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Original Article

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Assessing the information quality of YouTube™ videos on the effects of smoking on oral health: a cross-sectional study

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ABSTRACT

Aims: To evaluate the content on the effects of smoking on oral health.

Methods: YouTube™ was searched for videos on 16 May 2023 using the keyword "smoking and oral health," and "sort by relevance" was preferred as the filter. 102 video content was scored based on the DISCERN instrument, the criteria established by the Journal of the American Medical Association (JAMA) and the Global Quality Score (GQS). The data were analyzed using analysis of variance, the Kruskal-Wallis test, and the Spearman's rho test.

Results: The mean DISCERN, JAMA, and GQS scores of all 102 videos are 2.71 (SD 1.10), 1.56 (SD 0.87), and 3.13 (SD 1.26), respectively. A strong positive correlation was detected between DISCERN and GQS ($r=0.894$, $p<0.001$), JAMA and GQS ($r=0.709$, $p<0.001$), While the number of views strongly positively correlated with the viewing rate and likes ($r=0.840$, $p<0.001$; $r=0.849$, $p<0.001$, respectively).

Conclusion: YouTube videos on smoking and oral health were limited in number and had poor quality. Dentists/specialists should upload videos containing comprehensive information regarding the detrimental effects of smoking on oral health and smoking cessation.

Keywords: YouTube™, internet, tobacco, smoking, oral health

INTRODUCTION

Smoking is one of the most important causes of preventable deaths worldwide. One billion people will die due to its harmful effects has been estimated by the 21st century.[1] It is also detrimental to oral health and generally accepted as the major preventable risk factor in the incidence and progression of periodontal disease.[2-5] Non-smoking individuals exposed to environmental tobacco smoke are also prone to oral and systemic diseases.[4,6,7] A new category of nicotine products emerged between 2006 and 2009, electronic cigarettes or e-cigarettes. About 40 million users of e-cigarettes worldwide.[8] E-cigarettes' potential risks and benefits have been extensively debated in many disciplines and are still controversial.[9,10]

The internet has revolutionized the way accessing and sharing information; with the advent of new technological applications, has emerged as a powerful platform for effectively communicating and disseminating further health-related information to patients. This convenient and easily accessible medium has empowered people to actively manage their health by providing various resources, ranging from reputable medical websites to online forums and interactive tools. YouTube™ is a widely recognized online video platform launched in 2005 and has grown into a global phenomenon, attracting billions of users who watch videos on a wide range of topics. Seeking knowledge about health-related issues, treatment options, and patient experience, YouTube™ offers endless content that caters to every interest and curiosity. Easy accessibility and cost-free availability are definite advantages, allowing individuals to explore various health topics and gain preliminary knowledge.[11] Compared to professionally curated medical resources, YouTube™ needs a comprehensive screening process for the content published on its platform. Initial filtering can lead to a significant disadvantage as the reliability and adequacy of the information provided in health videos may vary significantly. Viewers must exercise discernment and critical thinking skills to differentiate between evidence-based, trustworthy sources and those that may offer incomplete or misleading information. It is crucial to share qualified healthcare professionals or trusted sources to ensure accurate and reliable health-related videos should obtain.

Several studies evaluated YouTube videos about smoking.[12-15] However, as far as we know, research has yet to be conducted on YouTube™ videos as a source of information on smoking and oral health. This study aimed to analyze and determine the sources of uploads, the nature of the content, and the availability of relevant information in videos related to

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smoking and oral health on the YouTube™ platform and to assess the quality of information provided in these videos using quantitative scoring with adapted DISCERN score, Journal of American Medical Association (JAMA) benchmark criteria, and Global Quality Score (GQS). By utilizing these scoring tools, the study sought to evaluate the reliability and accuracy of the information presented in YouTube smoking and oral health videos.

METHODS

No clinical data, human participants, or laboratory animals were utilized in this research. All the information in this study was sourced exclusively from publicly available YouTube videos, and no data accessed compromised personal privacy. Furthermore, this study did not involve direct user engagement, making an ethics review unnecessary. All procedures were carried out in accordance with the ethical rules and the principles.

To account for algorithm-based variability and personalization that may influence search rankings, a standardized search was conducted on May 16, 2023. The search was performed on the online video-sharing site YouTube (https://www.YouTube.com) using the keyword 'smoking and oral health' with the 'sort by relevance' filter to capture a representative snapshot of the content most likely to be encountered by a general user. As search results may vary on different days, the initial 250 video links were stored. 102 videos were also evaluated by two observers (DDS, Ph.D.) who are experts in endodontics and have at least five years of clinical experience. Among all, 148 videos were excluded with no audio or visual, longer than 30 minutes, were not in English, were duplicated, and were irrelevant to the topic term. Two observers further analyzed the remaining 102 videos that met the inclusion criteria (**Figure**).

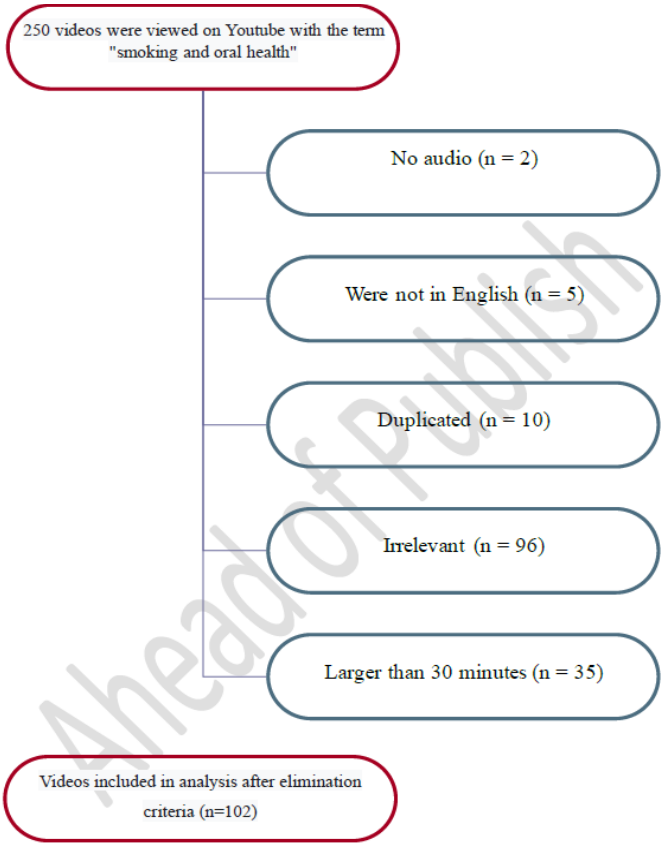

Figure. Flowchart of search strategy

Following the evaluation of the videos, the demographic features were documented, such as duration in seconds, upload time, the number of views, days since upload, and number of likes. The viewing rate was determined by the ratio of the number of views to the number of days since upload ×100%.

The uploaders were categorized as the source who prepared the videos into four categories dentist/specialist, hospital/dental clinic, dental hygienist, and other (layperson or an unclear source).

In assessing the quality of the selected YouTube™ videos, we employed the DISCERN instrument, the criteria established by the JAMA and the GQS. These widely recognized instruments are common standards for evaluating health information, ensuring a comprehensive quality assessment of the videos.

The DISCERN, originally designed to assist patients, general consumers, and caregivers in discerning the reliability of online information, has been adapted by Kılınc and Sayar[16] to create a modified version. This modified tool utilizes a five-point scale to assess accuracy and reliability. A total modified DISCERN score ranging from 0 to 25 is calculated by evaluating five specific questions as shown in the **Table 1**. The resulting score is then used to determine the level of reliability, categorized as poor, generally poor, moderate, good, or excellent. This modified DISCERN tool provides a systematic approach for evaluating the credibility and trustworthiness of web-based information, empowering individuals to make informed decisions about the reliability of online content.

Table 1. Modified DISCERN

1.	Is the aim clear concise, understandable?
2.	Are sources of information reliable? (cited publication video content were from valid studies, dentists endodontists)
3.	Is the information presented balanced and unbiased? (any reference to other treatment choices)
4.	Are additional sources of information listed?
5.	Does the video address areas of uncertainty?

The JAMA benchmark criteria, as proposed by Silberg et al.,[17] 17 utilize a scoring system ranging from 0 to 4. This scale comprises four individual criteria, with one point assigned for the presence of each item. **Table 2** shows the JAMA scale offers a nonspecific evaluation of source quality. A total score of 4 signifies a high-quality source, while a score of zero suggests poor quality.

Table 2. JAMA benchmark criteria

Authorship	Authors partnerships and contributors, their affiliations and relevant credentials should be provided
Attribution	References and resources used for the content and a copyright statement should be listed clearly
Disclosure	Dates when content was posted and updated should be indicated
Currency	Sponsorship, advertising commercial financing potential conflicts of interest should be prominently and fully disclosed

JAMA: Journal of the American Medical Association

The 5-point GQS Index was scored according to the flow, quality, and educational usefulness in a range between 1 (poor quality) to 5 (excellent quality), as explained in **Table 3**.[18]

Table 3. Global quality scores

Scores	
1	Poor quality poor flow, most information missing, not useful for education
2	Generally poor quality and flow of limited use to patients because only some information is present but many important topics missing
3	Moderare quality, suboptimal flow, somewhat useful for patients as some important information is adequately discussed but others poorly discussed
4	Good quality generally good flow useful to patients because most relevant information is covered but some topics not covered
5	Excellent quality and flow, highly useful to patients

Statistical Analysis

Shapiro-Wilks test was used to evaluate the distributions in the descriptive analysis of the data. Cohen's kappa analysis was used to assess inter-observer agreement. The resulting coefficients were interpreted as follows: 0.00-0.20: insignificant; 0.21-0.40: weak; 0.41-0.60: moderate; 0.61-0.80: significant; 0.81-1.00: almost perfect. Nonparametric data across different groups were compared using the Kruskal-Wallis test. The Spearman's rho test was used to evaluate the relationship between the quantitative variables. $p<0.05$ was accepted to indicate statistical significance.

RESULTS

Video duration, likes, views, viewing rate, and number of days elapsed since upload date were analyzed and presented in **Table 4**.

According to video sources, 47.05% (48/102) of the videos were posted by dentists/specialists. The proportion of the rest of the sources were hospitals/dental clinics 26.47% (27/102), other 18.62% (19/100), and dental hygienists 7.84% (8/102), respectively.

Agreement between different scores given by two experts on 102 videos was evaluated; Modified DISCERN (κ:0.895, $p<0.01$) and GQS (κ:0.899, $p<0.01$) scores showed near perfect agreement, while the JAMA (κ:1.000, $p<0.01$) score showed perfect agreement. Due to the high degree of inter-rater agreement, a single common score was determined for the different scores, and the study continued based on this commonly agreed score.

