respiratory diseases, liver disease, malabsorption syndrome and nutrional anemia's. Systemic disease Hypertension and diabetes mellitus.Supplementation of vitamins. Modified physiological status- Pregnancy, psychological & mental disorderssuch as depression.

Sample collection- 10 ml Venous Blood sample was collected.
Samples were incubated & centrifuge at 3000 rpm for 15 min and serum was separated from all blood samples to analyse various Biochemical parameters Like S. Total serum cholesterol(TC),S. Triglycerides(TG),S. HDL-Cholesterol ,S. LDL-Cholesterol , Liver function test (LFT) and s. Homocysteine.

Precautions were taken to avoid hemolysis of sample.

Homocysteine was measured by Advia centure CP analyzer using principle of electro chemiluminescence immunoassay(ECLIA).

Biochemical parameter Lipid profile, Uric acid, liver function test was done on Simens RXL clinical chemistry analyzer.

Statistical analysis- The statistical analysis was performed using SPSS. All the participants were made aware about the main aim of the study and they were informed that the participation is voluntary. Written consent was taken before data collection.

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RESULTS

Study includes total 300 healthy adults of age group from 20-40 year and majority of the healthy adults in smoker group is 21-25 year age.(Table 1)

	Smokers		Non -Smokers		P-value
Age group (yrs)	**No.**	**%**	**No.**	**%**	
21-25	62.00	41.33%	40.00	26.66%	**0.001**
26-30	38.00	25.33%	58.00	38.66%	
31-35	12.00	08%	26.00	17.33%	
36-40	38.00	25.33%	26.00	17.33%	
Total	**150**	**50.00%**	**150**	**50.00%**	

Table 1: Age group (yrs) with Smokers Vs Non-Smokers

	Smokers		Non- Smokers		Total	
BMI	**No.**	**%**	**No.**	**%**	**No.**	**%**
Normal	108	72.00%	100	66.67%	218	72.67%
Over Weight	28	18.67%	40	26.67%	68	22.67%
Obese	14	9.33%	10	6.67%	14	4.67%
Total	**150**	**100%**	**150**	**100%**	**300**	**100%**

Table 2: showing BMI in Smokers and Non-Smokers

P= 0.213 (NS)

	Smokers		Non- Smokers		Total	
Education Status	**No.**	**%**	**No.**	**%**	**No.**	**%**
uneducated	14	9.33%	0	0.00%	14	4.67%
primary	38	25.33%	20	13.33%	58	19.33%

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high	30	20.00%	72	48.00%	102	34.00%
Under college	68	45.33%	58	38.67%	126	42.00%
Total	**150**	**100.00%**	**150**	**100.00%**	**300**	**100.00%**

TABLE 3: Educational status in Smokers vs Non-Smokers

P= <0.001 (HS)

The prevalence of smoking is high in educated person mostly college students as compared to uneducated persons.(Table 3)and the difference among them is found to be highly significant.

According to locality ,there is no such significant difference in rural and urban residents.(Table 4)

	Smokers		**Non- Smokers**		**Total**	
Rural : Urban	**No.**	**%**	**No.**	**%**	**No.**	**%**
rural	68	45.33%	64	42.67%	132	44.00%
urban	82	54.67%	86	57.33%	168	56.00%
Total	**150**	**100.00%**	**150**	**100.00%**	**300**	**100.00%**

Table 4: Smokers Vs Non-Smokers in Rural Vs Urban areas

P=0.642 (NS)

	Smokers	
Residence	**No.**	**%**
Occasional	56	37.33%
Regular	94	62.67%
Total	**150**	**100.00%**

Table 5: Type of Smoking in Smokers

	Smokers		**Non-Smokers**		**Total**		
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**P value**
TC(mg/dl)	205.31	39.47	174.08	25.88	189.69	36.81	<0.001
TG(mg/dl)	170.10	25.60	135.11	37.85	147.21	67.36	<0.001
HDL(mg/dl)	35.3	7.89	42.56	9.85	38.96	9.61	<0.001
LDL(mg/dl)	128.0	30.57	102.56	11.07	115.31	26.27	<0.001

