ISSN: 2309-5288 (Print)
ISSN: 2309-6152 (Online)
CODEN: JCLMC4

Journal of Coastal Life Medicine

An Analysis of How Smoking Affects Assessments for the Cardiovascular and Autonomic Functioning: A Tertiary Care Study.

Received: 23 October 2022, **Revised:** 14 November 2022, **Accepted:** 26 December 2022

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Key Words:
Smoking, cardiovascular function, autonomic function, heart rate variability, oxidative stress

Abstract:

Introduction: Smoking is a significant risk factor for CVD, however it is unclear how smoking affects tests of cardiovascular and autonomic functioning at tertiary care facilities.

Methods: In a tertiary care context, this study looked at the relationship between smoking and tests of cardiovascular and autonomic functioning. 200 people took part in the study, 100 of whom were smokers and 100 were not. Blood pressure, heart rate, cholesterol levels, and autonomic function were assessed in the subjects.

Results: Smokers had a higher mean BMI than non-smokers ($p < 0.05$). Other baseline characteristics were similar between groups. Smokers had greater systolic, diastolic, and heart rates than non-smokers ($p<0.05$). Smokers had significantly higher HDL and LDL cholesterol than non-smokers ($p < 0.05$). Other comparisons between the groups indicated no changes. After correcting for potential confounders, smokers had higher risks of elevated SBP (OR=2.34, 95% CI: 1.21-4.54), DBP, and HR than non-smokers. Smokers had a higher risk of low HDL cholesterol (OR=2.64, 95% CI: 1.49-4.67) and high LDL cholesterol (OR=2.49).

Conclusion: Smoking causes problems with the heart and the autonomic nervous system. There may be a dose-dependent relationship between smoking's detrimental effects on cardiovascular and autonomic function, with heavier smokers reporting more severe impairments. To lower the risk of CVD and other harmful cardiovascular events, healthcare practitioners should encourage smokers to quit.

1. Introduction

Smoking is a major cause of unnecessary deaths and a global public health concern [1]. Numerous illnesses, including “chronic obstructive pulmonary disease (COPD)”, “cardiovascular disease (CVD)”, and other cancers have been linked to smoking [2]. Smoking significantly increases the chance of developing CVD which is the main cause of morbidity and mortality worldwide [3].

Smoking is known to have an impact on the cardiovascular and autonomic systems, which are crucial for preserving healthy levels of blood pressure, heart rate, and vascular tone. The "autonomic nervous system (ANS)" regulates the cardiovascular system through regulating blood pressure, vascular resistance, and heart rate [4]. The two branches of the ANS are the sympathetic and parasympathetic nervous systems. The parasympathetic neural system mediates the "rest and digest" response while the sympathetic nervous system mediates the "fight or flight" response [5].

Smoking is known to have an impact on the ANS, which can result in a number of cardiovascular conditions. Smoking has been linked to higher heart rate, blood pressure, and vascular resistance as well as worse arterial compliance, heart rate variability (HRV), and baroreflex sensitivity, according to studies [6]. Additionally, smoking has been proven to enhance oxidative stress and inflammation, which can

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result in atherosclerosis and dysfunction of the endothelium [7].

Cardiovascular and autonomic functional evaluations are essential for the diagnosis and treatment of CVD. The LiDCO system and the ANSWatch gadget are two methods for evaluating cardiovascular and autonomic functioning that have been developed. The LiDCO system is a non-invasive method for measuring systemic vascular resistance, cardiac output, and stroke volume [8]. The ANSWatch gadget is a non-invasive method for measuring sympathetic and parasympathetic activity, baroreflex sensitivity, and HRV [9].

There are few research looking at how smoking affects tests for cardiovascular and autonomic functioning, despite the evidence connecting smoking to cardiovascular and autonomic dysfunction. This study was done to ascertain how smoking affected patients receiving tertiary care's assessments of their cardiovascular and autonomic functioning.

The results of this study have significant ramifications for CVD management and prevention. If it is discovered that smoking significantly impairs tests of cardiovascular and autonomic functioning, it emphasises the significance of quitting smoking for the therapy and prevention of CVD. Quitting smoking is known to minimise the risk of CVD and enhance cardiovascular and autonomic functions [10]. As a result, this research may shed light on the advantages of quitting smoking for patients receiving tertiary care. The study will underline the significance of quitting smoking for the prevention and management of CVD and offer significant new insights into the potential effects of smoking on cardiovascular and autonomic functioning.

2. Materials and Methods

Study Design & subjects:

This cross-sectional study was done in India at a tertiary care facility. Between 2021and 2022, the study was carried out. Patients who were sent to the cardiology division for evaluation of their cardiovascular and autonomic functions made up the study population. Patients between the ages of 25 and 60 who had no prior history of CVD or any other chronic illnesses, such as diabetes or hypertension, met the inclusion criteria. Patients with a history of quitting smoking within the previous six months, those who were pregnant or nursing, and those who had a contraindication for the tests of cardiovascular and autonomic functioning were all excluded from the study. Ethical clearance was obtained for the study and consent was taken from the subjects.

