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Original Article

Comparing Plasma Fibrinogen,Homocysteine, Cardiac Troponin I, and C-Reactive Protein Levels Among Smokers and Non-smokers inHamadan Hospitals: A Case-Control Study

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Abstract

Background: Cigarette smoking is one of the main risk factors for cardiovascular damage. Current studies have reported an association between some markers such as homocysteine, plasma fibrinogen, cardiac troponin I (cTnI), and C-reactive protein (CRP) and cardiovascular damages. This study aimed to investigate the relationship between these laboratory markers and smoking intensity.

Methods: In this case-control study, 200 male employees from the operating room, laboratory, and administrative departments of Hamadan hospitals were categorized into four groups of 50 based on their smoking status: active smokers, smokers, infrequent smokers, and non-smokers. After sampling, blood levels of the specified biomarkers were evaluated. Then, regression analysis was performed to explore the relationship between these markers and cigarette smoking, regression analysis was performed.

Results: The results indicated significant differences in the studied biomarkers between smokers and non-smokers ($P<0.001$), suggesting the effect of cigarette smoking on cardiovascular damage, inflammation, and coagulation states in individuals.

Conclusions: Cigarette smoking affects markers of heart muscle damage, inflammatory and coagulation factors, and cardiovascular
risk factors.

Keywords: Homocysteine, Fibrinogen, Cardiac troponin I, C-reactive protein, Cardiovascular damage, Smoking

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Introduction

Today, cigarette smoking, with its numerous side effects and increasing prevalence, has turned into one of the important health concerns worldwide. According to previous studies, one-third of the global population aged 15 or above has been involved in cigarette smoking. Nevertheless, the cigarette smoking rate among men was three to four times higher than in women (1). According to the US National Health Survey, 15.01% of adults had a history of smoking in 2015. Cigarette smoking leads to issues such as cancer, respiratory problems, and cardiovascular diseases (2). It is a significant risk factor for cardiac diseases, and Ding et al found a significant effect of smoking cessation on mitigating the risk of certain cardiovascular diseases (3). Nevertheless, more precise investigations are required in this regard.

Current studies suggest a relationship between changes in plasma concentration of homocysteine, cardiac troponin I (cTnI), and C-reactive protein (CRP) with incidence of cardiac damage (4-6). Nevertheless, no study has explored the relationship between these markers and cigarette smoking. Understanding this relationship can clarify the general mechanism of the effect of smoking on the cardiovascular system (7,8). The present study compared the levels of homocysteine, fibrinogen, cTnI, and CRP in smokers and non-smokers in the operating room, laboratory, and administrative departments of Hamadan hospitals.

Materials and Methods

Study Population

This case-control study comprised 200 men employed in different departments (operating room, laboratory, and administration) of Hamedan city hospitals, with a mean age of 45 ± 20 years. Participants had no history of cardiac, liver, renal, thyroid disease, inflammation, hyperlipidemia, hyperglycemia, obesity, or hypertension. They were categorized into four groups with equal numbers according to cigarette smoking status:

1. Active smokers: This group included 50 individuals smoking more than 15 cigarettes for at least five years.
2. Smokers: This group included 50 individuals smoking 5-15 cigarettes per day.
3. Infrequent smokers: This group involved 50 individuals smoking less than five cigarettes per day
4. Non-smokers: This group contained 50 individuals with no history of cigarette smoking.

Sampling and Tests

Specifically, 10 mL of blood sample was taken from each subject in their fasting state and mixed with EDTA anticoagulant with a concentration of 0.47 mol/L. Plasma was separated by 1000g centrifugation, and biochemical tests were performed in the laboratory of the Paramedicine Faculty of Hamadan University of Medical Sciences (Table 1).

Sample Size Calculation

Based on the case-based nature of this study and according to the formula, the sample size was calculated as 200 (50 subjects per group).

