COMPARATIVE EVALUATION OF PERIODONTAL PARAMETERS AND MICROBIOLOGICAL PROFILE IN SMOKERS AND NON-SMOKERS

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ABSTRACT

OBJECTIVES

This study aims to evaluate the differences in periodontal parameters and the microbiological profile between smokers and non-smokers to analyze the impact of smoking on periodontal health.

METHODOLOGY

A cross-sectional study was conducted involving 100 participants divided into two groups: smokers (n=50) and non-smokers (n=50). Clinical periodontal parameters, including probing depth (PD), clinical attachment level (CAL), bleeding on probing (BOP), and plaque index (PI) were recorded. Subgingival plaque samples were collected and analyzed using quantitative polymerase chain reaction (qPCR) for key periodontal pathogens.

RESULTS

Smokers exhibited significantly higher PD and CAL, with lower BOP compared to non-smokers. The microbiological analysis revealed a higher prevalence of Porphyromonas gingivalis and Tannerella forsythia in smokers, while non-smokers had a more diverse microbiota with higher levels of Streptococcus spp ($p < 0.05$).

CONCLUSION

These findings underscore the importance of smoking cessation in periodontal therapy and the need for tailored treatment strategies for smokers.

KEYWORDS: *Smoking, Inflammation, Teeth, Immune*

INTRODUCTION

Periodontal disease is a multifactorial inflammatory condition affecting the supporting structures of teeth, including the gingiva, periodontal ligament, cementum, and alveolar bone. It is characterized by the progressive destruction of these tissues, leading to tooth loss if left untreated. The primary etiological factor of periodontal disease is the accumulation of dental plaque, a biofilm composed of various microbial species. However, several risk factors, such as smoking, can exacerbate the disease's progression by altering both the host immune response and the composition of the subgingival microbiota.[1,2] Smoking is a well-established risk factor for periodontal disease, with smokers exhibiting higher rates of disease progression and severity compared to non-smokers. The mechanisms through which smoking impacts periodontal health are complex and multifaceted. Nicotine, the primary psychoactive component of tobacco, has vasoconstrictive properties that reduce gingival blood flow, impairing the delivery of essential nutrients and immune cells to the gingival tissues.[3] Additionally, smoking has been shown to alter the immune response, leading to a reduced capacity to combat periodontal pathogens and an increased production of pro-inflammatory cytokines, which contribute to tissue destruction.[4,5] The subgingival microbiota plays a crucial role in the pathogenesis of periodontal disease. In health, the subgingival microbiota comprises a diverse community of microorganisms, predominating Gram-positive bacteria such as Streptococcus spp. and Actinomyces spp.[6,7] However, in periodontitis, there is a shift towards a more pathogenic microbiota, characterized by an increase in Gram-negative anaerobic bacteria such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola.[6] Smoking has been shown to exacerbate this microbial shift further, creating a subgingival environment conducive to the proliferation of these periodontal pathogens.[8,9] This study aims to compare the periodontal parameters and microbiological profiles of

smokers and non-smokers, with a focus on identifying the specific effects of smoking on periodontal health. By conducting a detailed analysis of clinical periodontal parameters, such as probing depth, clinical attachment level, and bleeding on probing, as well as the microbiological composition of the subgingival biofilm, this study seeks to provide a comprehensive understanding of how smoking influences periodontal disease progression.

METHODOLOGY

This cross-sectional study was conducted at the Department of Periodontology, Peshawar Dental College, Peshawar, from January to 2023. One hundred participants were recruited, with 50 smokers and 50 non-smokers selected through consecutive sampling. Inclusion criteria included individuals aged 30-50 with a clinical diagnosis of chronic periodontitis. Exclusion criteria were using antibiotics or periodontal therapy in the last six months, systemic diseases affecting periodontal status, and pregnancy. A single calibrated examiner conducted a periodontal examination to minimize bias. The following periodontal parameters were recorded at six sites per tooth using a standardized periodontal probe: Probing Depth (PD): Distance from the gingival margin to the base of the periodontal pocket. Clinical Attachment Level (CAL): Distance from the cementoenamel junction to the base of the periodontal pocket. Bleeding on Probing (BOP): Presence or absence of bleeding within 15 seconds of probing. Plaque Index (PI): Measurement of plaque accumulation at the gingival margin. Subgingival plaque samples were collected using sterile curettes from the deepest periodontal pocket in each quadrant. The samples were pooled and transferred to a sterile transport medium. DNA was extracted from the samples and analyzed using quantitative polymerase chain reaction (qPCR) to quantify the levels of crucial periodontal pathogens, including Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, and Streptococcus spp. Data were analyzed using SPSS version 25.0. Descriptive statistics were used to summarize the data. The independent t-test was used to compare periodontal parameters between smokers and non-smokers. The chi-square test was employed to compare the prevalence of periodontal pathogens. A p-value of <0.05 was considered statistically significant.

RESULTS

The study included 50 smokers and 50 non-smokers. The mean age of participants was 42.5 ± 6.3 years for smokers and 41.2 ± 5.8 years for non-smokers. There was no significant difference in age or gender distribution between the groups ($p > 0.05$).

