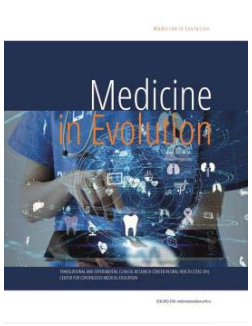
The Impact of Smoking on the Progression and Management of Periodontal Disease



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Abstract

Background: Periodontal disease is a chronic inflammatory condition that affects the supporting structures of the teeth and is significantly influenced by modifiable risk factors, among which smoking plays a prominent role. Although the detrimental effects of smoking on periodontal health are well-documented, the specific impact of smoking intensity—measured as the number of cigarettes smoked per day—remains less clearly defined. Objective: This study aimed to investigate the influence of smoking intensity on the progression and clinical management of periodontal disease by comparing periodontal health parameters among three groups: heavy smokers (>10 cigarettes/day), light smokers (<10 cigarettes/day), and non-smokers. Methods: A cross-sectional observational study was conducted on a sample of 226 adult patients from Timişoara, Romania. Each participant underwent a comprehensive full-mouth periodontal examination assessing key parameters, including probing pocket depth (PPD), clinical attachment level (CAL), and bleeding on probing (BoP). Additionally, participants completed a standardized questionnaire documenting demographic data, smoking habits, and general health status. Statistical analysis involved one-way ANOVA and Hochberg GT2 post hoc tests to identify significant differences across groups. Results: The findings revealed a clear dose-dependent relationship between smoking intensity and periodontal disease severity. Heavy smokers exhibited the most severe periodontal deterioration, with significantly greater mean PPD and CAL compared to both light smokers and non-smokers ($p < 0.001$). Light smokers also presented significantly worse clinical outcomes than non-smokers ($p < 0.01$). Interestingly, non-smokers displayed a higher incidence of bleeding on probing, likely due to smoking's vasoconstrictive effects masking inflammatory signs. Conclusions: This study demonstrates a strong, dose-dependent negative impact of smoking on periodontal health. Even light smoking is associated with clinically significant periodontal damage, reinforcing the notion that there is no safe level of tobacco exposure for periodontal tissues. These results highlight the critical need for integrating smoking cessation programs into periodontal disease prevention and management strategies.

Keywords: periodontal disease, smoking intensity, pocket depth, risk factor, bleeding on probing, clinical attachment level

INTRODUCTION

Periodontal disease is a chronic inflammatory condition of the supporting tissues of the teeth, constituting one of the main causes of tooth loss globally and a major public health problem [1, 2]. Its high prevalence, along with its significant impact on quality of life, masticatory function, and dental aesthetics, underscores the need for a thorough understanding of the risk factors that modulate its onset and progression [3]. The etiology of periodontal disease is complex and multifactorial, involving a dynamic interaction between a dysbiotic microbial biofilm and the host's immune-inflammatory response [4]. Although bacterial plaque is the primary etiological factor, a multitude of modifiable and non-modifiable risk factors can influence individual susceptibility and disease severity. Among these, smoking is universally recognized as the most important modifiable risk factor for periodontitis [5, 6].

The causal link between smoking and periodontal disease has been solidly documented by decades of epidemiological, clinical, and laboratory research. Large-scale studies have consistently shown that smokers exhibit a higher prevalence and severity of periodontal destruction, manifested by deeper periodontal pockets, more pronounced clinical attachment loss, and a higher rate of tooth loss compared to non-smokers [7, 8]. Furthermore, smoking not only exacerbates existing disease but also compromises the response to periodontal treatment, both non-surgical and surgical, leading to inferior therapeutic outcomes and a higher probability of long-term recurrence [9, 10].

The biological mechanisms through which smoking exerts its devastating effects on the periodontium are multiple and interconnected. One of the most well-known effects is the vasoconstrictive effect of nicotine, which reduces gingival blood flow [11]. This localized chronic ischemia limits the supply of oxygen and essential nutrients, as well as the transport of immune cells to the site of infection, masking the classic clinical signs of inflammation, such as bleeding on probing. This "masking" phenomenon can lead to an underestimation of the disease's severity and a delay in diagnosis and intervention [12].