Determine the sample size:

The formula used to determine the sample size was

"n = [(Z/2 + Z)2 (p1 (1 - p1) + p2 (1 - p2)] / (p1 – p2)"

Where n is the sample size for each group, Z/2 is the level of significance's crucial value for a two-tailed test, Z is the test's power, and p1 and p2 are the proportions of smokers and non-smokers having aberrant results for cardiovascular and autonomic functioning, respectively. We calculated the proportion of smokers and non-smokers with aberrant assessments for cardiovascular and autonomic functioning to be 60% and 30%, respectively, based on the prior research and assuming a power of 80% and a significance threshold of 5%. Thus, we determined that 75 patients each group would be the appropriate sample size.

Data gathering

The cardiology department was used to recruit research volunteers in succession. All participants provided written informed consent after being told of the study's goals and methods by the patients. All participants provided a thorough medical history, and a physical examination was performed to make sure the inclusion and exclusion criteria were met.

Using a standardised questionnaire, smoking status was evaluated. The questionnaire asked about current and prior smoking status, daily cigarette consumption, and length of smoking. Smokers were defined as patients who reported smoking at least five cigarettes per day for more than a year. Non-smokers were defined as patients who said they had never smoked or had smoked fewer than five cigarettes per day for less than a year.

The LiDCO system and the ANSWatch gadget, respectively, were used to assess the autonomic and cardiovascular systems. The cardiac output, stroke volume, and systemic vascular resistance were all

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measured using the LiDCO system. HRV, baroreflex sensitivity, sympathetic and parasympathetic activity were all measured using the ANSWatch gadget.

All participants' blood was drawn in order to test endothelial function and oxidative stress indicators. The oxidative stress indicators were “glutathione peroxidase (GPx)” and “malondialdehyde (MDA)”. GPx is an enzyme that scavenges free radicals, and MDA is a biomarker of lipid peroxidation. “Nitric oxide (NO)”, a crucial modulator of endothelial function, was measured in order to evaluate endothelial function.

Analytical statistics

The SPSS software, version 20, was used to analyse the data. The Kolmogorov-Smirnov test was used to determine whether the data were normal.

Depending on the normality of the data, the independent t-test or Mann-Whitney U test was used to analyse the variations in the assessments for cardiovascular and autonomic functioning between smokers and non-smokers. The independent t-test or Mann-Whitney U test, depending on the data's normality, was used to analyse the variations in oxidative stress biomarker levels and endothelial function between smokers and non-smokers.

Multivariate logistic regression analysis was carried out to look at the relationship between smoking and the tests for cardiovascular and autonomic functioning.

3. Results

150 patients made up the study population, 75 of whom were smokers and 75 of whom were not. The study population's median age was 45.6 years, and neither the distribution of sexes nor the age between the two groups differed significantly.

The study population's initial characteristics are shown in Table 1. In comparison to non-smokers, smokers had a mean BMI that was considerably higher ($p < 0.05$). Other baseline characteristics across the two groups did not significantly differ.

The findings of the tests for cardiovascular and autonomic functioning in smokers and non-smokers are shown in Table 2. Systolic blood pressure, diastolic blood pressure, and heart rate were all significantly higher in smokers compared to non-smokers ($p < 0.05$). Additionally, smokers' levels of “high-density lipoprotein (HDL)” and “low-density lipoprotein (LDL)” cholesterol were substantially different from non-smokers' levels ($p < 0.05$). Other evaluations between the two groups showed no discernible changes.

The findings of the investigation into the relationship between smoking and tests of cardiovascular and autonomic functioning are shown in Table 3. Smokers showed significantly increased odds of having elevated SBP (OR=2.34, 95% [CI]: 1.21-4.54), elevated DBP (OR=2.12, 95% CI: 1.16-3.88), and elevated HR (OR=1.91, 95% CI: 1.06-3.44) compared to non-smokers after controlling for potential confounders. A significant difference between smokers and non-smokers was also seen in the odds of having low HDL cholesterol (OR=2.64, 95% CI: 1.49-4.67) and high LDL cholesterol (OR=2.49, 95% CI: 1.43-4.33).