Table 5 presents data on all videos and videos grouped by sharing source, including information from DISCERN, JAMA, and GQS. There was no statistically significant difference in DISCERN, JAMA, and GQS between the uploaders of dentist/specialist and hospital/dental clinics ($p>0.05$). However, these values were significantly different from the uploader of the others and dental hygienists ($p=0.001$). No statistically significant difference was detected for DISCERN, JAMA, and GQS between dental hygienists and the other uploaders ($p>0.05$).

Table 5. GQS, modified DISCERN and JAMA scores by source of upload

Sources	GQS	Modified DISCERN	JAMA
All sources	3.13±1.26	2.71±1.10	1.56±0.87
Dentist/specialist	3.38±1.12	2.85±0.92	1.71±0.62
Hospital/dental clinic	3.63±1.24	3.15±1.23	1.59±0.57
Dental hygienist	1.63±0.74	1.00±0	0.75±0.71
Other	1.42±0.51	1.05±0.40	0.47±0.51

GQS: Global Quality Score, JAMA: Journal of the American Medical Association

A strong positive correlation was detected between DISCERN and GQS ($r=0.894$, $p<0.001$), JAMA and GQS ($r=0.709$, $p<0.001$), While the number of views strongly positively correlated with the viewing rate and likes ($r=0.840$, $p<0.001$; $r=0.849$, $p<0.001$, respectively), it moderately positively correlated with the number of days since upload ($r=0.442$, $p=0.001$). **Table 6** shows the relationship level between video variables. The relationship level between video variables.

Table 4. Characteristics of videos

Duration (secs)	Like	View	Viewing rate	Days since upload
200.31±198.18 (range 12 to 896)	5.304.38±42.640.88 (range 0 to 419.000)	216.354.60±1.701.666 (range 2 to 17.070.411)	21.559.67±138.534.30 (range 0.56 to 1.185.445.21)	1.515.476±1.421.227 (range 2 to 5040)

Table 6. The relationship level between video variables

	Like	Day since upload	View	Viewing rate	Duration (secs)	GQS	Modified DISCERN	JAMA
Like		r=0.265 p=0.007	r=0.840 p<0.001	r=0.779 p<0.001		-	-	-
Day since upload	r=0.265 p=0.007		r=0.442 p<0.001	-	-	-	-	-
View	r=0.840 p<0.001	r=0.442 p<0.001		r=0.849 p<0.001	-	-	-	-
Viewing rate	r=0.690 p<0.001	-	r=0.849 p<0.001		-	-	-	-
Duration (secs)	-	-	-	-		r=0.376 p<0.001	r=0.411 p<0.001	r=0.206 p=0.038
GQS	-	-	-	-	r=0.376 p<0.001		r=0.894 p<0.001	r=0.709 p<0.001
Modified DISCERN	-	-	-	-	r=0.411 p<0.001	r=0.894 p<0.001		r=0.662 p<0.001
JAMA	-	-	-	-	r=0.206 p=0.038	r=0.709 p<0.001	r=0.662 p<0.001	

[r]Spearman correlation coefficient. [-]No significant results found. GQS: Global Quality Score, JAMA: Journal of the American Medical Association

The majority of the videos were often focused on many contents of adverse effects of smoking, such as gum disease (n=63), oral cancer (n=63), stain (n=43), healing after surgery (n=34), terrible smell (n=35), lung cancer (n=13), implant surgery (n=13), immune response (n=11), diminished taste (n=10), cardiovascular disease (n=9), hairy tongue (n=7), anxiety (n=5) and leukoplakia (n=4). Half the videos included smoking cessation advice, while twelve gave smokers oral hygiene tips.

The videos shared not only the harmful effects of smoking cigarettes but also e-cigarettes (n=21), smokeless tobacco (n=11), pipes (n=3), and hookahs (n=2).

DISCUSSION

In the era of digitalization, when we have inquiries or need information, websites like Google and YouTube have become our primary go-to sources. Internet platforms shape our daily routines and are critical in essential matters. Internet and video-sharing media such as YouTube™ have gained immense popularity among individuals searching for health-related information and medical procedures. However, it is concerning that the information and videos uploaded to these platforms often need more scientific filtering, leading to potential misinformation and incomplete understanding for the viewers, especially patients. Despite concerns regarding the reliability and quality of such information, patients may likely continue to rely on YouTube™ as a prominent online resource. Therefore, professionals must conduct further research, considering the increased Internet use for health-related issues. To our awareness, this study represents one of the initial attempts to specifically evaluate YouTube™ videos regarding the effects of smoking on oral health, a niche that has remained relatively unexplored despite the abundance of general dental literature on the platform.[12,13,15,19-30] The aim of this study was to examine the quality and content of the videos about the impact of smoking on oral health and to shed light on its potential effects on individuals.

In the analysis studies on YouTube™, Instagram, and other internet platforms focusing on dental and medical issues in the literature, different results were reported regarding the quality evaluations of the videos. While some researchers report that the content quality of the posts was sufficient, others identified them as incomplete and inadequate. These disparities may arise from variations in the analyzed topics, evaluation criteria, and the diversity among video uploaders.

There are various filters available to sort the videos based on "view count," "upload date," and "video duration." In this study, the default filter "sort by relevance" was used for YouTube™ search, as it is the most commonly preferred option by users and research studies.

Smoking is a critical lifestyle factor with systemic effects on human health. Long-term exposure to cigarette smoke triggers pathophysiological processes such as systemic oxidative stress, chronic inflammation, and vascular endothelial dysfunction. This increases the incidence of systemic diseases such as atherosclerosis, chronic obstructive pulmonary disease, and coagulation disorders, and also negatively affects the prognosis of existing diseases.[31] Although our search term was "smoking and oral health," some videos mentioned that smoking contributes to lung cancer, cardiovascular disease, osteoporosis, anxiety, dementia, diabetes, and *Candida*.

The mouth is the only body part where the effects of smoking are visible, and unfortunately, it is often neglected by smokers. Most of the videos mentioned that smoking had been linked to various oral health problems, including more severe gum disease (periodontitis), increased tooth loss, gum recession, loose teeth, and more significant bone loss in the jaw. Smoking weakens the immune system and reduces blood flow to the gums, making it harder for the body to neutralize pathogens and heal damaged tissues.[32] This can lead to a higher risk of developing gum disease and susceptibility to more severe periodontal tissue disease. In the videos reviewed in this study, periodontologists shared more detailed content stating that smoking increases periodontal tissue destruction.

Electronic cigarettes, commonly known as e-cigarettes, have gained significant global popularity, mainly due to the widespread accessibility provided by the Internet.[33] Batteries power these devices by vaporizing a solution containing nicotine, flavors, and various chemicals. The advent of e-cigarettes has introduced a new option for nicotine consumption, attracting a substantial user base worldwide. Nicotine consumption, the puff duration of e-cigarettes, is typically longer and requires more potent suction than conventional cigarettes. This difference arises from the unique mechanism of e-cigarettes, where users inhale and activate the device through a more forceful suction. The prolonged puff duration and increased suction intensity contribute to nicotine delivery in e-cigarettes, potentially affecting user behavior and nicotine intake patterns. In the study focusing on YouTube™ videos, Merianos et al.[34] reported the results related to how e-cigarettes can assist individuals in quitting smoking, the purported health benefits of e-cigarettes compared to traditional cigarettes, and the absence of smoke or second-hand smoke exposure associated with e-cigarette use. This result can be attributed to the fact that 53.1% (n=55/34) of the videos reviewed by the researchers were advertising/marketing and personal/testimonial uploaders. Contrary to the study, the videos in this study indicated that e-cigarettes have similar, or even worse, harmful effects on oral health compared to conventional cigarettes. This result may be attributed to the majority of uploaders being dentists/specialists 4.05% (48/102) or hospitals/dental clinics 26.47% (27/102). The use of e-cigarettes may lead to higher levels of nicotine addiction, potentially increasing the likelihood of quitting to cigarette smoking.[35,36]

Smoking reduces blood circulation in the mouth, facilitating the formation and progression of oral cancers.[37] In videos shared by specialists, it is stated that approximately 90% of those with mouth and throat cancer use tobacco or have used it for a while. Garg et al.[20] have highlighted that healthcare professionals, academic institutions, and professional organizations could enhance the quality of oral cancer-related content on YouTube™ by sharing informative videos and guiding patients toward trustworthy sources of information in India. In line with the findings of this study, the videos shared by periodontologists and surgeons regarding the effects of smoking on oral health included pre-cancerous lesions such as leukoplakia and hairy tongue, emphasizing the importance of early diagnosis. The videos shared by the professionals examined in this study stated that individuals who start smoking before the age of 16 have a higher risk of developing oral cancer and that the combination of smoking and alcohol is responsible for 90% of oral cancers. The lack of

references for these statements may explain the low DISCERN and JAMA scores.

Smoking is known to have adverse effects on oral health, including an increased risk of implant failure and complications.[38] Smoking may affect the success rate of dental implants by impairing osseointegration and reducing the body's ability to limit and attenuate disease progression infections.[39] It may also lead to reduced bone density, which is essential for implant stability and integration. In line with the study of Bulut et al.[40] examining YouTube™ and peri-implantitis, the present research similarly revealed that the uploaders of the analyzed videos emphasized the role of smoking as a significant risk factor for peri-implantitis.

An examination of video viewership patterns reveals that videos with a duration of less than 3 minutes tend to attract significant attention from viewers, as in a previous study.[41] However, it should be noted that these videos often suffer from poorer video quality. Moreover, the YouTube™ platform offers duration filters that enable users to refine their search based on video length. Notably, in scientific settings, there may be a clear correlation between the duration of a video and the attention of the viewers, with an evident decline as the video length increases. This underscores the critical importance of creating concise and focused video content to maximize viewer engagement. This study revealed that individuals are more interested in lasting 4-5 minutes of videos.

Owing to the functioning of the YouTube™ algorithm, social media content creators can make alterations to their videos and modify comments and associated data. Such modifications can lead to variations in search results during different periods. It is important to note that the evaluation for this study involved a search conducted on 16 May 2023; thus, the outcomes may differ subsequently. The calculation of interaction index and dislike data was not feasible due to YouTube's decision to remove the visibility of dislike counts for videos.

Grönholm et al.[42] indicated a need for increased awareness and education regarding local smoking cessation treatment guidelines and effective cessation interventions within dental practice, aiming to address the existing obstacles and enhance the promotion of successful smoking cessation efforts. Although there were videos emphasizing the necessity of smoking cessation for oral health in this study, Richardson et al.[12] and Grönholm et al., found that there was limited focus on delivering a comprehensive explanation. Videos explaining the adverse effects of smoking on oral health should provide viewers with accurate and up-to-date information to help them learn proper oral care practices and maintain oral health. Furthermore, in videos uploaded by dentists/professionals, greater emphasis should be aimed on increasing oral health awareness among viewers and highlighting the importance of quitting smoking with more scientific content.