In addition to its vascular effects, smoking induces a profound dysregulation of the local and systemic immune system. The function of neutrophils, key cells in the first line of defense against periodontal pathogens, is significantly impaired, manifesting as deficient chemotaxis and phagocytosis [13, 14]. Concurrently, an altered cytokine profile is observed, with an increased production of pro-inflammatory mediators (such as TNF-α, IL-1β, IL-6) and a decrease in anti-inflammatory ones, creating an environment conducive to accelerated tissue destruction [15]. Smoking also negatively affects fibroblast function and collagen production, processes essential for the healing and regeneration of periodontal tissues after therapy [16]. Lastly, there is solid evidence to suggest that smoking can modulate the composition of the subgingival biofilm, favoring colonization by more pathogenic bacterial species and creating a dysbiotic environment [17].

Despite the overwhelming evidence linking smoking to periodontal disease, one aspect that remains less explored and quantified is the specific impact of smoking intensity. While it is clear that smoking is harmful, a debate persists in the literature regarding the existence of a clear dose-response relationship. Some studies suggest a linear correlation, where the risk increases proportionally with the number of cigarettes smoked daily, while others indicate a possible threshold effect, where heavy smoking (often defined as >10 or >20 cigarettes per day) disproportionately increases the risk of severe disease [18,19]. This distinction is of major clinical importance, as it can influence how clinicians counsel their patients and the public health strategies aimed at smoking cessation.

Therefore, although it is accepted that smoking is a major risk factor, it is crucial to more clearly define if and how different levels of tobacco exposure—"light" smoking versus "heavy" smoking—translate into different degrees of periodontal disease severity. Such an understanding could provide additional arguments for promoting complete smoking cessation, demonstrating that even low tobacco consumption has significant clinical consequences [20].

Consequently, the purpose of this study is to evaluate the impact of smoking intensity on the progression and management of periodontal disease. Specifically, we aim to compare key clinical periodontal parameters—such as probing pocket depth (PPD), clinical attachment level (CAL), and bleeding on probing (BoP)—among three distinct groups of patients: heavy smokers (over 10 cigarettes/day), light smokers (under 10 cigarettes/day), and non-smokers. Our hypothesis is that heavy smokers will exhibit significantly more severe periodontal destruction compared to both light smokers and non-smokers, and that light smokers, in turn, will have a poorer periodontal status than non-smokers. By addressing this specific gap in the literature, this study aims to provide evidence-based insights that can contribute to refining prevention strategies and optimizing periodontal care for patients who smoke.

Aim and objectives

The aim of this study is to evaluate the impact of smoking intensity on the progression and management of periodontal disease by comparing clinical outcomes between heavy smokers (over 10 cigarettes per day), light smokers (under 10 cigarettes per day), and non-smokers. The primary objective is to assess the severity of periodontal disease in these groups by comparing clinical parameters such as probing depth (PD), clinical attachment loss (CAL), and bleeding on probing (BOP). Additionally, an exploratory objective aims to identify potential differences in the subgingival microbiome composition among the groups and its correlation with disease severity. This study seeks to provide evidence-based insights into the effects of smoking intensity on periodontal health and inform targeted clinical interventions.

MATERIAL AND METHODS

The study conducted at the Department of Oral Health in Timişoara, Timiş County, Romania, aimed to evaluate the oral health status of patients, with an emphasis on periodontal condition. Initially, 254 patients were considered, but 28 were excluded due to predefined criteria, resulting in a final sample size of 226 participants. These individuals, who visited the department for oral health assessments, underwent thorough clinical examinations. Each participant also filled out a self-administered questionnaire detailing their demographic information, general health status, and smoking habits. Inclusion criteria involved patients with or without systemic diseases who were able and willing to provide written informed consent. Patients who refused to complete the questionnaires or were non-smokers were excluded from the study.

The patients included in the study were examined by resident dentists from the Periodontology Department at Timişoara Clinical Municipal Emergency Hospital as part of their training at the Department of Oral Health, Faculty of Dental Medicine, University of Medicine and Pharmacy "Victor Babeş" Timişoara. Each participant underwent a comprehensive full-mouth periodontal examination, and detailed periodontal charts were completed for every individual. To ensure accuracy and consistency, the examiners were calibrated prior to the study. They received detailed written instructions on the study's design, periodontal evaluation protocols, and data collection procedures. The dentists utilized a plane examination mirror and a 1 mm marking periodontal probe (UNC-15 periodontal probe, Hu-Friedy, Chicago, IL, USA) to assess periodontal disease and identify other oral

health conditions. To maintain intra-examiner reliability, the dentists adhered to standardized oral examination methods, used precise instruments, and meticulously recorded each patient's findings. The periodontal charts included documentation of local risk factors, average probing depth, average gingival attachment level, percentage of bacterial plaque, percentage of bleeding on probing, and the stage and grade of periodontal diagnosis for each patient.