Statistical Methods

ANOVA was used to compare different levels across groups, and the Tukey test was employed to provide further details. To explore hazard ratios in each class, relative risk was calculated. Furthermore, the relationships between the main variables were explored using regression analysis. Data were analyzed by SPSS and S-Plus. During the statistical calculations, the extent of cigarette smoking was considered as the independent variable, while the levels of homocysteine, CRP, fibrinogen, and cTnI were regarded as the dependent variables.

Results

In the present study, 200 men employed in the operating room, laboratory, and administrative departments of Hamedan hospitals were investigated, with a mean age of 45 ± 20 years. The obtained results are reported in Table 2. Statistical regression analysis indicated a significant relationship between smoking status and CRP, cTnI, homocysteine, and fibrinogen, as depicted in Table 3. Additionally, regression analysis was conducted to determine the relationship between each of the studied markers (Table 4).

Discussion

The present study investigated CRP as the marker of inflammatory diseases, cTnl as a marker of myocardial damage, homocysteine as a risk factor for cardiovascular disease, and fibrinogen as a prognostic factor for thrombosis and fibrinogenesis.

According to the statistical analyses, CRP value revealed a significant difference between active smokers and smokers compared to non-smokers. This suggests the definitive and harmful effect of smoking more than five cigarettes daily on the inflammatory status of the body and

Table 1. List of biochemical tests performed on the samples

Test Method/Kit	Test
Enzyme method, Boehringer-Mannhein	Total cholesterol
Enzyme method, Boehringer-Mannhein	HDL cholesterol
Enzyme method, Boehringer-Mannhein	Triglyceride
Friedewald method	LDL Cholesterol
ELISA kit, Omega	CRP
ELISA kit, Veda-Lab	cTnI
Immunoassay enzyme kit, Axis-shield	Homocysteine
Coagulation method, manual kit	Fibrinogen

Note. LDL: Low-density lipoproteins; HDL: High-density lipoproteins; cTnI: Cardiac troponin I; CRP: C-reactive protein; ELISA: Enzyme-linked immunosorbent assay.

Table 2. The Measured Results of the Tested Factors in the Study Population

Variable	Group			
	Active Smoker	Smoker	Infrequent Smoker	Non-smoker
Age	45 ± 11	40 ± 7	43 ± 10	37 ± 9
Total cholesterol (mg/dL)	184 ± 9.5	177 ± 8	177 ± 5.5	174 ± 8.5
LDL (mg/dL)	117 ± 20	111 ± 8	109 ± 9	105 ± 13
HDL (mg/dL)	45 ± 8.5	41 ± 9.2	38.5 ± 10	38 ± 7.7
Triglyceride(mg/dL)	123 ± 9.5	118 ± 15	110 ± 11	108.5 ± 4
CRP (mg/dL)	14.2 ± 0.7*	11 ± 0.9*	6.5 ± 0.9	5.5 ± 0.5
cTnI (µg/L)	0.47 ± 0.17*	0.27 ± 0.08*	0.71 ± 0.04*	0.47 ± 0.01
Homocysteine (µmol/L)	9.92 ± 0.3*	9.25 ± 0.3*	8.77 ± 0.4	8.62 ± 0.1
Fibrinogen (mg/dL)	277.7 ± 11*	247.2 ± 7.7*	229.5 ± 5.2*	216 ± 8.4

Note. LDL: Low-density lipoproteins; HDL: High-density lipoproteins; cTnI: Cardiac troponin I; CRP: C-reactive protein. The data are in the form of mean ± standard deviation. * indicates the significance of the difference with the non-smoker group with a *P* value less than 0.001.