Limitations

The limitation of this study conducted on YouTube is that the platform's search algorithm filters results based on user history, location, and its current ranking criteria (relevance, view count, engagement). This can lead to different results being listed for searches performed at different times or on different devices using the same keywords. This can create difficulties in conducting comprehensive and consistent analyses. The research was limited to English keywords only. This resulted in the exclusion of high-quality scientific content or diverse clinical approaches produced in other languages (Turkish, Spanish, Chinese, etc.) from the analysis.

CONCLUSION

As social media platforms continue to play an increasingly significant role in society, it may become paramount to establish quality-control mechanisms to ensure information accuracy and reliability, particularly in health-related content on platforms like YouTube™. As a result, the quality of smoking and oral health-related videos needs to be improved. In particular, there is a need for comprehensive scientific videos prepared by specialists.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study did not require ethical approval as it did not involve any human subjects or animal experiments.

Informed Consent

Because the study has no study with human participants, no written informed consent form was obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: DH, EC, HEO; Design: DH, EC, HEO; Control: DH, EC, HEO; Resources: DH, EC, HEO; Materials: DH, EC, HEO; Data Collection and/or Processing: DH, EC, HEO; Analysis and/or Interpretation: DH, EC, HEO; Literature Review: DH, EC, HEO; Writing the Article: DH, EC, HEO; Critical Review: DH, EC, HEO.

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Retrospective evaluation of the effects of non-surgical periodontal therapy on clinical periodontal parameters and deep periodontal pocket burden

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ABSTRACT

Aims: This study investigated the impact of initial non-surgical periodontal therapy (NSPT) on clinical periodontal status and deep periodontal pocket burden (DPPB) in individuals with periodontitis, with a focus on how baseline disease severity relates to treatment response.

Methods: Clinical data from 38 patients treated for periodontitis at the Periodontology Clinic of Adıyaman University were analyzed retrospectively. Periodontal measurements were recorded at baseline and at a 6-8-week follow-up. Evaluated parameters included probing pocket depth (PPD), Plaque Index, and Gingival Index. DPPB was quantified as both the number and percentage of sites with PPD≥5 mm.

Results: At the post-treatment evaluation, all periodontal parameters indicated a clear shift toward improved clinical conditions ($p<0.001$). Mean and maximum probing depths were lower compared to baseline values, accompanied by a marked decrease in both the number and proportion of deep periodontal pockets. Improvements were also evident in plaque accumulation and gingival inflammation scores. Moreover, higher baseline pocket burden was associated with greater reductions in probing depth ($r=0.681$, $p<0.001$).

Conclusion: NSPT was associated with notable clinical recovery and a reduction in inflammatory burden. Assessing DPPB may enhance the evaluation of treatment response and support clinical decision-making for further periodontal management.

Keywords: Periodontitis, non-surgical therapy, periodontal pocket burden

INTRODUCTION

Periodontal diseases represent one of the most frequently encountered groups of oral conditions worldwide, with gingivitis and periodontitis being the main clinical forms.[1] Periodontitis is a chronic inflammatory disease of multifactorial origin that leads to the gradual breakdown of the tooth-supporting tissues, including clinical attachment loss, periodontal pocket formation, and alveolar bone resorption.[2]

Periodontitis, beyond affecting oral health, negatively impacts patients' quality of life and also places a significant economic burden on governments in the healthcare sector.[3] Its prevalence increases with advancing age, with a more pronounced rise typically observed from early adulthood onwards.[4] Increasing evidence suggests that periodontitis extends beyond the oral cavity and may contribute to the onset or progression of various systemic conditions, including diabetes mellitus,[5] rheumatoid arthritis,[6] respiratory diseases,[7] cardiovascular diseases,[8] adverse pregnancy outcomes,[9] and neurodegenerative disorders.[10]

The main goals of periodontal treatment (PT) are to prevent disease progression and overall tooth loss, as well as to preserve the natural tooth structure and improve quality of life. Achieving these goals depends on accurate diagnosis and careful evaluation of disease severity by dental professionals.[1] The diagnosis of periodontal disease currently relies on both clinical and radiographic assessments.[11] Clinical evaluation mainly involves visual examination of the periodontal soft tissues together with periodontal probing. In addition, periodontal probing allows the detection of subgingival calculus and irregularities on root surfaces.[12] Clinical indicators including clinical attachment loss (CAL), probing pocket depth (PPD), plaque index (PI), and Gingival Index (GI) are commonly utilized to diagnose periodontitis, assess disease severity, and evaluate treatment outcomes.[13,14] Periodontal pockets are among the most characteristic clinical features of periodontitis. These structures create a favorable environment for the accumulation of pathogenic microorganisms and contribute to the continuation of the inflammatory response through persistent subgingival biofilm.[15] In clinical examination, the distance between the free gingival margin and the base of the gingival sulcus should be measured using a standardized graduated periodontal probe to determine the

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presence of periodontal pockets. PPD is considered an indirect indicator of the patient's inflammatory status, as inflamed periodontal tissues exhibit reduced resistance to probing, which is clinically perceived as increased probing depth[12]. In periodontal health, probing depths are generally expected to be ≤3 mm in the absence of CAL, together with a full-mouth bleeding on probing score of <10%.[16] D'Aiuto et al.[17] categorized 4-5 mm pockets as moderate pockets and pockets ≥6 mm as deep pockets at both baseline and re-evaluation. Similarly, in the 2017 World Workshop Classification of Periodontal and Peri-Implant Diseases and Conditions, probing pocket depth was recognized as one of the principal clinical parameters reflecting disease complexity. According to this classification, the maximum PPD is generally ≤4 mm in Stage I cases and ≤5 mm in Stage II cases, whereas periodontal pockets ≥6 mm are considered among the complexity criteria for Stage III and Stage IV periodontitis.[18]

Scaling and root planning (SRP) is widely accepted as the standard non-surgical periodontal therapy (NSPT) approach for periodontitis, primarily aiming to reduce inflammation and decrease periodontal pocket depth.[19] As pocket depth increases, the effective removal of subgingival deposits becomes more challenging, and residual calculus is more frequently detected in deeper pockets compared to shallower sites.[13] NSPT has been shown to significantly reduce periodontal disease burden.[20,21] Nevertheless, a number of periodontal pockets, commonly referred to as "residual pockets (RPs)" frequently persist following non-surgical treatment.[22-24] Residual periodontal pockets are defined as periodontal pockets that remain after non-surgical periodontal therapy. Previous studies have demonstrated that RPs, particularly those measuring ≥5 mm, represent important risk factors for periodontal disease progression and tooth loss. In addition, RPs with depths ≥6 mm have been associated with a greater risk of continued periodontal breakdown and often require advanced periodontal or surgical intervention. Therefore, the elimination of PPD≥5 mm has been emphasized as one of the primary objectives of periodontal therapy.[23]

Therefore, relying only on mean probing depth may not fully reflect the clinical condition of the patient. Evaluating both the number and distribution of periodontal pockets exceeding specific threshold values may provide a more comprehensive assessment of treatment response. In this context, the concept of "deep periodontal pocket burden (DPPB)" defined as the number and proportion of sites exceeding predefined thresholds (e.g., ≥5 mm or ≥6 mm), may serve as a quantitative indicator of disease severity and the distribution of clinically critical sites within the dentition. Previous studies evaluating the effectiveness of NSPT have mainly focused on mean changes in PPD and CAL. In contrast, limited attention has been given to the distribution and burden of clinically critical deep periodontal pockets persisting after treatment. In particular, the relationship between baseline DPPB and the early clinical response to NSPT has not been sufficiently investigated.

Therefore, the present study aimed to evaluate the effects of initial NSPT on clinical periodontal parameters and DPPB in patients with periodontitis, as well as to investigate the relationship between baseline periodontal pocket burden and early treatment response.

METHODS

Study Design and Patient Population

This retrospective clinical study was conducted using the clinical records of patients diagnosed with periodontitis and treated with NSPT at the Periodontology Clinic of Adıyaman University Faculty of Dentistry. The study protocol was approved by the Adıyaman University Non-interventional Clinical Researches Ethics Committee (Date: 24.03.2026 Decision No: 2026/2-6). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Patients with complete clinical records at baseline (T0) and at 6-8 weeks post-treatment (T1) were included in the study. Patients with incomplete clinical data or missing periodontal measurements were excluded. Out of a total of 122 patients, 38 met the inclusion criteria and were enrolled in the study. All periodontal examinations and initial treatments were performed by a single specialist.

Initial Periodontal Therapy Protocol

All patients received standardized NSPT. The treatment protocol included oral hygiene, tartar removal, and SRP. All procedures were performed using ultrasonic devices and conventional hand instruments. No surgical periodontal treatment or adjunctive pharmacological agents were administered during the study period. Patients were instructed to brush their teeth (twice daily) and to perform interdental cleaning once daily using dental floss or interdental brushes.

Clinical Periodontal Measurements

Clinical periodontal evaluations were performed at baseline (T0) and at 6-8 weeks (approximately 2 months) following treatment (T1). Probing pocket depth (PPD) measurements were recorded at six sites per tooth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) using a Williams periodontal probe (Hu-Friedy, Chicago, IL, USA). Missing teeth were excluded from all calculations.

Mean Probing Pocket Depth

For each patient, mean probing pocket depth at T0 and T1 was calculated as the arithmetic mean of all recorded PPD values across measurement sites.

Deep Periodontal Pocket Burden (PPD≥5 mm)

DPPB was defined, for each patient at T0 and T1, as the total number of sites with PPD≥5 mm. In addition, to better reflect the distribution of deep pockets, the proportion (%) of sites with PPD≥5 mm was calculated by dividing the number of such sites by the total number of measurement sites (number of present teeth ×6).

Maximum Probing Pocket Depth

For each patient, the maximum probing pocket depth was defined as the highest PPD value recorded among all measurement sites at T0 and T1.

Plaque and Gingival Indices

Oral hygiene status and gingival inflammation were assessed using the PI and GI described by Löe and Silness.[16] Both indices were scored on a scale of 0- 3. For each patient, PI and GI values at T0 and T1 were calculated as the mean of the

scores obtained from all present teeth. Missing teeth were excluded from index calculations.

Demographic and Anamnestic Data

Demographic and medical history data, including age, sex, smoking status, and presence of systemic disease (hypertension, diabetes mellitus, and cardiovascular diseases), were obtained from patient records. The presence of systemic disease was recorded as a binary variable (yes/no).

Statistical Analysis

Data analyses were performed using the IBM SPSS Statistics software package. The normality of data distribution was assessed using the Shapiro-Wilk test. As the data did not follow a normal distribution, non-parametric tests were applied. Comparisons between T0 and T1 clinical parameters were performed using the Wilcoxon signed-rank test. Differences in changes (Δ) of periodontal parameters according to smoking status and presence of systemic disease were analyzed using the Mann-Whitney U test. Associations between changes in clinical parameters were evaluated using Spearman correlation analysis. A p-value of <0.05 was considered statistically significant. Sample size analysis was performed using G*Power software version 3.1 (Heinrich Heine University, Düsseldorf, Germany). Based on an a priori power analysis for the Wilcoxon signed-rank test, assuming a large effect size (dz=0.80), a significance level of α=0.05, and a statistical power of 95%, the minimum required sample size was calculated as 20 participants.