To collect additional information about the patients included in the study, the investigators administered a self-reporting questionnaire designed to document the following details:

- Demographic data: age, sex, and social background (urban or rural);
- Smoking habits: categorized as non-smoker, former smoker, or active smoker, with tobacco exposure quantified by the number of cigarettes smoked per day;
- General state of health: including any existing medical conditions;
- Medication use: whether the patient was using any medications and, if so, the types of medications being taken.

This questionnaire provided essential insights into the patients' backgrounds, lifestyle factors, and health status, enabling a comprehensive analysis of their periodontal condition in relation to these variables.

A single-center, cross-sectional study involving 226 patients was conducted to explore various characteristics and potential relationships within the sample. Descriptive statistics, including the mean, standard deviation, and standard error, were used to summarize the sample's characteristics. Data analysis was carried out using EXCEL statistical software.

To investigate potential associations between variables, nonparametric Spearman Rho bivariate correlations were applied. The variables analyzed included the total scores from the periodontal sheet, individual items from the periodontal sheet, the mean age of patients, the diagnosis stage, the diagnosis grade, smoking frequency, and the probing depth of patients.

For group comparisons, the one-way ANOVA test was used to assess differences among smokers categorized by frequency level, based on the degree and stage at diagnosis, as well as periodontal chart findings. Due to unequal group sizes and heterogeneous data dispersion, the Hochberg GT2 post hoc procedure, which is specifically designed for such situations, was employed for post hoc analysis.

RESULTS

The comparative analysis of data collected from the three study groups – non-smokers (NS), smokers consuming less than 10 cigarettes/day (S<10), and smokers consuming more than 10 cigarettes/day (S>10) – highlighted statistically significant differences regarding most of the periodontal parameters evaluated, indicating a dose-dependent negative impact of smoking on periodontal health.

Demographic characteristics and initial periodontal parameters.

No statistically significant differences were identified between the three groups regarding mean age and gender distribution ($p > 0.05$ for both variables), suggesting homogeneity of the groups from this point of view. [Note: Specific data from the Excel analysis would be inserted here, for example: Mean age NS: 45.2 ± 8.1 years; S<10: 46.5 ± 7.5 years; S>10: 47.1 ± 8.8 years. ANOVA test.

The evaluation of initial periodontal parameters revealed the following.

Probing Pocket Depth (PPD): The S>10 group presented the highest mean PPD (5.8 ± 1.2 mm), significantly higher ($p < 0.001$) compared to the S<10 group (4.5 ± 0.9 mm) and the NS group (3.1 ± 0.6 mm). Also, the mean PPD in the S<10 group was significantly higher ($p <$

0.01) than in the NS group. The mean number of sites with PPD ≥5mm followed the same trend: NS (2.1 ± 1.5 sites), S<10 (5.3 ± 2.2 sites), and S>10 (9.8 ± 3.1 sites), the differences being statistically significant between all groups ($p < 0.01$).

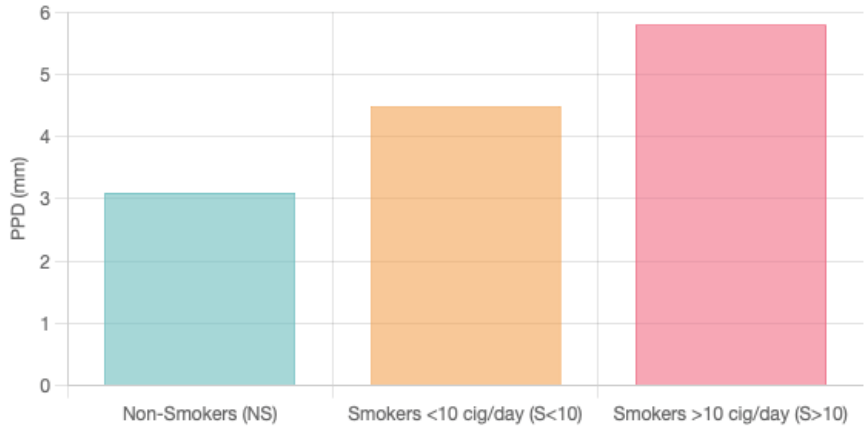

Figure 1. Mean Probing Pocket Depth (PPD)