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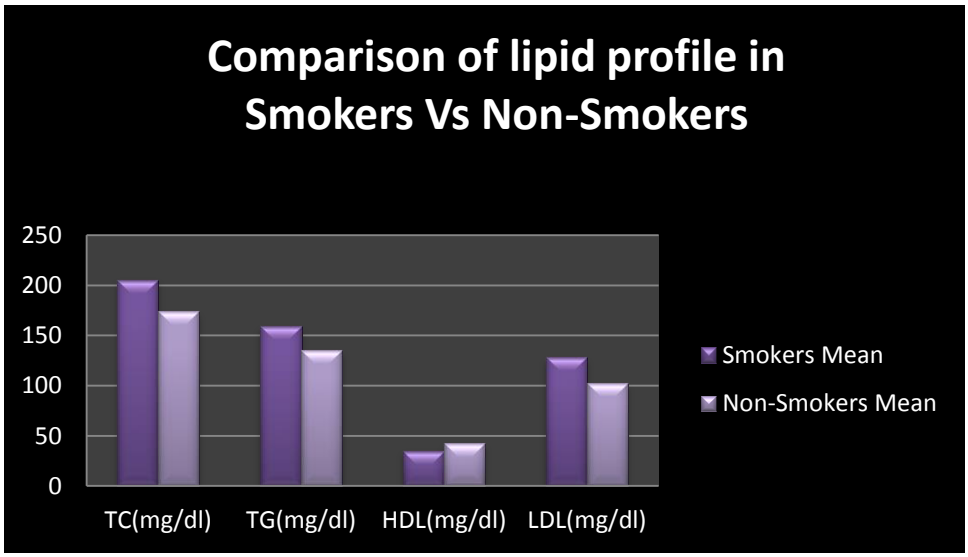
Table 6: Comparison of lipid profile in Smokers Vs Non-Smokers

TC:Total cholesterol, TG:triglyceride, HDL:High density lipoprotein, LDL: Low density lipoprotein

The Mean concentration of Total cholesterol in smoker group was 205.31 ±39.47 while that of Non smoker control group 189.69 ±36.81 and the difference among them found to be highly significant .



Graph 1 :showing Graphical presentation of lipid profile in Smokers and Non-Smokers

The Mean concentration of S.HDL in case group was 35.3±7.89 and 42.56±39.85 in case and control group respectively and the difference among them found to be highly significant. similar to that S.LDL concentration in case group was 128 ±30.57 and in control group 115.31 ±26.27 and difference among them found to be highly significant .

Serum Triglyceride concentration in case group was 170.10±25.60while that of control group 147.21 ± 67.36 and p value is <0.001 so the difference among them is also significant.

Parameter	**Smokers**		**Non-Smokers**		**Total**		**P-value**
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	
Homocysteine(µml/L)	27.32	17.41	6.66	3.06	16.99	16.21	<0.001

Table 7: Comparison of Homocysteine in Smokers Vs Non-Smokers

The Mean concentration of S.homocysteine in smoker group was 27.32±17.41 and 6.66±3.09 in control group and the difference among them found to be highly significant.

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DISCUSSION

Smoking produces adverse effects on lipid profile and homocysteine, thus increasing the cardiovascular disease risk in coronary heart disease patients.

Some investigators consider increased homocysteine levels as an independent risk factor of cardiovascular disease, while its involvement in mechanisms of thrombosis has well been documented.[13]Moreover, other studies suggest that an elevated plasma total homocysteine concentration substantially increases the risk associated with some of the conventional cardiovascular risk factors. However, there are findings that do not confirm or recognize homocysteine importance in actually causing coronary artery disease, while recent studies have considered homocysteine more as a result than a cause of arteriosclerosis, especially due to the confounding effect of various nutrient and other lifestyle-related factors.

As reported by different research groups, smoking increases the concentration of serum total cholesterol, triglycerides, LDL-cholesterol, VLDL-cholesterol and decreases the levels of antiatherogenic HDL cholesterol (Adam et al., 2011;; Kavita et al., 2013; Muscat et al., 1991). [14,15,16] Smoking, in different forms, is a major risk factor for atherosclerosis and coronary heart disease Passive smoking could affect blood lipid metabolism in women, which might contribute to coronary heart diseases (He et al., 2007) [17]. A positive association between elevated plasma total homocysteine levels and a number of cardiovascular risk factors, smoking, particularly, was shown, in a study conducted in Norway (Wilcken, 2002), to be well associated. In the comprehensive analysis, now reported from the large European Concerted Action Project case control study[18]

Muhammad afzal et.al 2011 founded that Study population consisted of thirty healthy individuals. Majority (50%) of individuals belonged to 46-55 years of age. Mean age was 51.1 years. There was male predominance. Out of total, 57% participants were smoker while43% were non-smoker. There was statistically significant association of smoking with elevated serum Homocysteine levels ($p<0.05$) Smoking appeared to be strongly associated with elevated serum Homocysteine levels in healthy individuals. [19]

Naeem afzal et al. 2010 concluded from our study that smoking is significantly associated with elevated homocysteinelevels in healthy asymptomatic adults. [20]

In general, smoking increased many lipid parameters and decreased the HDL plasma levels in CHD patients when compared to the control group. At the same time the level of homocysteine also increased in those patients. It seems that an increase in both the homocysteine and lipid profiles amplifies the risk factors toward cardiovascular diseases; this is in agreement with the findings reported by O'Callaghan and colleagues (Callaghan et al., 2002). A positive association is observed between elevated plasma total homocysteine and a number of cardiovascular risk factors including, smoking as reported by Nygard et al. (1995). [21] This is in agreement with our results. Callaghan et al. [22] showed that cardiovascular risk in smokers was markedly increased when plasma homocysteine also increased. Smokers with plasma homocysteine levels more 12 μmol./l had, a 12-fold increase of cardiovascular risk when compared with the risk in non-smokers with plasma homocysteine less than 12 μmol./l.