RESULTS

A total of 38 patients with periodontitis were included in the analysis. The mean age was 37.2±9.5 years (range: 25-61 years). Among the participants, 28.9% were smokers and 44.7% presented with at least one systemic condition. The most frequently observed systemic conditions were hypertension (n=10) and diabetes mellitus (n=6). The average number of remaining teeth was 26.7±1.8 (**Table 1**).

Table 1. Demographic and baseline clinical characteristics of the study population

Variable	Value
n	38
Age (years), mean±SD	37.2±9.5
Age range (min-max)	25-61
Sex (female/male), n (%)	27 (71.1%)/11 (28.9%)
Smokers, n (%)	11 (28.9%)
Presence of systemic disease, n (%)	17 (44.7%)
Hypertension, n (%)	10 (26.3%)
Diabetes mellitus, n (%	6 (15.8%)
Cardiovascular disease, n (%)	3 (7.9%)
Number of present teeth, mean±SD	26.7±1.8
Mean probing pocket depth (mm)_T0	3.39±0.75
Number of pockets ≥5 mm_T0	25.6±15.5
Proportion of pockets ≥5 mm (%), T0	24.2±14.9
Number of pockets ≥6 mm, T0	9.6±11.0
Maximum probing pocket depth (mm), T0	8.18±1.47
PI_T0	1.48±0.59
GI_T0	1.60±0.56

SD: Standard deviation, Min: Minimum, Max: Maximum, T0: Baseline (pre-treatment), PI: Plaque Index, GI: Gingival Index

Following the initial periodontal therapy, all evaluated clinical parameters improved markedly at the 6-8-week follow-up. Mean probing pocket depth decreased from 3.39±0.75 mm at baseline to 2.93±0.45 mm after treatment (p<0.001). A pronounced reduction was also observed in deep periodontal pocket burden. The number of sites with PPD≥5 mm declined from 25.6±15.5 to 10.8±10.7, while the corresponding percentage decreased from 24.2%±14.9 to 10.3%±10.3 (p<0.001). Similarly, sites with PPD≥6 mm showed a substantial decrease, with mean values dropping from 9.6±11.0 at baseline to 3.9±6.2 after treatment (p<0.001). The proportion of these sites also demonstrated a statistically significant reduction. Maximum probing pocket depth was reduced from 8.18±1.47 mm to 7.05±1.89 mm following therapy (p<0.001). In addition, clear improvements were recorded in oral hygiene and gingival inflammation parameters. PI values decreased from 1.48±0.59 to 0.44±0.23, while GI values declined from 1.60±0.56 to 0.72±0.39 (p<0.001 for both) (**Table 2**).

Table 2. Changes in clinical periodontal parameters following initial periodontal therapy

Parameter	T0 (mean±SD)	T1 (mean±SD)	p-value	r-value
Mean probing pocket depth (mm)	3.39±0.75	2.93±0.45	<0.001*	0.87
Number of pockets ≥5 mm	25.6±15.5	10.8±10.7	<0.001*	0.87
Proportion of pockets ≥5 mm (%)	24.2±14.9	10.3±10.3	<0.001*	0.87
Number of pockets ≥6 mm	9.6±11.0	3.9±6.2	<0.001*	0.78
Proportion of pockets ≥6 mm (%)	9.15±10.64	3.70±5.91	<0.001*	0.78
Maximum probing pocket depth (mm)	8.18±1.47	7.05±1.89	<0.001*	0.64
PI	1.48±0.59	0.44±0.23	<0.001*	0.87
GI	1.60±0.56	0.72±0.39	<0.001*	0.87

*Statistically significant (p<0.05). r: Effect size (Wilcoxon signed-rank test), T0: Baseline (pre-treatment), T1: 6-8 weeks post-treatment, SD: Standard deviation, PI: Plaque Index, GI: Gingival Index

Effect size analysis indicated that all clinical changes were of large magnitude, with values ranging between r=0.64 and r=0.87. No significant differences were identified in treatment-related changes when patients were grouped according to smoking status or the presence of systemic disease (p>0.05) (**Table 3, 4**).

Table 3. Changes in periodontal parameters according to smoking status (Δ)

Parameter	Non-smokers (n=27), mean±SD	Smokers (n=11), mean±SD	p-value*
ΔPPD (mm)	0.47±0.53	0.43±0.45	0.987
ΔNumber of pockets ≥5 mm	15.85±9.90	12.00±9.05	0.245
ΔNumber of pockets ≥6 mm	5.85±8.32	5.36±8.69	0.758
ΔPI	1.00±0.49	1.02±0.46	0.961
ΔGI	0.91±0.49	0.81±0.46	0.421
ΔMaximum PPD (mm)	1.15±1.23	1.09±1.45	0.770

*p<0.05 indicates statistical significance (Mann-Whitney U test). Δ: Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, PI: Plaque Index, GI: Gingival Index

Correlation analysis revealed a strong positive relationship between baseline deep pocket burden and the reduction in mean probing depth (r=0.681, p<0.001). A similar positive association was observed between baseline pocket burden and

Table 4. Comparison of changes (Δ) in periodontal parameters according to the presence of systemic disease

Parameter	No systemic disease (n=21), mean±SD	Systemic disease present (n=17), mean±SD	p-value*
ΔPPD (mm)	0.54±0.63	0.36±0.26	0.860
ΔNumber of pockets ≥5 mm	16.19±10.37	12.94±9.23	0.418
ΔNumber of pockets ≥6 mm	6.38±9.36	4.94±7.39	0.451
ΔPI	1.08±0.50	0.99±0.45	0.628
ΔGI	0.93±0.50	0.82±0.45	0.428
ΔMaximum PPD (mm)	1.38±1.28	0.82±1.24	0.154

*p<0.05 indicates statistical significance (Mann-Whitney U test). Δ: Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, PI: Plaque Index, GI: Gingival Index

the decrease in GI values (r=0.548, p<0.001). Furthermore, reductions in probing depth were positively correlated with improvements in gingival inflammation (r=0.627, p<0.001) (**Table 5**).

Table 5. Spearman correlation analysis between baseline periodontal pocket burden and changes in clinical parameters

Variables	1. Baseline proportion of sites with PPD ≥5 mm	2. ΔPPD	3. ΔGI
1. Baseline proportion of sites with PPD ≥5 mm	1		
2. ΔPPD	0.681*	1	
3. ΔGI	0.548*	0.627*	1

*r: Spearman correlation coefficient; p<0.001 indicates statistical significance. Δ: Change between T0 and T1; SD: Standard deviation, PPD: Probing pocket depth, GI: Gingival Index

DISCUSSION

PPD is widely used as a clinical parameter for evaluating periodontal status and monitoring treatment outcomes.[25] However, mean PPD values alone may not adequately reflect the clinical relevance of localized deep periodontal pockets. In daily clinical practice, the persistence of deep pockets after initial therapy is often a determining factor in deciding whether further periodontal intervention is required.[20]

In this regard, the present study highlights the importance of assessing DPPB as an additional parameter. The findings demonstrated that NSPT led to substantial reductions not only in mean probing depth but also in both the number and proportion of deep periodontal pockets. This indicates that initial therapy contributes to a reduction in overall disease burden rather than merely improving average clinical measurements.

Despite the overall improvements, deep periodontal pockets (>5mm) were not completely eliminated in all patients. This is clinically significant, as RPs-particularly those exceeding 4 mm-may serve as sites prone to disease recurrence. When evaluated together with clinical indicators such as bleeding on probing or suppuration, these RPs may represent ecological niches that facilitate bacterial persistence and increase the risk of reinfection.[26] Ramseier et al.[27] reported that, for maintaining periodontal stability, the number of periodontal pockets with probing depths ≥5 mm or ≥6 mm may be considered an important indicator of periodontal deterioration and that these sites should be taken into account for additional periodontal treatment or shorter recall intervals. Therefore, the ≥5 mm threshold used in the present study represents not only disease severity but also a clinically relevant residual periodontal pocket burden associated with future periodontal progression and treatment requirements.[27] In a long-term retrospective study published by Saleh et al.,[28] the presence of RPs following periodontal therapy, particularly an increased proportion of RPs≥5 mm, was identified as a significant risk factor for periodontal-related tooth loss. Furthermore, the presence of RPs≥5 mm in more than 15% of patients was shown to increase the risk of periodontal tooth loss. The authors emphasized that RPs have important prognostic value in predicting periodontal progression and should be carefully considered during post-treatment evaluation.[28] Consistent with these findings, previous studies have reported that periodontal pockets smaller than 4 mm are generally associated with periodontal stability,[29] whereas pockets greater than 6 mm are associated with an increased risk of disease progression and future tooth loss.[30] In line with these findings, the present study demonstrated significant reductions in probing depth, as well as in the number and proportion of deep periodontal pockets. Additionally, marked improvements were observed in PI and GI values. These findings support the effectiveness of NSPT in controlling inflammation and improving clinical periodontal conditions.[31-33] Citterio et al.[33] reported that although NSPT eliminates a substantial proportion of periodontal pockets, RPs frequently persist and should be carefully considered during further treatment planning. In particular, residual pockets ≥5 mm were emphasized as important risk indicators for periodontal disease progression and tooth loss. The authors also stated that evaluating the effectiveness of NSPT not only through mean PPD reduction but also through the number and proportion of residual pockets may provide more clinically meaningful outcomes. In the aforementioned meta-analysis, the proportion of pockets ≥5 mm decreased from 28.23% to 11.71% following NSPT; however, patients still presented with a mean of 14.13 residual pockets ≥5 mm after treatment. Furthermore, the authors highlighted that the incomplete elimination of residual pockets ≥5 mm may indicate the need for additional periodontal therapy and may be important for maintaining periodontal stability.[33]

Feres et al.[34] proposed that the presence of "≤4 sites with PD≥5 mm" following periodontal therapy may be used as a clinical endpoint (treat-to-target endpoint) for evaluating disease control. Similarly, the concept of the "successfully treated stable periodontitis patient," defined in the 2017 World Workshop Classification of Periodontal and Peri-Implant Diseases and Conditions, describes periodontal stability as the presence of PD≤4 mm, the absence of sites with PD≥4 mm accompanied by bleeding on probing, and low full-mouth bleeding scores.[35] In a long-term supportive periodontal therapy study conducted by Bertl et al.,[36] periodontal risk was shown to increase in patients who failed to achieve a successfully treated stable periodontitis status. These findings support the concept that periodontal treatment success should be evaluated not only through mean PPD reduction but also by considering the distribution and magnitude of residual DPPB. The DPPB approach used in the present study is consistent with this clinical perspective. Despite improvements in overall periodontal parameters, residual deep periodontal pockets persisted in some patients. This finding suggests that evaluating DPPB may be clinically useful for identifying persistent disease activity and the potential need for further periodontal treatment following therapy.

