Quantitative Assessment of Serum C - Reactive Protein Levels among Smokers and Non Smokers

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ABSTRACT

Background: Recent progress in the comprehension of intricate tumor interactions has resulted in the identification of a link between inflammation and cancer. A high level of pro-inflammatory cytokines within the tumor microenvironment can stimulate angiogenesis, thereby promoting neoplastic proliferation. Serum C-reactive protein serves as a sensitive indicator of inflammation and could possess considerable prognostic significance as an early biomarker for cancer diagnosis. Aims and Objectives: To quantitatively measure serum C-reactive protein levels in both smokers and non-smokers, as well as to assess its role in cancer development. Materials and Methods: The study included a sample of 25 non-smokers and 25 smokers. All samples underwent C-reactive protein testing using immunoturbidimetry. The observations were analyzed statistically. **Statistical analysis:** The data was entered into the database management system of SPSS version 20.0. An independent t-test and Mann-Whitney U test were utilized to compare the C-reactive protein levels between two groups. For pairwise comparisons, Tukey's multiple post hoc procedure was employed. **Results:** The average C-reactive protein level in non-smokers was 55.24±12.0312, while in smokers, it was 55.88±11.76. In smokers, the C-reactive protein level was slightly higher compared to non-smokers. The comparison between non-smokers and smokers regarding C-reactive protein values showed statistical significance, with a p-value of ≤0.05. **Conclusion:** Our results indicate that the concentration of serum C-reactive protein was higher in smokers and is linked to an increased risk of developing oral cancer, potentially serving as a biomarker.

Key words: C reactive protein, Smokers, Non – smokers, Cancer

INTRODUCTION

The link between smoking and the rise in morbidity and mortality is well-established, and current statistics suggest that out of the one billion smokers globally, 500 million are likely to die prematurely due to smoking-related illnesses. Smoking has been proven to adversely affect many organs, and the array of diseases associated with smoking as a contributing factor is vast. C-reactive protein (CRP) is a well-recognized biomarker indicating systemic inflammation, synthesized by the liver in reaction to pro-inflammatory cytokines, especially interleukin-6 (IL-6). Elevated levels of CRP correlate with a higher risk of cardiovascular diseases, diabetes, and various other chronic health issues. As a highly sensitive marker of inflammation, CRP is frequently utilized in both clinical and research environments to evaluate an individual's inflammatory condition.[4] Tobacco smoking is a significant modifiable risk factor for several illnesses, such as several

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malignancies, coronary artery disease, and chronic obstructive pulmonary disease (COPD).[5] Additionally, it causes systemic inflammation via oxidative stress and the activation of inflammatory pathways. According to numerous studies, smokers often have higher CRP levels than nonsmokers, which may be a sign that the body is continuously reacting to the hazardous components of tobacco smoke with inflammation.[6]

There are two theories that explain how CRP functions within cages during inflammation. The first is referred to as the induction hypothesis, which suggests that acute or chronic inflammation raises SRB levels, resulting in excessive cell growth and permanent DNA damage. The second theory focuses on the host's immune response and is termed the "response hypothesis." [7]This theory posits that the immune response to cancer triggers an increase in CRP levels. However, it remains unclear how SRB levels rise at the onset of cancer, and whether they should be regarded as a potential risk factor for cancer development is consistently highlighted.[8] The aim of the study was quantitative assessment of serum C reactive protein levels in potentially malignant disorders (PMDS) and to evaluate their significant role as a prognostic marker.