CONCLUSION

In conclusion, smoking produces adverse effects on lipid profile and homocysteine, thus increasing the cardiovascular disease risk. Further studies are needed to establish that smoking-related alterations have influences on the atherosclerotic lesions of smokers.

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DECLARATIONS

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Conflict of interest: None

Ethical approval: The study was done after permission of institution Ethics committee.

REFERENCES

1. World Health Assembly. Global Strategy for the Prevention and Control of Noncommunicable Diseases.WHA A53/14.Geneva, World Health Organization, 2000 (http://apps.who.int/gb/archive/pdf_files/WHA53/ea14. pdf, accessed 23 September 2010).
2. Fowdwer ,finkelstein J.D. and martin J.J, 1986 methionine metabolism in mammals
    a. ;journal of biological chemistey.
3. Jacobs, A. and Worwood, M. Ferritin in serum. Clinical and biochemical implications. N. Engi. J. Med., 1975;292: 951.
4. Lipschitz, DA., Cook, J.D. and Finch, C.A. A clinical evaluation of serum ferritin as an index of iron stores. N. Engi. J. Med., 1974; 290: 1213.
5. Gupta R, Prakash H, Majumdar S, Sharma S, Gupta VP. Prevalence of coronary artery disease and coronary risk factors in an urban population of Rajasthan. Indian Heart J 1995; 47: 331–338..
6. Wilson PWF. Established risk factors and coronary artery disease.The Framinghamstudy. Am J Hypertens 1994; 7: S7–S12.
7. Balarajan R, Bulusu L, Adlstein AM, Shukla V. Patterns of mortality among migrants to England and Wales from the Indian subcontinent. BMJ 1994; 289: 1185–1187.
8. Refsum H, Ueland PM. Homocysteine and cardiovascular disease. Annu Rev Medicine 1998; 49: 31–62.
9. Relationship between increase of serum homocysteine caused by smoking and oxidativedamage in elderly patients with cardiovascular disease Shengfang
10. Tungtrongchitr R, Pongpaew P, Soonthornruengyot M, Viroonudomphol D, Vudhivai N, Tungtrongchitr A, et al. Relationship of tobacco smoking with serum vitamin
11. B12, folic acid and haematological indices in healthy adults. Public Health Nutr 2003;6(7):67 81Chen1, Ping Wu1, Lin Zhou2,2015
12. Clarke R et al. Screening for vitamin B-12 and folate deficiency in older persons American journal of clinical nutrition, 2003, 77:1241–7

ISSN 2515-8260 Volume 09, Issue 07, 2022

13. Association of Smoking with Serum Homocysteine Level in Healthy Adults: A Cross Sectional study muhammad naeem afzal 1, mehr-ul-nisa2,2010
14. He Y, Jiang B, Wan ZH, Zheng QS and Li LS. 2007. Study on the relationship between passive smoking and blood lipids, fibrinogen and viscosity among women who never smoke. Zhonghua Liu Xing Bing Xue Za Zhi., **28**:1167-1170
15. Meenakshisundaram R, Rajendiran Cand Thirumalaikolundusubramanian, P. (2010). Lipid and lipoprotein profiles among middle aged male smokers: a study from southern India. Tobacco Induced Diseases, **8**:11-15
16. Kavita SG, Meeta GN, Priyanka MG and Gonsa R.N. 2013. Effects of smoking on lipids profile. JCRR., **5**:36-42
17. He R, Lopez D, Boreham J et al. Mortality From Smoking In Developed Countries 1950–2000. Oxford: Oxford University Press, 1994.
18. Wilcken,, Azinheira J, Reis HP, Pina JE, Correia JM, Luis AS. Influence of smoking on homocysteinemia at baseline and after methionine load. Rev Port Cardiol 2000; 19: 471–4.
19. Muhammad afzal , Hou J, Goldenberg RL, Cliver SP, Tamura T. Effect of smoking on serum concentrations of total homocysteine and B vitamins in mid-pregnancy. Clin Chim Acta 2001; 306: 103–9.
20. Naeem afzal. Increased homocysteine associated with smoking chronic inflammation and aging may reflect acute-phase induction of pyridoxal phosphatase activity. Med Hypotheses 2000; 55: 289–93.
21. Nygard v, Vollset SE, Refsum H, Stensvold I, Tverdal A, Nordrehaug JE, Ueland M and Kvale G. 1995. Total plasma homocysteineand cardiovascular risk profile. The Hordaland Homocysteine Study. JAMA., **274**: 1526–1533.