Studies have reported that sites with deeper baseline periodontal pockets tend to exhibit greater reductions in probing pocket depth following NSPT. However, it has also been demonstrated that the clinical response to periodontal therapy may not occur uniformly across all periodontal parameters. Raittio et al.[37] reported that following NSPT, more homogeneous improvements were observed in PPD, bleeding on probing (BOP%), and the proportion of shallow pockets (PPD%≤3 mm), whereas greater inter-individual variability was detected in the CAL response. The authors suggested that this variability in CAL may be influenced by several factors, including baseline disease severity, differences in inflammatory response, measurement sensitivity, and the biological heterogeneity of periodontal destruction. Furthermore, it was emphasized that the greater PPD reduction observed in deeper periodontal pockets may result in a more predictable clinical response in PPD-related parameters following NSPT.[37] PPD quantitatively reflects the current periodontal condition without accounting for changes in the gingival margin position. The gingival margin itself does not represent a fixed reference point in relation to the cementoenamel junction. In contrast, CAL is considered a diagnostic parameter used to quantify periodontal attachment loss over longer periods of time. Recent studies have suggested that CAL reflects long-term periodontal destruction, whereas PPD may better represent current disease activity and the biological processes associated with periodontal progression. In the 11-year follow-up study conducted by Matuliene et al.,[30] similar conclusions were reported regarding sites with residual PPD≥6 mm. The authors also stated that higher CAL values negatively affected periodontal prognosis. In particular, sites with CAL≥7 mm were associated with a significantly increased risk of tooth loss[30]. Therefore, CAL changes, together with PPD, should be considered important clinical parameters in the evaluation of periodontal treatment outcomes. Although CAL measurements were not evaluated in the present study, the marked reduction in DPPB supports the effectiveness of NSPT in reducing periodontal inflammation and promoting pocket elimination.

An important observation of the present study, consistent with previous reports, was the positive relationship between baseline deep periodontal pocket burden and the degree of clinical improvement. Patients with a higher initial disease burden demonstrated greater reductions in probing pocket depth and gingival inflammation following treatment. This finding may be attributed to the higher inflammatory burden present in these sites, where the removal of subgingival biofilm results in a more pronounced therapeutic response. Furthermore, the significant association between reductions in probing depth and improvements in gingival index values indicates that changes in inflammation and pocket depth are closely interconnected. These findings suggest that the resolution of periodontal inflammation and the reduction of probing pocket depth should be considered interrelated outcomes rather than independent processes.

Accordingly, the response of the soft tissues to NSPT represents an important clinical criterion for treatment evaluation. This response is typically assessed during reevaluation appointments following SRP.[38,39] Previous studies have reported that the most pronounced reductions in probing depth and gains in clinical attachment occur within the first 1-3 months after SRP, whereas complete periodontal healing and maturation may continue for up to 9-12 months.[40-43] The timing of re-evaluation after initial NSPT is another important consideration. It has been reported that a 4-8 week interval is appropriate for assessing treatment outcomes.[44] During this period, a substantial proportion of periodontal pockets may resolve following successful NSPT, and when adequate debridement is achieved, early clinical improvements such as reductions in probing depth, gains in clinical attachment, and control of bleeding on probing may be observed.[38,39] The 6-8-week follow-up period in the present study reflects the early healing and reevaluation phase following non-surgical periodontal therapy. Therefore, the study was primarily designed to assess the early clinical response to initial NSPT rather than long-term periodontal stability. Nevertheless, longer follow-up studies are needed to determine the long-term sustainability of changes in deep periodontal pocket burden. In this regard, in a recent study published by Liss et al.,[45] patients were reevaluated at 3 and 6 months following initial NSPT. Since RPs were still present at the 3-month evaluation, re-instrumentation was performed; however, deep RPs still persisted in approximately 30% of patients at 6 months. At the 18-month evaluation, pocket closure was achieved in approximately 39% of RPs measuring 5-6 mm following re-instrumentation, whereas this rate decreased to 28% in RPs≥7 mm. The authors also reported that non-surgical re-instrumentation of RPs may provide additional improvements in periodontal health conditions.[45]

Importantly, besides these, NSPT plays a significant role in reducing inflammation and preparing periodontal tissues, thereby improving the predictability of subsequent surgical procedures.[46]

Limitations

The retrospective design and relatively small sample size are factors that limit the generalizability of the study. However, despite the moderate sample size, the paired longitudinal design increased statistical efficiency by reducing inter-individual variability, and the observed effect sizes for the primary periodontal outcomes were substantial. Furthermore, although smoking status and systemic diseases did not show statistically significant associations with treatment outcomes in the present study, this finding should be interpreted cautiously due to the limited sample size and potential heterogeneity of the study population. Despite these limitations, the present study provides clinically relevant insights by emphasizing the role of deep periodontal pocket burden as an outcome parameter. In addition, the absence of clinical attachment level measurements represents another limitation in the comprehensive evaluation of periodontal status. Evaluating both the number and distribution of deep periodontal pockets may offer a more comprehensive assessment of treatment response compared to mean probing depth alone.

CONCLUSION

NSPT leads to significant improvements in clinical periodontal parameters by effectively reducing periodontal inflammation. In evaluating treatment outcomes, reliance solely on changes in mean probing pocket depth may be insufficient. Consideration of the number and distribution of deep periodontal pockets provides a more comprehensive assessment of periodontal disease burden. This approach

may enhance the clinical evaluation of treatment success and support more informed decision-making regarding the need for further periodontal intervention.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Adıyaman University Non-interventional Clinical Researches Ethics Committee (Date: 24.03.2026 Decision No: 2026/2-6).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Author Contributions

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Success rate of apical resection: a retrospective evaluation of associated factors

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ABSTRACT

Aims: The aim of this retrospective study was to evaluate the long-term success rate of apical resection procedures and to investigate factors potentially associated with treatment outcomes under routine clinical conditions.

Methods: Clinical records of patients who underwent apical resection at a single academic center between January 2015 and January 2025 were retrospectively reviewed. Demographic variables, tooth characteristics, root-end filling materials, and operator type were recorded. Treatment outcome was assessed based on the absence of clinical symptoms and radiographic reduction in periapical lesion size. Preoperative and follow-up periapical radiographs were calibrated and analyzed using ImageJ software. Statistical analyses were performed using Pearson's Chi-square test or Fisher's exact test for categorical variables. Variables potentially associated with treatment outcome were further evaluated using multivariate binary logistic regression analysis. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to determine independent predictors of treatment success. Inter-observer reliability was assessed using intraclass correlation coefficient (ICC) analysis.

Results: A total of 357 apical resection cases were included, with a mean follow-up period of 72 months (range: 18-120 months). Thirty-six teeth were extracted due to failure of apical resection and were therefore classified as unsuccessful outcomes. Among the remaining 321 cases, successful healing was observed in 240 cases, while unsuccessful healing was observed in 81 cases. Statistically significant associations with treatment outcome were observed for sex, jaw location, operator type, and root-end filling material ($p<0.001$). Multivariate logistic regression analysis demonstrated that sex, root-end filling material, and root morphology were independently associated with treatment outcome.

Conclusion: Within the limitations of this retrospective study, apical resection demonstrated acceptable long-term outcomes under routine clinical conditions. Treatment outcomes appeared to be associated with several clinical and procedural factors, particularly root-end filling material and root morphology.

Keywords: Apical surgery, endodontic microsurgery, retrospective evaluation, treatment outcome

INTRODUCTION

Current evidence highlights the central role of microorganisms in the pathogenesis of pulpal and periradicular diseases; therefore, the primary objective of endodontic treatment is the elimination of infection and the establishment of a long-term seal that prevents bacterial recontamination of the periapical tissues.[1] Conventional orthograde root canal treatment generally demonstrates high success rates. However, when primary treatment or nonsurgical retreatment fails-or when retreatment is not feasible due to anatomical, restorative, or procedural limitations-surgical intervention becomes a necessary alternative.[2]

In such cases, apical surgery, particularly modern endodontic microsurgery (EMS), aims to preserve teeth affected by persistent apical periodontitis. The procedure typically includes root-end resection, ultrasonic root-end cavity preparation, retrograde filling with biocompatible materials, and thorough removal of pathological periapical tissues.[3] Advances in magnification, illumination, ultrasonic instrumentation, cone-beam computed tomography (CBCT), and calcium silicate-based root-end filling materials have substantially improved the predictability and outcomes of EMS.[4,5]

Recent studies consistently report favorable short- and long-term outcomes for EMS. A 2023 clinical study reported a 96.3% success rate at one-year follow-up in teeth with persistent apical periodontitis after failed nonsurgical treatment.[6] Similarly, a 2024 investigation using a simplified microsurgical protocol in selected single-rooted teeth demonstrated sealing ability and clinical outcomes comparable to those of conventional EMS.[7] Furthermore, a systematic review and meta-analysis published in 2021 reported pooled long-term success rates of approximately 91.3% in randomized clinical trials and 78.4% in prospective clinical studies, with follow-up periods ranging from 2 to 13 years.[8]

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Radiographic assessment remains the cornerstone for evaluating healing following apical surgery. The classic radiographic classification proposed by Rud and Molven[9] categorizes healing as complete, incomplete, uncertain, or unsatisfactory. Although strict criteria consider only complete and incomplete healing as successful outcomes, several studies have shown that a substantial proportion of lesions classified as "uncertain" at one-year follow-up subsequently progress to successful healing.[10] Consequently, many contemporary studies adopt more lenient criteria, defining success as a measurable reduction in radiolucency rather than complete resolution alone-particularly when CBCT imaging is used.[11]

Despite the generally favorable outcomes reported for EMS, long-term success is influenced by multiple prognostic factors, including tooth type, preoperative lesion size, crestal bone level, the presence of through-and-through defects, and patient-related variables.[12] Therefore, evaluating real-world data from long-term retrospective studies remains essential to better understand the effectiveness and predictability of apical surgery in routine clinical practice.

Accordingly, the present retrospective study evaluates all apical resection procedures performed at our institution over a 10-year period, aiming to determine overall success rates and to assess associated prognostic factors within the context of contemporary microsurgical standards.