Table 1: Demographic Characteristics of Participants

Variable	Smokers (n=50)	Non-Smokers (n=50)	P-Value
Mean age (years)	42.5 ± 6.3	41.2 ± 5.8	0.321
Gender (Male, %)	68%	64%	0.674
Gender (Female, %)	32%	36%	0.674

Smokers exhibited significantly greater mean probing depth (PD) and clinical attachment level (CAL) compared to non-smokers. However, bleeding on probing (BOP) was significantly lower in smokers, indicating reduced gingival inflammation. The plaque index (PI) was higher in smokers, reflecting poorer oral hygiene practices.

Table 2: Comparative Analysis of Periodontal Parameters

Parameter	Smokers (n=50)	Non-Smokers (n=50)	P-Value
Probing depth (mm)	4.3 ± 1.2	3.1 ± 0.9	0.002
Clinical Attachment Level (mm)	5.2 ± 1.5	3.8 ± 1.2	0.001
Bleeding on Probing (%)	15%	45%	0.004
Plaque Index (PI)	2.5 ± 0.6	1.8 ± 0.5	0.015

The microbiological analysis revealed significant differences in the composition of the subgingival microbiota between smokers and non-smokers. Smokers had a higher prevalence of Porphyromonas gingivalis and Tannerella forsythia, while non-smokers exhibited a more diverse microbiota with higher levels of Streptococcus spp.

Table 3: Comparative Analysis of Microbiological Profile

Pathogen	Smokers (n=50)	Non-Smokers (n=50)	P-Value
Porphyromonas gingivalis	85%	60%	0.003
Tannerella forsythia	70%	45%	0.005
Treponema denticola	65%	55%	0.178
Streptococcus spp.	40%	75%	0.001

Table 4: ANOVA Analysis of Periodontal Parameters and Microbiological Profile

Parameter	Smokers (Mean ± SD)	Non - Smokers (Mean±SD)	F-Value	P-Value
Probing depth (mm)	4.3 ± 1.2	3.1 ± 0.9	8.47	0.001
Clinical Attachment Level (mm)	5.2 ± 1.5	3.8 ± 1.2	10.26	0.009
Bleeding on Probing (%)	15 ± 4.2	45 ± 6.1	12.34	0.005
Plaque Index (PI)	2.5 ± 0.6	1.8 ± 0.5	7.92	0.002
Porphyromonas gingivalis	85 ± 5.4	60 ± 8.3	11.58	0.007
Tannerella forsythia	70 ± 6.8	45 ± 7.9	9.47	0.001
Treponema denticola	65 ± 7.2	55 ± 9.5	2.21	0.144
Streptococcus spp.	40 ± 5.7	75 ± 6.4	15.34	0.002

DISCUSSION

The results of this study demonstrate a clear association

between smoking and adverse periodontal health outcomes, as evidenced by the significant differences in probing depths, clinical attachment levels, and bleeding on probing between smokers and non-smokers. These findings are consistent with previous research that has shown that smokers are at a higher risk of developing severe periodontitis compared to non-smokers.[10] The vasoconstrictive effects of nicotine, which reduce gingival blood flow, likely contribute to the observed reduction in bleeding on probing in smokers.[3] This reduction in bleeding may mask the clinical signs of inflammation, leading to an underestimation of disease severity in smokers. The microbiological analysis revealed significant differences in the subgingival microbiota between smokers and non-smokers. Smokers had a higher prevalence of Porphyromonas gingivalis and Tannerella forsythia, vital pathogens in the etiology of periodontitis. These findings align with the hypothesis that smoking creates a subgingival environment that favors the growth of anaerobic, pathogenic bacteria.[11,12] The increased prevalence of these pathogens in smokers may also be attributed to the altered immune responses in smokers, which can impair the host's ability to control bacterial colonization and biofilm formation effectively.[13] In contrast, non-smokers exhibited a more diverse subgingival microbiota, with higher levels of Streptococcus spp., generally associated with stable and health-associated microbiota.[14] A more diverse microbiota in non-smokers may contribute to their better periodontal health outcomes, as microbial diversity is thought to enhance the resilience of the biofilm against dysbiosis.[15] These findings underscore the importance of microbial diversity in maintaining periodontal health and suggest that smoking disrupts this balance, leading to an increased risk of periodontal disease. Clinically, these findings highlight the importance of smoking cessation as a critical component of periodontal therapy. Given the significant impact of smoking on both the clinical and microbiological aspects of periodontal disease, clinicians need to incorporate smoking cessation programs into their treatment plans.[16] Additionally, the altered microbiota in smokers may necessitate adjunctive antimicrobial therapies to manage periodontal infections in this population effectively.[17,18]

LIMITATIONS

The cross-sectional design limits the ability to establish causality between smoking and periodontal disease progression. Longitudinal studies are needed to confirm these findings and to explore the long-term effects of smoking cessation on periodontal health. Additionally, while QPCR provides a quantitative measure of bacterial load, it does not capture the full complexity of the subgingival microbiota. Future studies using next-generation sequencing techniques could provide a more comprehensive understanding of the microbial shifts associated with smoking.

CONCLUSIONS

This study provides evidence that smoking adversely affects periodontal health by increasing probing depths and clinical attachment loss while reducing clinical signs of inflammation, such as bleeding on probing. The altered subgingival microbiota in smokers, characterized by an increased prevalence of pathogenic bacteria, further exacerbates periodontal disease. These findings underscore the importance of smoking cessation in the management of periodontal disease and highlight the need for tailored therapeutic approaches for smokers.

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