MATERIALS AND METHODS

The cross-sectional study involved individuals who visited the department of Oral & Maxillofacial Pathology and Oral Microbiology for hematological assessments. This study received ethical approval (132/IEC/SIBAR/2023) from the Institutional Ethics Committee. The sample size was calculated using G*Power 3.1.9, with an α of 0.05, an effect size of 0.060, and a power level of 0.08, corresponding to a p-value of 0.05, resulting in a total of twenty-five samples in each group. The study included twenty-five non-smokers and twenty-five smokers, with the inclusion criteria requiring participants to be individuals attending for oral prophylaxis, with a history of smoking or no smoking habits. The exclusion criteria encompassed individuals with systemic illnesses or those on long-term medication. Informed consent was obtained from all individuals willing to participate in the study. Blood samples of 5 ml were collected from all fifty participants, and serum was separated. The serum levels of C-reactive protein were measured using immunoturbidimetry methods (Bio Majestry R JCA-BM), with the normal reference range defined as ≤ 10 mg/dl.

Statistical analysis

Data entry and analysis was performed using the software SPSS version 20.0. Descriptive statistics was used to determine the frequency, percentage, mean, median, SD and range. The mean C reactive protein in both the groups was done using independent t-test and pair wise comparisons of two groups with respect to C- reactive protein values was done by Mann-Whitney U test.

RESULTS

The total number of individuals who participated in this study was fifty, consisting of twenty-five non-smokers and twenty-five smokers. Within the non-smoker group, there were twelve males and thirteen females, while all twenty-five individuals in the smoker group were male. When comparing non-smokers and smokers regarding mean age using an independent t-test, the non-smokers had a mean age of 55.24 ± 12.03, compared to 55.88 ± 11.76 for smokers. The maximum age range for non-smokers was 56-65 years, whereas for smokers it was 46-55 years. The comparison of mean ages between non-smokers and smokers using an independent t-test did not yield statistically significant results ($p \geq 0.05$) (Graph 1). When examining C-reactive protein levels between non-smokers and smokers using the Mann-Whitney U test, smokers exhibited higher levels at 55.88 ± 11.76, while non-smokers had levels of 55.24 ± 12.03. The comparison of C-reactive protein values between non-smokers and smokers was statistically significant with $p \leq 0.05$ (Table 1). The normality of C-reactive protein values in both non-smokers and smokers, assessed by the Shapiro-Wilk test, showed a highly statistically significant result with $p \leq 0.05$ (Table 2). The receiver operating characteristic curve (ROC) graphical plot illustrates the predictive capabilities of C-reactive protein values for identifying smokers (Graph 2).

DISCUSSION

The connections between smoking and higher rates of illness and death are well documented, and current

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projections suggest that out of the one billion smokers globally, 500 million will suffer premature deaths due to tobacco-related illnesses.[1] Smoking has harmful effects on various body organs, and a comprehensive list of diseases associated with smoking is extensive. [2]It has been widely recognized that cigarette smoking is a significant and traditional risk factor for the onset of cardiovascular disease (CVD) and atherosclerosis.[10,5] Recently, it has been acknowledged that CVD has an inflammatory component and is even referred to as an inflammatory condition.[11] Furthermore, a connection has been established between smoking and several chronic inflammatory conditions, including chronic obstructive pulmonary disease (COPD), rheumatoid arthritis, systemic lupus erythematosus, and Crohn's disease.[12,13,14] While the precise mechanisms that link smoking to these conditions are not fully understood, there has been a growing interest in exploring the relationship between smoking and inflammatory markers as a means of uncovering smoking-related disease explanations. One such marker, C-reactive protein (CRP), can be measured easily and sensitively in various clinical contexts to track disease progression.[4] The incidence of smoking-related illnesses has been rapidly increasing in our country over the past twenty years, primarily due to habits like tobacco use in forms such as chewing or smoking. Numerous researchers are actively working to combat this deadly addiction.[15]

The current research quantitatively measured serum C-reactive protein (CRP) levels in both non-smokers and smokers to investigate the inflammatory effects of tobacco consumption. Our results indicate that smokers have notably higher C-reactive protein levels than non-smokers, which supports the idea that smoking leads to a persistent low-grade inflammatory condition. These results align with the findings of Black S et al. from 2004. The increase in CRP levels in smokers is consistent with existing research that shows tobacco smoke contains various pro-inflammatory agents, such as reactive oxygen species (ROS) and other harmful substances, which contribute to endothelial dysfunction and systemic inflammation[16] Notably, nicotine and carbon monoxide are known to trigger the release of cytokines like interleukin-6 (IL-6), which is a key promoter of hepatic CRP production. Various population-based studies, including the National Health and Nutrition Examination Survey (NHANES) and the Framingham Heart Study, have also found a positive association between smoking status and elevated CRP levels. Our research supports these findings and provides quantitative evidence relevant to our demographic population.[17]

