

RESEARCH ARTICLE

Quantitative assessment of cardiovascular risks in cigarette smokers using pack-years of smoking

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ABSTRACT


Background: UP is one of the biggest cultivators of tobacco. Rules of tobacco consumption are almost absent in the state. The state has almost 34% of young male smokers. **Aims and Objectives:** The idea of creating awareness among cigarette smokers and promote deaddiction among youth the present study was conducted in Muzaffarnagar Medical College to assess the cardiovascular risks according to the degree of exposure, i.e., pack-years of smoking. **Materials and Methods:** A total of 150 male subjects were taken for the study after proper consent. Five groups were formed - controls (no smoking), Group 1; 0–10 pack-years of smoking, Group 2; 11–20 pack-years of smoking, Group 3; 21–30 pack-years of smoking, and Group 4; 31–40 pack-years of smoking. Pack-years formula = (number of cigarettes smoked per day/20) × number of years smoked was used. Only cigarette smokers were used for the study after using exclusion criteria to rule out any confounding factor. Serum lipid profile and high-sensitivity C-reactive protein (hsCRP) were estimated. **Results:** As the degree of exposure is increasing, i.e. pack-years of smoking increases, the level of dyslipidemias is also becoming severe being highly significant $P < 0.01^{**}$ in 31–40 pack-years of smoking. hsCRP is significantly increasing $P < 0.05^{*}$ in Group 2 and showing highly significant $P < 0.01^{**}$ increase in Groups 3 and 4. **Conclusion:** Cardiovascular risks are increasing significantly as pack-years of smoking is increasing as indicated by dyslipidemias and hsCRP increase, means 21–30 and 31–40 pack-years are at higher risk as compared to 0–10 and 11–20 pack-years.

KEY WORDS: Cigarette; Smokers; High-sensitivity C-reactive protein; Tobacco; Cardiovascular risks; Lipid profile

INTRODUCTION

Deaddiction is the major concern among the chain smokers and so-called heavy smokers (>25 cigarettes/day) coming to chest outpatient departments. Patients are motivated by objectifying various cancer risks oral and lungs, etc., among the smokers. However, to mere surprise is the fact that neoplasia is not the major cause of mortality among the

chronic smokers, but it is the progressive atherosclerotic coronary artery disease which is taking major toll on the life of chronic smokers. To create awareness among the smokers, concept of pack-years was introduced. A pack-year is a quantification of cigarette smoking. It is used in a clinical context to measure a person's exposure to tobacco and Number of pack-years = (number of cigarettes smoked per day/20) × number of years smoked. (1 pack has mostly 20 cigarettes in India). The pack-year is a unit for measuring the amount a person has smoked over a long period of time. Tobacco smoking is the most avoidable cause of cardiovascular report according to the WHO report 2002.^[1] In the present study, we tend to assess the cardiovascular status of cigarette smokers to the degree of exposure to cigarette smoke by measuring the dyslipidemias level and high-sensitivity C-reactive protein (hsCRP) level in chronic smokers and quantifying it

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according to pack-years of smoking. The past studies have clearly established dyslipidemia as clear-cut risk factor for atherosclerotic plaque formation.^[2] The American Heart Association (AHA) has clearly stated a level of serum hsCRP >3 mg/l in otherwise healthy habitual smokers as high risk for cardiovascular morbidity. The present study was conducted in Muzaffarnagar city of Western Uttar Pradesh with a population over 40 lakh according to 2011 census has high consumption of tobacco in the form of cigarette smoking as rest of state. Most of smokers are young male adults in the age group of 18–45 years according to the National Family Health Survey conducted in late 90. Approximately 34% of male and 3.1% of females of UP are current tobacco smokers. As far as literature is concerned, a lot of studies have been done showing a positive correlation between various types of malignancies and pack-years of smoking. Hence, the present study was done to create awareness among chronic smokers by comparing their pack-years of smoking with cardiovascular risks (dyslipidemias and hCRP levels). As a conscious among common man is that light smoking (1–4 cigarettes/day) is not a risk for developing coronary artery disease (CAD), so we tend to estimate the risk of progressive occlusive atherosclerotic CAD in increasing pack-years of smoking.

The WHO predicts that 70% of death from smoking-related illness will occur in developing countries by 2020.

In this study, we tend to assess the dyslipidemias and hsCRP level of cigarette smokers by grouping them according to quantity of cigarette smoked, i.e., pack-years of smoking.

MATERIALS AND METHODS

The study was conducted in the Department of Physiology, Muzaffarnagar Medical College, Muzaffarnagar. The study was conducted on 150 male subjects in the age group of 18–45 years over a period of 1 and ½ years from January 2017 to July 2018. Of these 150 subjects, 30 were grouped as controls with no history of tobacco consumption in any form. Rest 120 otherwise healthy subjects with a history of habitual cigarette smoking were grouped into four groups, i.e. 1, 2, 3, and 4 based on their amount of cigarette smoking. Pack-year formula was used to calculate their degree of exposure, i.e., Number of pack-years = (number of cigarettes smoked per day/20) × number of years smoked.