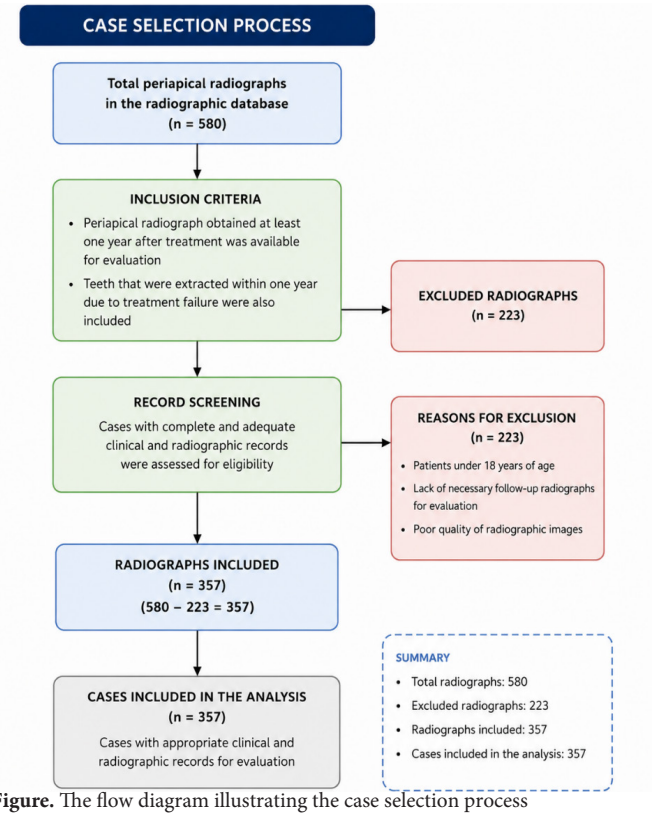

Figure. The flow diagram illustrating the case selection process

METHODS

The study was conducted in accordance with the principles of the Declaration of Helsinki (2013 revision). Ethical approval was obtained from the Karabük University Non-interventional Clinical Researches Ethics Committee (Date: 26.06.2024, Decision No: 2024/1856). As this study had a retrospective design, the requirement for individual informed consent was waived.

Clinical records of all patients who underwent apical resection at the Karabük University Oral and Dental Health Training and Research Hospital between January 2015 and January 2025 were retrospectively reviewed. Demographic variables, including sex, and systemic conditions, as well as tooth number and the type of retrograde filling material used, were extracted from patient charts.

Cases were included if periapical radiographs obtained at least one year after treatment were available for evaluation. Teeth extracted within one year due to treatment failure were also included in the analysis. Cases with incomplete or insufficient clinical or radiographic records were excluded. Of the 580 radiographs initially identified in the radiographic database, 223 were excluded from the study. Reasons for exclusion included age below 18 years, absence of the required follow-up radiographs, and poor radiographic image quality.

Evaluation Criteria for Success

Outcome measures comprised clinical signs and symptoms, including pain, swelling, and sinus tract formation, as well as radiographic changes in the periapical lesion area. Healing was considered successful when (a) no clinical signs or symptoms were present and (b) radiographic evaluation demonstrated a reduction in lesion size. Healing was classified as unsuccessful when clinical symptoms persisted or when the lesion area remained unchanged or increased.

For multirooted teeth, failure in any individual root resulted in classification of the entire tooth as unsuccessful. Periapical bone healing was assessed by comparing calibrated preoperative and follow-up periapical radiographs. Because projection angles differed between radiographs, direct comparison of raw lesion measurements was not feasible. Therefore, the ImageJ TurboReg plugin was used to calibrate both images, after which radiolucent lesion areas were measured and compared using ImageJ software. Imaging procedures and radiographic devices were consistent with those used at the time of treatment.

To assess inter-observer reliability, all 321 radiographic measurements were independently re-evaluated by two experienced examiners. Inter-observer agreement was assessed using intraclass correlation coefficient (ICC) analysis based on a two-way mixed-effects model with absolute agreement. The ICC value was 0.91 ($p<0.001$), indicating excellent agreement between observers.

RESULTS

A total of 357 cases were included in this retrospective cohort study. The mean follow-up period was 72 months (range: 18-120 months). Thirty-six teeth were extracted due to failure of apical resection and were therefore classified as unsuccessful outcomes. Among the remaining 321 cases, successful healing was observed in 240 cases, while unsuccessful healing was observed in 81 cases.

Patient- and tooth-related characteristics of the included cases are summarized in **Table 1**.

Ninety cases involved mandibular teeth, whereas 231 involved maxillary teeth. Twelve teeth were multirooted, and 309 were single-rooted.

Table 1. Demographic and clinical characteristics of the study population

Variable	Category	n (%)
Sex	Female	167 (52.0)
	Male	154 (48.0)
Jaw location	Maxilla	231 (72.0)
	Mandible	90 (28.0)
Root morphology	Single-rooted	309 (96.3)
	Multirooted	12 (3.7)
Root-end filling material	MTA	87 (27.1)
	Other materials	234 (72.9)
Operator	Oral surgeon	231 (72.0)
	General dentsit	90 (28.0)

MTA: Mineral trioxide aggregate

Regarding root-end filling materials, 87 teeth were treated with mineral trioxide aggregate (MTA), whereas 234 teeth received other materials, including amalgam and intermediate restorative material (IRM). With respect to the operator, 231 procedures were performed by oral surgeons, whereas 90 procedures were performed by general dentists. Case allocation was randomly.

Comparisons between successful and unsuccessful cases are presented in **Table 2**. Statistically significant associations with treatment outcome were observed for sex, jaw location, operator type, and root-end filling material (p<0.001). Male patients, mandibular teeth, procedures performed by oral surgeons, and teeth treated with MTA demonstrated higher success rates. No statistically significant association was observed between root morphology and treatment outcome (p=0.783).

Table 2. Comparison of demographic and clinical variables according to treatment outcome

Variable	Failure, n (%)	Success, n (%)	p-value
Sex			<0.001
Female	123 (73.7)	44 (26.3)	
Male	25 (16.2)	129 (83.8)	
Jaw location			<0.001
Mandible	9 (10.0)	81 (90.0)	
Maxilla	139 (60.2)	92 (39.8)	
Operator			<0.001
General dentist	59 (65.6)	31 (34.4)	
Oral surgeon	89 (38.5)	142 (61.5)	
Root-end filling material			<0.001
Other materials	137 (58.5)	97 (41.5)	
MTA	11 (12.6)	76 (87.4)	
Root morphology			0.783
Single-rooted	142 (46.0)	167 (54.0)	
Multirooted	6 (50.0)	6 (50.0)	

MTA: Mineral trioxide aggregate

The results of the multivariate logistic regression analysis are presented in **Table 3**. Sex, root-end filling material, and root morphology were independently associated with treatment outcome. Male sex (OR=14.77, 95% CI: 6.49-33.60, p<0.001) and the use of MTA as a root-end filling material (OR=24.53, 95% CI: 7.68-78.34, p<0.001) were associated with higher odds of treatment success. In contrast, multirooted teeth demonstrated significantly lower odds of successful healing (OR=0.004, 95% CI: 0.001-0.031, p<0.001). After adjustment for potential confounding variables, jaw location and operator type were not independently associated with treatment outcome.

Table 3. Multivariate logistic regression analysis for treatment outcome

Variable	OR	95% CI	p-value
Sex	14.77	6.49-33.60	<0.001
Jaw location	0.32	0.09-1.19	0.089
Operator type	1.13	0.46-2.77	0.797
Root-end filling material	24.53	7.68-78.34	<0.001
Root morphology	0.004	0.001-0.031	<0.001

OR: Odds ratio, CI: Confidence interval

DISCUSSION

The primary limitation of this study is its single-center retrospective design, which may limit the generalizability of the findings and highlights the need for validation through multicenter prospective research. Nevertheless, recent evidence continues to support the predictability and clinical relevance of apical surgery. A 2024 prospective study reported an 88.9% complete healing rate at 12 months and demonstrated that the choice of root-end filling material may influence radiographic healing outcomes.[13] Similarly, a 2024 investigation using a simplified microsurgical protocol in selected short, single-rooted teeth reported sealing ability and clinical outcomes comparable to those achieved with conventional endodontic microsurgery.[7]

Long-term outcomes reported in the literature remain encouraging. A retrospective cohort study documented a 91.4% success rate at one-year follow-up, independent of lesion size, smoking status, or lesion histopathology.[12] In addition, a recent in vitro investigation demonstrated that the bond strength of retrograde filling materials was influenced primarily by the cavity preparation technique-tungsten carbide burs versus ultrasonic retro-tips-rather than by the type of filling material used.[14]

Recent systematic reviews have shown that advances in magnification, ultrasonic retro-preparation, and bioceramic materials have substantially improved the predictability of endodontic microsurgery, with reported success rates exceeding 90%.[8] These findings underscore the importance of appropriate case selection, surgical technique, material choice, and operator experience. They also emphasize the value of long-term retrospective studies, which provide real-world data and may reveal prognostic factors that are not always captured in controlled prospective trials.

Evidence suggests that the most pronounced reduction in periapical lesion size occurs shortly after treatment.[15] One-year follow-up studies report healing rates exceeding 85-90% after root canal treatment and approximately 90-95% after apical surgery.[12,16] Accordingly, and in line with the recommendations of Wu et al.,[17] a minimum follow-up period of one year was adopted in the present study.

Rud and Molven[18] proposed four radiographic healing categories-complete, incomplete, uncertain, and unsatisfactory. Although strict criteria consider only complete and incomplete healing as successful outcomes, longitudinal data indicate that a substantial proportion of lesions classified as

uncertain at one year subsequently progress to successful healing.[12] Consequently, many contemporary studies apply more lenient criteria, particularly when advanced imaging modalities are unavailable.

Given the anatomical complexity of the root canal system and the potential persistence of residual microorganisms, a reduction in lesion size may reflect a favorable host response without posing systemic risk.[15] Therefore, reduction in radiolucency size was adopted as the criterion for successful radiographic healing in the present study.

Periapical radiography remains widely used for postoperative evaluation because of its accessibility, low cost, and minimal radiation exposure. However, its two-dimensional nature limits the detection of buccolingual lesion extension. Previous studies have demonstrated that lesions appearing to heal on periapical radiographs may still show progression on CBCT images.[11] Accordingly, the absence of CBCT imaging represents an important limitation of this study. To address this limitation, CBCT will be incorporated into outcome assessment in future prospective investigations.

Overall, the success rate of endodontic microsurgery observed in this study was lower than the rates reported in recent systematic reviews.[8] This discrepancy may be attributed to the retrospective design and operator variability, as procedures were performed by both oral surgeons and general dentists. These findings further emphasize the potential influence of multiple clinical and procedural factors on surgical outcomes under routine clinical conditions.

Limitations

The prognosis of apical surgery is influenced by multiple clinical factors, including lesion characteristics, preoperative lesion size, presence of "through-and-through" lesions, root-end filling material, coronal restoration quality, systemic factors (e.g, diabetes, smoking) and operator experience. In the present study, statistically significant associations were observed between treatment outcome and several clinical variables, including sex, jaw location, operator type, and root-end filling material. Multivariate logistic regression analysis further demonstrated that sex, root-end filling material, and root morphology were independently associated with treatment outcome. However, these findings should be interpreted cautiously because of the retrospective design and the limited availability of certain clinically relevant variables, including lesion size, smoking status, systemic conditions, and coronal restoration quality. In addition, the limited number of multirooted teeth may have affected the stability of the regression model. Therefore, the regression results should be interpreted with caution.