Elevated CRP serves as an independent indicator of cardiovascular disease, which may help clarify the heightened cardiovascular issues and mortality rates observed in smokers. Testing for high-sensitivity CRP (hs-CRP) has been suggested as a method for assessing cardiovascular risk, making it particularly beneficial for identifying individuals at risk within the smoking population.[18] Our findings also indicate a potential dose-response correlation between daily cigarette consumption and CRP levels; however, additional studies with larger participant groups and longitudinal approaches are necessary to validate this link. The potential for CRP levels to decrease with smoking cessation is an encouraging avenue for research, as there is some evidence that systemic inflammation may reduce after quitting tobacco.

CONCLUSION

The quantitative evaluation of CRP levels underscores the inflammatory burden associated with smoking. These results highlight the need to incorporate inflammatory biomarkers into the risk evaluation of smokers and further support the public health message regarding the comprehensive harm of tobacco use. Future studies should aim to track CRP changes following smoking cessation and explore its possible role in informing intervention strategies.

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Conflict of Interest: None

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Tables and Graphs

Graph 1: Comparison of non – smokers and smokers with mean age

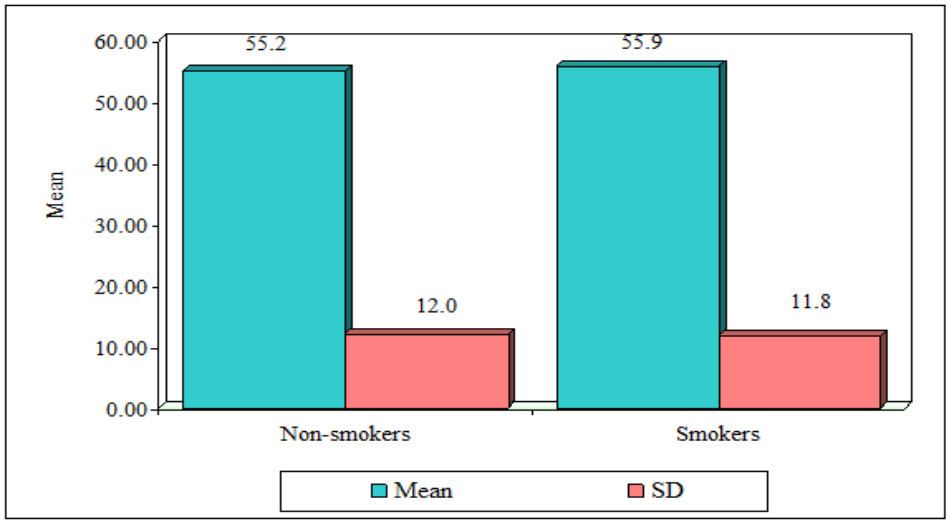

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Table 1: Comparison of non-smokers and smokers with C-reactive protein values by Mann-Whitney U test

Groups	Mean	SD	Mean rank	U-value	Z-value	P-value
Non-smokers	55.24	12.03	20.22			
Smokers	55.88	11.76	30.78	180.50	-2.5515	0.0107*

*p≤0.05

Table 2: Normality of C-reactive protein values in non-smokers and smokers by Shapiro-Wilk test

Group	Kolmogorov-Smirnov	df	p-value	Shapiro-Wilk	df	p-value
Non-smokers	0.1720	25	0.0500*	0.8500	25	0.0020*
Smokers	0.2590	25	0.0001*	0.6370	25	0.0001*

*p≤0.05

Graph 2: ROC curve in prediction of smokers by -reactive protein values