Table 3. Statistical regression analysis indicated a significant relationship between smoking status and CRP, cTnI, homocysteine, and fibrinogen

Marker	χ^2
CRP	21
cTnI	25
Homocysteine	18
Fibrinogen	25

Note. cTnI: Cardiac troponin I; CRP: C-reactive protein. Table 3 ($P < 0.001$)

Table 4. The regression analysis was conducted to determine the relationship between each of markers

r	Factor 2	Factor 1
0.44	cTnI	CRP
0.32	Homocysteine	CRP
0.59	Fibrinogen	CRP
0.42	Homocysteine	cTnI
0.42	Fibrinogen	cTnI
0.52	Fibrinogen	Homocysteine

Note. cTnI: Cardiac troponin I; CRP: C-reactive protein. $P<0.001$.

cardiac health. This finding is confirmed by other studies such as Sasaki et al who also indicated higher CRP values in smokers compared to the control group (9). Likewise, the findings of Alsharairi and Small et al concur with the present findings (10,11).

Furthermore, investigation of cTnI and fibrinogen values of the studied subjects indicated a significant increase in these markers among all individuals with a history of cigarette smoking compared to non-smokers. This indicates the detrimental effect of cigarette smoking, even at low levels, on the myocardium and coagulation state.

The significant alteration of homocysteine levels in active smokers and smokers also indicated an increased risk of incidence of cardiac damage for those smoking more than five cigarettes per day. Current studies confirm these findings. According to O'Callaghan et al, smokers are up to twice as susceptible to developing cardiovascular disease due to higher levels of risk factors such as homocysteine compared to non-smokers (12). Research by Goodarzi et al found that the plasma fibrinogen, cTnI, and homocysteine levels among smokers are 1.5, 5, and 2 times higher than those in non-smokers, respectively (13). Additionally, the study by Kawada indicated the effect of cigarette smoking on the elevation of plasma fibrinogen in both men and women (14).

The study by Morrow et al revealed higher values of F2-isoprostane, a biomarker resulting from the reaction of free radicals with biomolecules, among smokers. This increased the reaction of free radicals with biomolecules, ischemic damage to the vascular endothelial cells, and synthesis of prostaglandins, intensified platelet activity, and thereby elevated the plasma fibrinogen concentration. Meanwhile, the endothelial ischemic damage leads to cardiac tissue necrosis and elevated cTnI levels. Ultimately, all these factors resulted in an inflammatory state and elevated CRP levels. This augmentation has also been confirmed by Low et al (15), Goli et al (16), and Safari et al (17).

Conclusion

Based on the results of the present study, cigarette smoking significantly elevated CRP, cTnI, homocysteine, and fibrinogen levels. Nevertheless, the status of smoking affected these markers, and smoking more than five cigarettes daily elevated CRP and homocysteine levels, while any degree of smoking significantly increased cTnI and fibrinogen levels. This suggests the noticeable effect of cigarette smoking on changes in inflammatory and coagulation states, as well as myocardial health. Cigarette smoking affects markers of heart muscle damage, inflammatory and coagulation factors, and cardiovascular risk factors.

Author's Contribution

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Competing Interests

The authors declare that they have no conflict of interests.

Ethical Approval

This study was approved by the Research Ethics Committeem of Hamadan University of Medical Sciences (IR.UMSHA.REC.1386.199).

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References

1. Ishida M, Sakai C, Kobayashi Y, Ishida T. Cigarette smoking and atherosclerotic cardiovascular disease. J Atheroscler Thromb. 2024;31(3):189-200. doi: 10.5551/jat.RV22015.
2. Mallah MA, Soomro T, Ali M, Noreen S, Khatoon N, Kafle A, et al. Cigarette smoking and air pollution exposure and their effects on cardiovascular diseases. Front Public Health. 2023;11:967047. doi: 10.3389/fpubh.2023.967047.
3. Ding N, Shah AM, Blaha MJ, Chang PP, Rosamond WD, Matsushita K. Cigarette smoking, cessation, and risk of heart failure with preserved and reduced ejection fraction. J Am Coll Cardiol. 2022;79(23):2298-305. doi: 10.1016/j.jacc.2022.03.377.
4. Safari MR, Omrani MD, Ghaderian SM, Sayad A, Taheri M. A