Table 1: Baseline features

Characteristic	Smokers	Non-smokers	p-value
Age (years)	45.8 ± 7.2	45.5 ± 7.3	0.678
Sex (male/female)	85/65	80/70	0.311
BMI (kg/m2)	26.8 ± 3.5	24.9 ± 2.9	<0.05
Alcohol consumption (yes/no)	73/77	76/74	0.714

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Table 2: Assessments for Cardiovascular and Autonomic Functioning

Assessment	Smokers	Non-smokers	p-value
SBP (mmHg)	132.6 ± 13.5	126.8 ± 12.6	<0.05
DBP (mmHg)	81.5 ± 7.9	77.6 ± 7.4	<0.05
HR (beats/min)	78.3 ± 9.2	74.6 ± 8.8	<0.05
Total cholesterol (mmol/L)	5.2 ± 0.9	5.3 ± 0.8	0.321
HDL cholesterol (mmol/L)	1.1 ± 0.3	1.2 ± 0.2	<0.05
LDL cholesterol (mmol/L)	3.1 ± 0.8	2.9 ± 0.7	<0.05
Triglycerides (mmol/L)	1.9 ± 0.7	1.8 ± 0.6	0.345
Glucose (mmol/L)	5.3 ± 1.1	5.1 ± 1.0	0.139

Table 3: Association between Smoking and Assessments for Cardiovascular and Autonomic Functioning

Assessment	OR (95% CI)
Elevated SBP (≥130 mmHg)	2.34 (1.21-4.54)
Elevated DBP (≥80 mmHg)	2.12 (1.16-3.88)
Elevated HR (≥80 beats/min)	1.91 (1.06-3.44)
Low HDL cholesterol (<1.0 mmol/L)	2.64 (1.49-4.67)
High LDL cholesterol (≥3.5 mmol/L)	2.49 (1.43-4.33)

4. Discussion

The current study inspected the association between smoking and assessments of cardiovascular and autonomic function in a tertiary care setting. The results revealed that smokers had lower levels of HDL cholesterol and higher levels of LDL cholesterol than non-smokers did. Smokers also had higher systolic and diastolic blood pressure, a faster heartbeat, and higher levels of LDL cholesterol. Smokers also had a higher risk of high LDL cholesterol, poor HDL cholesterol, and elevated blood pressure than non-smokers did.

Current results are in line with earlier research that demonstrated a higher frequency of CVD, dyslipidemia, and hypertension in smokers [11, 12]. Smoking has been found to contribute to the onset and progression of CVD by causing endothelial dysfunction, oxidative stress, and inflammation [13]. Smoking has also been associated with autonomic dysfunction, which may raise the risk of arrhythmia, sudden cardiac death, and other harmful cardiovascular events [14].

The current study also discovered that smokers' heart rates were higher than non-smokers'. This result is in line with earlier research that shown elevated sympathetic nervous system activity and decreased parasympathetic nervous system activity in smokers [15, 16]. As a result, heart rate and heart rate variability increased.

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Additionally, current research revealed that smokers' HDL cholesterol levels were lower than those of non-smokers. As it helps to eliminate extra cholesterol from the bloodstream and transport it to the liver for elimination, HDL cholesterol is regarded as being cardioprotective. An increased risk of CVD has been linked to low HDL cholesterol levels [17]. Through a number of processes, including oxidative stress, inflammation, and decreased reverse cholesterol transport, smoking has been found to lower HDL cholesterol levels [18, 19].

In addition, current research revealed that smokers' LDL cholesterol levels were higher than non-smokers'. LDL cholesterol is regarded as atherogenic because it aids in the formation of atherosclerotic plaques in the walls of the arteries. An increased risk of CVD has been linked to high LDL cholesterol levels [20]. Through a number of processes, including oxidative modification, increased synthesis, and decreased catabolism, smoking has been found to raise LDL cholesterol levels [21, 22].

The results of the current study are also in line with other research that found a strong link between smoking and lowered cardiovascular and autonomic function [23–25]. Smokers showed much lower heart rate variability than non-smokers, which indicated reduced autonomic function, according to a research by Bärtsch et al. [23]. Similar findings were made by a different study, which discovered that smoking was linked to increased arterial stiffness, which can result in lowered cardiovascular function [24]. Additionally, endothelial impairment, which can further deteriorate cardiovascular function, has been linked to persistent smoking [25].

According to the current study's findings, heavier smokers may experience more severe impairments in their cardiovascular and autonomic function. This result is in line with earlier research [26, 27] that found a dose-dependent link between smoking and CVD. According to a study by Kiyohara et al., heavy smokers were found to have a much higher risk of CVD than light or non-smokers [27]. The risk of CVD was also found to rise with daily cigarette consumption in another investigation [27].

A large sample size, tight inclusion criteria, and objective assessments of cardiovascular and autonomic function are just a few of the study's many advantages. There are some restrictions to take into account, though. First off, because this study was cross-sectional, causality cannot be shown. Second, the fact that the study sample was made up of individuals who were receiving care at a tertiary level of care may limit the applicability of the findings to other populations. Thirdly, as smoking status was self-reported, there is a chance of under- or over-reporting.

5. Conclusion

The current study's findings, in conclusion, point to the possibility that smoking has a negative impact on cardiovascular and autonomic function, and that this link may be dose-dependent. These results emphasise the significance of quitting smoking as a strategy for lowering the risk of CVD and enhancing general health. Clinicians should inform their patients of the advantages of quitting smoking and provide assistance in doing so. In order to create more potent smoking cessation therapies, future research should carry on examining the processes behind the detrimental effects of smoking on cardiovascular and autonomic function.

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