CONCLUSION

Within the limitations of this retrospective study, apical resection demonstrated acceptable long-term outcomes under routine clinical conditions. Treatment outcomes appeared to be associated with several clinical and procedural factors, particularly root-end filling material and root morphology.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Karabük University Non-interventional Clinical Researches Ethics Committee (Date: 26.06.2024, Decision No: 2024/1856).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: NYÇ, ML; Design: NYÇ, ML; Control: NYÇ; Resources: ML; Materials: ML; Data Collection and/or Processing: NYÇ, ML; Analysis and/or Interpretation: NYÇ, ML; Literature Review: NYÇ; Writing the Article: NYÇ; Critical Review: NYÇ, ML.

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Review

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The effect of hormonal changes observed in female patients on periodontium

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ABSTRACT

In women, physiological processes such as puberty, the menstrual cycle, pregnancy, oral contraceptive use, and menopause cause significant fluctuations in estrogen, progesterone, and androgen levels, increasing the inflammatory response in gum tissues and affecting the course of periodontal disease. Numerous studies in the literature have shown that hormonal changes directly affect periodontal cells, modulate inflammatory cytokine levels, and shape tissue destruction and healing processes. Furthermore, it has been emphasized that during these hormonal periods, there is an increase in the frequency of conditions such as gingivitis, periodontitis, and pyogenic granuloma seen in women. In periodontology practice, it is clear that variable hormonal conditions must be taken into account in treatment and preventive approaches for female patients. This study aimed to evaluate the effects of physiological hormonal processes in women on periodontal tissues.

Keywords: Hormonal changes, periodontal diseases, women, estrogen, pregnancy, menopause

PERIODONTAL DISEASE

Periodontal diseases are among the most common health problems in society. The most well-known of these diseases are gingivitis and periodontitis. However, both diseases have different subtypes, which are classified according to factors such as age of onset, clinical symptoms, rate of progression, types of pathogenic microorganisms, and systemic effects. Gingivitis is an inflammatory condition that affects only the gums, without accompanying attachment or alveolar bone loss. Early symptoms of this disease include increased gingival crevicular fluid and bleeding during probing. In some cases, gingivitis can progress to periodontitis. Periodontitis is a disease in which attachment and bone loss occur due to inflammation of the tissues supporting the teeth. Although it is usually chronic, factors such as diabetes, smoking, and stress can affect the host's response to bacterial plaque and increase the rate of disease progression.[1]

Periodontal Disease Etiology

The fundamental cause of periodontal disease is the disruption of subgingival microbiota balance over time due to an increase in disease-associated bacteria and a decrease in healthy bacteria. This change leads to a loss of homeostasis between the host and the microbiota.[2] The development of the disease is a gradual process, beginning with the adhesion and proliferation of both gram-negative and gram-positive bacteria on the tooth surface.[3] Over time, these bacteria colonize the subgingival region and alter the environmental conditions. The resulting environment becomes favorable for the growth and colonization of anaerobic gram-negative bacteria, particularly the orange and red complex groups.[4]

The orange complex bacteria include *Prevotella intermedia (P. intermedia)*, *Parvimonas micra*, and *Fusobacterium nucleatum (F. nucleatum)*, while the red complex consists of *Porphyromonas gingivalis (P. gingivalis)*, *Tannerella forsythia (T. forsythia)*, and *Treponema denticola (T. denticola)* species.[4] These bacteria are highly pathogenic and have the ability to secrete bacterial collagenases and other proteases. The release of these enzymes triggers a proinflammatory response in the host, causing damage to periodontal tissues.[5]

Etiological Factors Causing Periodontal Disease

The etiological factors causing periodontal disease are classified below:[6]

- Microbial dental plaque
- Local predisposing factors
- Systemic predisposing factors

Local predisposing factors:

- Dental calculus (Tartar)
- Materia alba
- Food debris
- Food impaction
- Dental anatomy
- Faulty restorations
- Malocclusion
- Mouth breathing
- Occlusal trauma

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Systemic predisposing factors:

- Hormonal imbalance
- Nutritional deficiency
- Blood disorders
- Genetic
- Immunological factors
- Metal intoxication
- Immune suppression
- Psychological factors

Periodontal Disease Pathogenesis

Recent studies have shown that bacterial plaque alone is not sufficient for the onset and progression of periodontal disease.[7] The most important factor in the destruction of hard and soft tissues in periodontal disease is the activation of the host immune-inflammatory response against bacterial plaque.[8] While gingivitis and mild periodontitis are common in the general population, advanced periodontitis is a rarer condition.[9]

Some individuals have been found to be more susceptible to periodontal disease, and this increased susceptibility has been linked to the host immune-inflammatory response that develops in periodontal tissues following the formation of bacterial plaque.[10] The microbial threat posed by subgingival plaque triggers the host immune-inflammatory response by triggering the excessive production of inflammatory cytokines [interleukins, tumor necrosis factor-α (TNF-α)], prostanoids (prostaglandin E_2), enzymes (matrix metalloproteinases-MMPs), and reactive oxygen species (ROS), thereby triggering the host immune-inflammatory response.[11] These inflammatory mediators play a significant role in the destruction of periodontal hard and soft tissues.[12]

Pathogenesis stages in periodontal disease:[6]

- Colonization
- Invasion
- Tissue destruction
- Healing and fibrosis

Colonization: Plaque accumulates on the tooth surface and is soon colonized by microorganisms. Plaque grows and develops through bacterial proliferation and the apical migration of motile bacteria.[6]

Invasion: Microorganisms and their products invade the depths of the connective tissue, even reaching the alveolar bone surface.[6]

Tissue destruction: Microorganisms and their products are observed when they penetrate tissues, either directly or through host-dependent effects.[6]

Healing and fibrosis: Antigens from plaque bacteria stimulate T and B lymphocytes, causing them to multiply. Thus, the humoral and cellular immune response begins.[6]

DISTRIBUTION OF PERIODONTAL DISEASE BY GENDER

Many studies examining the relationship between periodontal status and gender have indicated that the prevalence and severity of periodontal disease are higher in men.[13] This difference between genders has been attributed more to oral hygiene habits than to genetic factors.[14]

A study conducted at the Faculty of Dentistry, Van Yüzüncü Yıl University, found that 53.5% of the 1,758 individuals who visited the clinic were women (941) and 46.5% were men (817).

The results of this study show that men are at higher risk for periodontal disease.[13]

Similarly, a study conducted at Atatürk University indicated that men are more prone to periodontal diseases and that this is largely due to smoking, low oral hygiene awareness, and hormonal differences.[15]

There is strong evidence that men have a higher prevalence and severity of destructive periodontal disease than women with similar gum disease.[15]

One study found that gingivitis is more common in women than in men, while periodontitis is more common in men than in women.[16] This result may be related to higher smoking rates and poor oral hygiene (high PI, Gi, and SK values) among men.[17] Similarly, some studies emphasize that women may be more susceptible to periodontal diseases during certain periods due to hormonal changes (such as pregnancy and menopause).[18]

PREVALENCE OF PERIODONTAL DISEASE IN WOMEN

The prevalence of periodontal disease in women may be higher than in men due to factors such as hormonal changes, pregnancy, menopause, and birth control pill use. Hormonal fluctuations, especially during adolescence, pregnancy, and menopause, cause the gum tissue to become more sensitive to inflammation. Therefore, it has been stated that women's risk of developing periodontal disease increases significantly during these specific periods.[19]

A study conducted among patients attending the Necmettin Erbakan University Faculty of Dentistry found that the prevalence of periodontal disease was higher in women than in men. The study examined a total of 799 patients, including 510 women and 289 men, and found that 56% of women had signs of periodontal disease.[20]

Studies provide significant evidence that hormonal changes in women increase the risk of periodontal disease. The pregnancy period, in particular, is a time when the incidence of gum inflammation (gingivitis) increases. It has been emphasized that the decrease in estrogen levels after menopause increases the risk of bone loss and creates conditions conducive to the development of periodontal disease.[21]

EFFECTS OF HORMONAL CHANGES IN WOMEN ON PERIODONTAL TISSUES

The periodontium is a tissue complex consisting of the gingiva surrounding and supporting the tooth, the periodontal ligament, cementum, and alveolar bone. It is also responsible for the nutrition of the cells surrounding the tooth and the transmission of nerve impulses.[22] The homeostasis of the periodontium involves a multifactorial relationship in which the endocrine system plays an important role. Hormones play an important role in the human body, and women in particular undergo many hormonal changes throughout their lives. Biological changes such as puberty, menstruation,

pregnancy, menopause, and oral contraceptive use have suggested a possible link between sex steroid hormones and periodontal health.[23] Clinical observations have revealed that periodontal tissues may be target tissues for androgens, estrogens, and progestins due to the combination of hormone localization, receptors, and metabolism. Although the etiology of periodontal endocrinopathies varies, it has been suggested that periodontal pathologies may be related to the effect and interaction of steroid sex hormones on cells found in the periodontium.[24]

The currently accepted classification of periodontal disease acknowledges that endogenous steroid sex hormones have an effect on the periodontium.

These effects generally manifest as gingival symptoms.[25]

Studies suggest that changes in the periodontium may be related to fluctuations in hormones.[26]

Hormones

Sex steroid hormones: Estrogens, androgens, and progestins are known as sex steroid hormones. These hormones are lipid molecules with a common cyclopentanoperhydrophenanthrene skeleton. They are generally derived from 27-carbon cholesterol through sequential removal of carbon atoms and hydroxylation reactions, and are secreted by the adrenal cortex and gonadal cells. Steroid synthesis is the process by which the cholesterol molecule is irreversibly converted through sequential reactions into molecules belonging to the pregnane, androstan, and estrone families. Sex steroid hormones are synthesized in specific tissues and released directly into the bloodstream to reach their site of action. The tissues and cells they affect are called "target tissues"; the gingiva is one such target tissue. In target tissues, hormones can exert effects such as regulating the rate of metabolic pathways, stimulating or suppressing the synthesis and release of other hormones, and altering the production of non-hormonal compounds.[23]

Androgens: All natural androgens are based on a 19-carbon tetracyclic hydrocarbon skeleton known as androstan.[23] Androgens exert a number of important effects in the body; these include roles in spermatogenesis and the development of secondary sexual characteristics in male puberty.[27] Androgens are essentially found in two main types: gonadal androgens, which include dihydrotestosterone (DHT), and adrenal androgens, known as dehydroepiandrosterone (DHEA). Adrenal androgens can be converted into hormones such as testosterone and estrogen in the circulation. This conversion is the primary source of estrogen in men and postmenopausal women. Testosterone, one of the most potent androgenic hormones, is synthesized in the Leydig cells of the testes, the theca cells of the ovaries, and the adrenal cortex.[28]

The gingiva is one of the target tissues for androgen hormones. The presence of androgen receptors has been detected in fibroblasts found in periodontal and gingival tissues.

In the presence of testosterone, increased matrix synthesis has been observed in periodontal cells, and it has been reported that the number of testosterone receptors in fibroblasts increases in inflamed and hypertrophic gingiva.[29]

In their studies, Kasasa and Soory[30] reported that androgen metabolism increased in response to interleukin-1 (IL-1) in chronically inflamed gingival and periodontal ligament tissues. They also noted that DHT concentrations stimulated by insulin-like growth factor increased.