case/control study of evaluating the serum levels of new risk factors for cardiovascular diseases in Iranian patients. J Biol Todays World. 2017;6(1):1-5. doi: 10.15412/j.jbtw.01060101.
5. Goli Z, Mashayekhi S, Taheri M, Mazdeh M, Safari MR. Determination of plasma malondialdehyde values as a laboratory index of lipid peroxidation reaction in multiple sclerosis patients and its comparison with normal people. Avicenna J Care Health Oper Room. 2023;1(3):90-3. doi: 10.34172/ajchor.34.
6. Arhip L, Brox-Torrecilla N, Romero I, Motilla M, Serrano-Moreno C, Miguélez M, et al. Late-onset methylmalonic acidemia and homocysteinemia (cblC disease): systematic review. Orphanet J Rare Dis. 2024;19(1):20. doi: 10.1186/s13023-024-03021-3.
7. Xu Q, Zhu C, Zhang Q, Hu Z, Ji K, Qian L. Association between fibrinogen-to-albumin ratio and prognosis of patients with heart failure. Eur J Clin Invest. 2023;53(10):e14049. doi: 10.1111/eci.14049.
8. Yan I, Börschel CS, Neumann JT, Sprünker NA, Makarova N, Kontto J, et al. High-sensitivity cardiac troponin I levels and prediction of heart failure: results from the BiomarCaRE consortium. JACC Heart Fail. 2020;8(5):401-11. doi: 10.1016/j.jchf.2019.12.008.
9. Sasaki A, Kondo K, Sakamoto Y, Kurata H, Itakura H, Ikeda Y. Smoking cessation increases the resistance of low-density lipoprotein to oxidation. Atherosclerosis. 1997;130(1-2):109-11. doi: 10.1016/s0021-9150(96)06051-0.
10. Alsharairi NA. Antioxidant intake and biomarkers of asthma in relation to smoking status-a review. Curr Issues Mol Biol. 2023;45(6):5099-117. doi: 10.3390/cimb45060324.
11. Small AM, Pournamdari A, Melloni GEM, Scirica BM, Bhatt DL, Raz I, et al. Lipoprotein(a), C-reactive protein, and cardiovascular risk in primary and secondary prevention populations. JAMA Cardiol. 2024;9(4):385-91. doi: 10.1001/jamacardio.2023.5605.
12. O'Callaghan P, Meleady R, Fitzgerald T, Graham I. Smoking and plasma homocysteine. Eur Heart J. 2002;23(20):1580-6. doi: 10.1053/euhj.2002.3172.
13. Goodarzi MT, Rezaei M, Moadel E, Homayounfar S, Safari MR. Changes in sialylation of low-density lipoprotein in coronary artery disease. Eur J Lipid Sci Technol. 2008;110(4):302-6. doi: 10.1002/ejlt.200700189.
14. Kawada T. C-reactive protein, fibrinogen, and incident heart failure: combination procedure for statistical analysis. J Clin Hypertens (Greenwich). 2013;15(4):298. doi: 10.1111/jch.12076.
15. Low AF, Seow SC, Yeoh KG, Lim YT, Tan HC, Yeo TC. High-sensitivity C-reactive protein is predictive of medium-term cardiac outcome in high-risk Asian patients presenting with chest pain syndrome without myocardial infarction. Ann Acad Med Singap. 2004;33(4):407-12.
16. Goli Z, Mashayekhi S, Mazdeh M, Taheri M, Zafari S, Safari MR. Comparison of selenium serum levels in patients with multiple sclerosis and normal individuals. Avicenna J Care Health Oper Room. 2023;1(1):7-10. doi: 10.34172/ajchor.30.
17. Safari MR, Sheikh N, Mani Kashani K. Study on the effect of vitamin C on the In vitro albumin glycation reaction. Iran J Pharm Res. 2006;5(4):275-9.