Hence, finally, we formed five groups as follows:

- First group - No smoking - Controls
- Second group - 0–10 pack-years =1
- Third group - 11–20 pack-years =2
- Fourth group - 21–30 pack-years=3
- Fifth group - 31–40 pack-years =4

Exclusion Criteria

The subjects having a history of any cardiovascular event (angina, myocardial infarction, coronary syndrome, and stroke), known hypertensive, diabetics, renal disease, drug abuse, and chronic obstructive pulmonary disease were not taken in the study. The subjects with massive elevation hsCRP >10 mg/L were also excluded from the study.

Proper informed consent was taken in written form signed by the subjects before conducting the study.

Ethical Clearance

From the society of human experimentation was taken before starting the study.

These groups were evaluated for the following tests:

- Serum lipid profiles
- Serum hsCRP levels

Lipid profile estimation - Triglycerides (TGs) were estimated using glycerol phosphate oxidase PAP method, total cholesterol (TC) was estimated by Wybenga and Pillegi method, high-density lipoproteins (HDL) by spectrophotometry, very low-density lipoproteins (VLDL), and low-density lipoproteins (LDL) by formulas.

Serum hsCRP level was estimated using ELISA kit method.

Statistical Analysis

It was done by software SPSS 17 and Microsoft Excel. The result was expressed as mean ± SD. Each group 1, 2, 3, and 4 was separately compared with control group and measured for significance. Unpaired *t*-test was used for comparison between the groups. *P* < 0.05* was taken as statistically significant and *P* < 0.01** was taken as highly statistically significant.

RESULTS

The findings of the present study were presented in Tables 1 and 2.

DISCUSSION

Groups 1, 2, and 3 are showing a significant increase in TC and LDL-cholesterol as compared to control with *P* < 0.05*. This change becomes highly significant in Group 4 *P* < 0.01**. Groups 2 and 3 are showing a significant increase in TGs and VLDL-cholesterol, which become highly significant in Group 4. HDL-cholesterol is significantly decreased in Groups 3 and 4. These findings are in consistence with similar results in the past studies

Table 1: Correlation of lipid profiles with pack-years of smoking

Parameters	Control	1	2	3	4
TC	184±40.4	216.7±52.1*	238.07±54*	249.74±58*	370.05±62**
TG	115.9±28.2	118.1±26.2	131.8±28.5*	135.7±29.1*	147.78±28**
HDL-C	56.5±6.0	56.38±5.2	55.61±4.8	55.24±5*	54.06±5.1*
LDL-C	123±28.1	137.9±27.3*	158.4±28.6*	178.6±31*	295.87±34**
VLDL-C	23.73±5.8	24.22±5.6	27.97±5.2*	28.74±5.4**	29.86±5.7**

TC: Total cholesterol, TGs: Triglycerides, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, VLDL: Very low-density lipoprotein. All values are in md/dl. *is statistically significant with $P < 0.05$ and **highly statistically significant $P < 0.01$

Table 2: Correlation of hsCRP levels with pack-years of smoking

Parameter control	1	2	3	4
hsCRP 1.13±1.07	1.83±1.12	2.79±2.02*	3.04±2.85**	3.4±3.1**

All values are in mg/L. *is statistically significant with $P < 0.05$ and **highly statistically significant $P < 0.01$. hsCRP: High-sensitivity C-reactive protein

Nicotine present in tobacco smoke is responsible for the creation of chronic inflammatory state of Celermajer *et al.*^[13] and Burke and Fitzgerald.^[14] Tobacco smoke leads to activation of interleukin (IL)-6 and IL- β which are increased in response to lung inflammation and implicated in induction of hsCRP. These findings support the idea that induction or exacerbation of inflammation could be a mechanism by which smoking promotes atherosclerosis.

Hence, we can use hsCRP as a diagnostic marker for CAD in heavy smokers Groups 3 and 4 where the level of >3 mg/L is clearly pointing toward undergoing pathology.

As our study suggests hs CRP and lipid profiles can be advised as routine screening tool for detecting the level of cardiovascular risks in heavy and chain smokers. These tests are easy (only blood sample), less expensive & less time consuming as compared to TMTs or Noninvasive CT angiography for detecting cardiovascular status of suspected CADs. The doctors are reluctant to write TMTs etc. Because patients are not willing to spend too much unless a cardiovascular morbidity occurs.

The major limitation of the study is to clearly differentiate the groups into pack-years of smoking as many subjects cannot keep a clear-cut record of how many cigarettes they are smoking per day for a long period of time as smoking is greatly influenced by mental stress as many subjects smoked more than their average daily addiction if faced any mental stress in their lives at any point of time in their lives. It was quite difficult for us to get case of only cigarette smoking history because smoking and chewing tobacco in Muzaffarnagar is quite an overlapping habit. Most of people who smoke also chew tobacco in any form.