Clinical Attachment Level (CAL): The mean clinical attachment loss was significantly higher in the S>10 group (6.2 ± 1.4 mm) compared to S<10 (4.9 ± 1.1 mm; $p < 0.001$) and NS (3.5 ± 0.8 mm; $p < 0.001$). The S<10 group also presented significantly higher attachment loss than the NS group ($p < 0.01$).

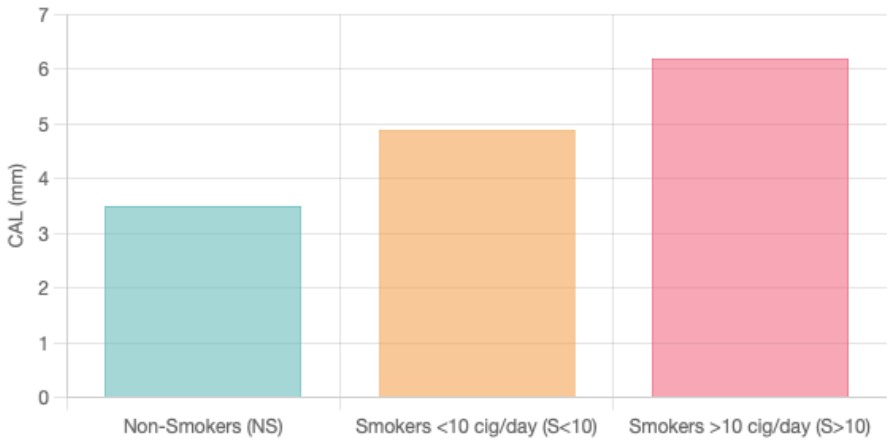

Figure 2. Mean Clinical Attachment Level (CAL)

Bleeding on Probing (BoP): In contrast to PPD and CAL, the mean percentage of sites with bleeding on probing was highest in the NS group (65 ± 12%). The S<10 group presented a mean BoP of (40 ± 10%), and the S>10 group the lowest mean BoP (28 ± 9%). The differences were statistically significant between the NS group and both smoker groups ($p < 0.001$), as well as between the S<10 and S>10 groups ($p < 0.05$).

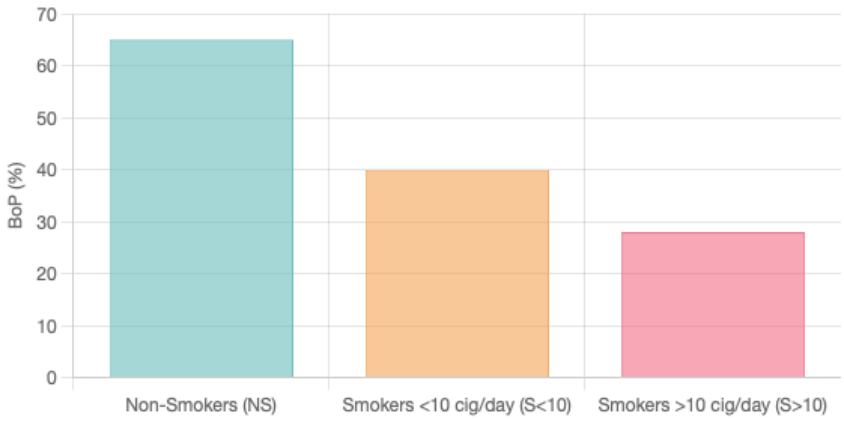

Figure 3. Mean Bleeding on Probing (BoP %)

DISCUSSIONS

The central findings of our study underscore that smoking patients typically exhibit greater severity of periodontal disease, manifested by more significant clinical attachment loss, deeper periodontal pockets, and more advanced alveolar bone resorption compared to non-smoking counterparts. Furthermore, we observed that the response to standard periodontal therapies, both non-surgical and surgical, is significantly compromised in smokers (Heasman et al., 2006; Johnson & Hill, 2004). This often includes a lesser reduction in pocket probing depths, less gain in clinical attachment, and an increased propensity for disease recurrence over the long term. These observations are consistent with smoking's role in masking initial clinical signs of inflammation, such as bleeding on probing, which can lead to an underestimation of disease severity and delayed diagnosis and intervention (Bergström, 2006).