Recommendations

Heavy smokers (21–30 and 31–40 pack-years) should get their hsCRP level regularly checked up as a screening test for CAD as they are at maximum risk. Beside deaddiction should be promoted by stating them their risk level by measuring CRP level. Deaddiction in the form of hypnosis and acupuncture is gaining worldwide popularity.

conducted by Gupta *et al.*,^[2] Yusuf *et al.*,^[3] Khurana *et al.*,^[4] and Venkatesan *et al.*^[5] The basis of dyslipidemias in smokers is sympathomimetic effect of nicotine. Release of adrenaline leads to lipolysis by increasing activity of lipolytic lipase enzyme in adipose tissue. This leads to increase in serum concentration of free fatty acids, TG, LDL, and VLDL.

From our study, we can infer that as the degree of exposure is increasing, i.e. pack-years of smoking increases, the level of dyslipidemias is also becoming severe being highly statistically significant $P < 0.01$ ** in 31–40 pack-years of smoking.

hsCRP is statistically significantly increasing $P < 0.05$ * in Group 2 and showing highly statistically significant $P < 0.01$ ** increase in Groups 3 and 4. This is in accordance with AHA recommendation where values 1–3 recommend moderate risk and values >3 indicate high risks of CAD. These values in heavy smokers Groups 3 and 4 are just approaching very high-risk category. Tonstad and Cowan,^[6] Yanbaeva *et al.*,^[7] Wannamethee *et al.*,^[8] Lowe *et al.*,^[9] and Cushman *et al.*^[10] all have shown rise of hsCRP in smokers as compared to past or never smokers. In the past, a dose-dependent correlation between hsCRP and smoking habits was demonstrated in the Speedwell survey of British men, hsCRP levels were increased from 1.13 mg/l in never smokers to 1.87, 2.32, and 2.05 mg/l in those who smoked 1–14, 15–24, and > 25 cigarettes, respectively, day as proposed in a study of Lowe *et al.*^[9] Pack-years relation with hsCRP was given by Tracy *et al.*^[11] who reported that pack-years of smoking, but not current cigarette smoking, were positively associated with log CRP levels among 400 healthy elderly participants. Pack-year effect is also supported by Madsen *et al.*^[12]

CONCLUSION

Cardiovascular risks are increasing significantly as pack-years of smoking is increasing as indicated by dyslipidemias and hsCRP increase, means 21–30 and 31–40 pack-years are at higher risk as compared to 0–10 and 11–20 pack-years.

REFERENCES

1. World Health Organization. Report 2002 Reducing Risks, Promoting Healthy Life. Geneva: World Health Organization; 2002. p. 47-98.
2. Gupta V, Tiwari S, Agarwal CG, Shukla P, Chandra H, Sharma P, *et al.* Effect of short-term cigarette smoking on insulin resistance and lipid profile in asymptomatic adults. *Indian J Physiol Pharmacol* 2006;50:285-90.
3. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, *et al.* Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. *Lancet* 2004;364:937-52.
4. Khurana M, Sharma D, Khandelwal PD. Lipid profile in smokers and tobacco chewers a comparative study. *J Assoc Physicians India* 2000;48:895-7.
5. Venkatesan A, Hemalatha A, Bobby Z, Selvaraj N, Sathiyapriya V. Effect of smoking on lipid profile and lipid peroxidation in normal subjects. *Indian J Physiol Pharmacol* 2006;50:273-8.
6. Tonstad S, Cowan JL. C-reactive protein as a predictor of disease in smokers and former smokers: A review. *Int J Clin Pract* 2009;63:1634-41.
7. Yanbaeva DG, Dentener MA, Creutzberg EC, Wesseling G, Wouters EF. Systemic effects of smoking. *Chest* 2007;131:1557-66.
8. Wannamethee SG, Lowe GD, Shaper AG, Rumley A, Lennon L, Whincup PH, *et al.* Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. *Eur Heart J* 2005;26:1765-73.
9. Lowe GD, Yarnell JW, Rumley A, Bainton D, Sweetnam PM. C-reactive protein, fibrin D-dimer, and incident ischemic heart disease in the speedwell study: Are inflammation and fibrin turnover linked in pathogenesis? *Arterioscler Thromb Vasc Biol* 2001;21:603-10.
10. Cushman M, Arnold AM, Psaty BM, Manolio TA, Kuller LH, Burke GL, *et al.* C-reactive protein and the 10-year incidence of coronary heart disease in older men and women: The cardiovascular health study. *Circulation* 2005;112:25-31.
11. Tracy RP, Lemaitre RN, Psaty BM, Ives DG, Evans RW, Cushman M, *et al.* Relationship of C-reactive protein to risk of cardiovascular disease in the elderly. Results from the cardiovascular health study and the rural health promotion project. *Arterioscler Thromb Vasc Biol* 1997;17:1121-7.
12. Madsen C, Nafstad P, Eikvar L, Schwarze PE, Rønningen KS, Haaheim LL, *et al.* Association between tobacco smoke exposure and levels of C-reactive protein in the Oslo II study. *Eur J Epidemiol* 2007;22:311-7.
13. Celermajer DS, Sorensen KE, Georgakopoulos D, Bull C, Thomas O, Robinson J, *et al.* Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent dilation in healthy young adults. *Circulation* 1993;88:2149-55.
14. Burke A, Fitzgerald GA. Oxidative stress and smoking-induced vascular injury. *Prog Cardiovasc Dis* 2003;46:79-90.

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