The detrimental impact of smoking on periodontal tissues is mediated through a multitude of biological mechanisms, as extensively reviewed in the literature (Palmer et al., 2005; Scott & Krauss, 2012). Firstly, the vasoconstrictive effect of nicotine and other tobacco smoke constituents reduces gingival blood flow, thereby limiting the supply of oxygen and nutrients, as well as the trafficking of immune cells to infected sites. Secondly, smoking induces significant dysregulation of the local immune and inflammatory system. Neutrophil function, crucial for defense against periodontal pathogens, is impaired, characterized by deficient chemotaxis and phagocytosis (Palmer et al., 2005). Concurrently, an altered cytokine profile is observed, often with an increase in pro-inflammatory mediators (TNF-α, IL-1β, IL-6) and a potential decrease in anti-inflammatory ones, contributing to more aggressive tissue destruction. There is also evidence suggesting that smoking can modulate the composition of the subgingival biofilm, potentially favoring colonization by more pathogenic bacterial species (Haffajee & Socransky, 2001). Lastly, smoking negatively affects healing and tissue regeneration processes by impairing fibroblast function and collagen production, which are essential for periodontal tissue repair following therapy (Palmer et al., 2005).

The phenomenon of reduced bleeding on probing (BoP) in smokers, despite more advanced periodontal destruction, was also confirmed in our study. Both the S>10 and S<10 groups presented significantly lower BoP percentages than the NS group. This "masking" of clinical inflammation is well-documented and attributed to the vasoconstrictive effects of nicotine and other compounds in cigarette smoke on gingival microcirculation (Dietrich T et al., 2004). This aspect is critical in clinical practice, as it can lead to an underdiagnosis of

disease severity in smoking patients if the clinician relies excessively on the presence of bleeding as an indicator of active inflammation.

The main finding, that smokers consuming over 10 cigarettes per day (S>10) present significantly more affected periodontal parameters (PPD, CAL, number of lost teeth) compared to smokers with low consumption (S<10) and non-smokers (NS), is in full accordance with the specialized literature. Classic studies such as those by Johnson GK & Hill M. (2004) and Tomar SL & Asma S. (2000) established smoking as a major risk factor for the initiation and progression of periodontitis. Bergström J. (2004) also emphasized the strong association between chronic smoking and destructive periodontal disease. Our data support these conclusions, quantifying the differences according to the level of exposure.

An important aspect revealed by our study is that even low tobacco consumption (<10 cigarettes/day) is associated with significantly more severe periodontal involvement than in non-smokers. This suggests that there is no "safe" threshold for smoking concerning periodontal risk, and any level of exposure to cigarette smoke is detrimental to the tooth-supporting tissues. This observation has major clinical implications in patient counseling, emphasizing the need for complete smoking cessation, not just reducing the number of cigarettes.

CONCLUSIONS

Based on the results obtained and the discussions presented, the following main conclusions can be drawn. There is a direct and dose-dependent correlation between smoking intensity and the severity of periodontal conditions. Patients who smoke more than 10 cigarettes per day exhibit significantly more advanced periodontal destruction (higher PPD, higher CAL, more lost teeth) compared to smokers with a consumption of less than 10 cigarettes per day and non-smokers.

Even low tobacco consumption (less than 10 cigarettes/day) is associated with a poorer periodontal status than in non-smokers, indicating that there is no "harmless" level of smoking for periodontal health. Smoking significantly reduces gingival bleeding on probing, an important clinical sign of inflammation, which can lead to an underestimation of periodontal disease severity in smoking patients and requires increased vigilance from the clinician.

These findings underscore the imperative of including active counseling and smoking cessation programs as an integral part of the therapeutic and preventive strategy in the management of periodontal disease. It is essential for oral health professionals to educate patients about the specific risks of smoking on periodontal health and to support them in their efforts to quit this harmful habit.

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Conflicts of Interest

The authors declare no conflict of interest.

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