Parkar and colleagues[31] demonstrated that increased DHT concentrations gradually reduced interleukin-6 (IL-6) production in gingival cells isolated from healthy individuals and gingivitis patients.

Similarly, Gornstein and colleagues[32] detected androgen receptors in both gingival and periodontal ligament fibroblasts and observed that androgens reduced IL-6 production in cells possessing these receptors.

IL-6 is a cytokine that plays an important role in tissue destruction during periodontal disease and is secreted by many cells, including oral fibroblasts. These findings suggest that testosterone may have anti-inflammatory effects on the periodontium and that androgens may protect the periodontium through their positive anabolic effect on periodontal cells, their negative effect on the production and presence of inflammation markers, and their ability to inhibit osteoclastic activity.[32]

Effects of androgens on periodontal tissues:

- Suppression of prostaglandin secretion
- Proliferation and differentiation of osteoblasts
- Reduction in IL-6 production during inflammation
- Increased matrix synthesis in periodontal ligament fibroblasts and osteoblasts.[32]

Estrogen and progesterone: Estrogen and progesterone are the primary hormones that cause physiological changes in women throughout various stages of life, beginning at puberty. Estrogen initiates pubertal development in women, while androgens, together with estrogen, regulate the menstrual cycle and suppress follicle-stimulating hormone release from the anterior lobe of the pituitary gland.[33] Estrogen stimulates the secretion of watery mucus in the cervix by imparting acidophilic properties to cells. This process reduces the viscosity of the mucus while increasing its elasticity. In addition, estrogens increase the synthesis of coagulation factors associated with vitamin K, reducing the production of antithrombin III, which increases the tendency of blood to clot. Estrogen also affects water and salt retention in the kidneys. Estradiol is the most potent estrogen during the premenopausal period and is synthesized in peripheral tissues such as the ovaries, testes, and placenta.[33]

Biological functions of estrogen:

- Development of secondary sex characteristics
- Uterine development
- Luteinizing hormone secretion from the anterior pituitary gland
- Peripheral and axial skeletal development[33]

Clinical observations have revealed that individuals with low estrogen levels experience more gum inflammation compared to those with normal levels.[34] This suggests that estrogen may modulate inflammatory mediators by affecting prostaglandin production. Furthermore, circulating estrogen levels are thought to play a critical role in maintaining periodontal health.

In their studies investigating the relationship between periodontal disease and hormones, Plancak and colleagues[35] found that estradiol levels were lower in patients with advanced periodontitis compared to healthy individuals.

Effects of estrogens on periodontal tissues:

- Absence of gingival inflammation despite increased plaque amount[34]
- Decreased keratinization while increasing epithelial glycogen[36]
- Increased cellular proliferation in blood vessels[37]
- Stimulation of PMNL phagocytosis activity[38]
- Inhibition of PMNL chemotaxis[39]
- Suppression of leukocyte production from bone marrow[40]
- Inhibition of proinflammatory cytokines[41]
- Suppression of T-cell-mediated inflammation[40]
- Proliferation of gingival fibroblasts[42]
- Stimulation of gingival connective tissue synthesis and maturation[42]

The biological activity of progesterone is primarily observed during the luteal phase of menstruation and during pregnancy.

Progesterone:

- Is necessary for the continuation of pregnancy
- Reduces the effect of insulin
- Stimulates the hypothalamic respiratory center
- Increases body temperature during ovulation
- Increases sodium excretion by the kidneys.[23]

Effects of progesterone on periodontal tissues:

- Increases vascular dilation and permeability[28]
- Weakens the anti-inflammatory effects of glucocorticoids[43]
- Increases prostaglandin production[44]
- Increases PMNL and prostaglandin E_2 levels in gingival crevicular fluid[44]
- Inhibition of collagen synthesis in periodontal ligament fibroblasts[27]
- Suppression of gingival fibroblast proliferation[26]
- Decreased repair potential due to altered collagen production[45]
- Increased metabolic breakdown of folate, which is necessary for tissue repair[45]

The second phase is the luteal phase. During this phase:

- The developing corpus luteum synthesizes estradiol and progesterone.
- Estrogen rises to 0.2 ng/ml, progesterone to 10 ng/ml. This creates a suitable endometrium for a fertilized egg.
- The corpus luteum ruptures.
- Ovarian hormone levels decrease.
- Menstruation occurs.

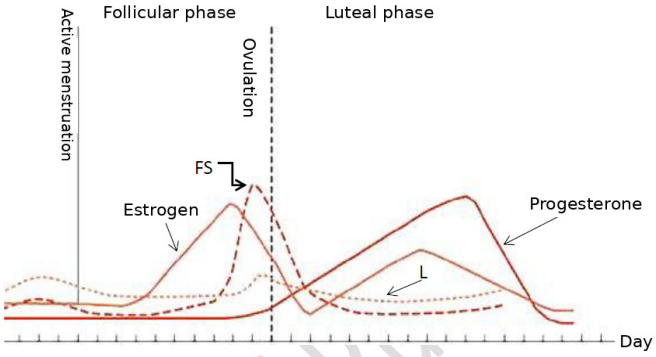

Figure 1. A graph showing hormone levels in women throughout the menstrual cycle.[46]

Various scientific data indicate that ovarian hormones increase inflammation in gingival tissues and intensify the response to local irritants. Increases or imbalances in sex hormones may cause gingival inflammation to worsen. In addition, these hormones may have indirect effects on periodontal tissues by modulating the functions of immune system cells.[46]

Progesterone alters collagen synthesis and structure in gum tissues, increases vascular permeability and folate metabolism, and regulates the immune response. Throughout the menstrual cycle, progesterone levels rise starting in the second week, peak approximately ten days later, and show a marked decline just before menstruation. This hormone promotes the production of prostaglandins, which regulate the body's inflammatory response. PGE_2, in particular, is one of the main mediators released by monocytes and found at higher levels in inflamed gums. Progesterone enhances the chemotaxis of polymorphonuclear cells, while estradiol suppresses this process. On the other hand, testosterone has been reported to have no significant effect on the chemotaxis of polymorphonuclear leukocytes. Physiological, experimental, and clinical studies have revealed significant differences in immune response between women and men.[46]

During menstruation, gingival tissues may become more edematous. In some individuals, erythema observed in the gums prior to menstruation may be a sign of an approaching cycle. In addition, an increase in gingival exudate and, consequently, noticeable changes in tooth mobility may be observed. At the onset of menstruation, osteitis cases that may develop after tooth extraction have been reported to be observed more frequently. Although a slight increase in platelet count and prolonged clotting time is observed during this process, no significant hematological changes have been detected in blood values.[46]

The peak levels of progesterone during the luteal phase may increase the incidence of recurrent aphthous ulcers, herpes labialis, and candidal infections in some women. Furthermore, progesterone's effect on relaxing the esophageal sphincter may increase susceptibility to gastroesophageal reflux disease (GERD). GERD can manifest with symptoms such as heartburn, vomiting, and chest pain, and in advanced cases, it may be associated with conditions such as coughing, hoarseness, sore throat, gingivitis, and asthma.[46]

Approximately 7-10 days before menstruation, progesterone levels reach their peak, which may cause premenstrual syndrome (PMS). It has been reported that levels of certain neurotransmitters such as enkephalin, endorphin, gamma-aminobutyric acid (GABA), and serotonin are lower in

women experiencing PMS. This can lead to symptoms such as depression, emotional sensitivity, sudden mood swings, difficulty concentrating, and memory impairment. In addition, it has been observed that individuals are more sensitive to treatments during this period, nausea reflexes increase, and the pain threshold decreases. PMS occurs in 70% of menstruating women, but only 5% are clearly diagnosed.[46]

Due to gum bleeding and increased sensitivity associated with the menstrual cycle, periodontal examination should be performed with care. Although periodontal control intervals vary according to individual needs, follow-up every 3-4 months is recommended in problematic cases. Antimicrobial mouthwashes may be an option in cases of cycle-related inflammation; however, unnecessary interventions should be avoided by first evaluating the individual's oral hygiene habits.[46]

Surgical procedures should be postponed until after the menstrual cycle, if possible, in patients with a history of excessive bleeding during menstruation or who are at high risk of bleeding after surgery. Considering the risk of anemia in such patients, it is recommended that the necessary blood tests be performed and consultation with relevant specialists be provided before surgical intervention.[46]

During PMS, many women experience:

- Fatigue
- Increased cravings for sweet and salty foods
- Bloating in the abdominal area
- Swelling in the hands and feet
- Headaches
- Breast tenderness
- Nausea or feeling nauseous
- Gastrointestinal discomfort and other physiological symptoms.[46]

PREGNANCY

During pregnancy, significant physiological changes occur in both the mother and the baby. During this process, changes in the mother's immune system can increase susceptibility to infections, and periodontal infections are among these conditions.[47] Although there is no definitive evidence, it has been suggested that periodontal infections may be associated with adverse pregnancy outcomes such as preterm birth, low birth weight, gestational hypertension, preeclampsia, gestational diabetes, and miscarriage (abortus).[48]

Effects of Pregnancy on Periodontal Tissues

Hormonal imbalances and decreased immune response during pregnancy can increase the response of periodontal tissues to irritation, affecting the clinical and biological characteristics of infections.[49] However, hormonal changes alone are not expected to cause gingivitis; bacterial plaque and gum inflammation are also necessary for this.[50] In many cultures, there is a widespread belief that every pregnancy causes tooth loss in the mother. Although epidemiological studies on the relationship between pregnancy and tooth loss have yielded different results, there is a general consensus that the prevalence and severity of gingivitis increase during pregnancy.[51]

Changes in estrogen and progesterone levels during pregnancy cause changes in the composition of the subgingival microflora. During this period, the ratio of anaerobic microorganisms to aerobic microorganisms increases. Some periodontal pathogens, such as *P. intermedia*, *Bacteroides* species, and *Campylobacter rectus* (*C. rectus*), have been found to be present at higher levels during pregnancy. This increase in pathogens affects the interaction between the periodontal microflora and the host, creating a higher susceptibility to periodontal tissue damage.[49]

Machado and colleagues[52] reported that oral bacteria such as *F. nucleatum*, *P. gingivalis*, *A.a.*, *T. denticola*, *C. rectus*, and *T. forsythia* can cross into the pregnant uterus regardless of the presence of clinical periodontitis, leading to local inflammation and adverse pregnancy outcomes. They emphasized that high levels of these pathogens in pregnant individuals may increase the risk of adverse pregnancy outcomes and that early diagnosis and additional care are important for controlling these microorganisms.

Although the exact mechanism of the increase in gingival inflammation observed during pregnancy is not fully understood, changes in neutrophil function, modifications in the cellular and humoral immune systems, hormone-induced changes in cellular physiology, and local effects on the microbial flora are thought to play an important role in this process.[49]

Pregnancy gingivitis: Pregnancy gingivitis is a condition that can be seen in 30 to 100 percent of pregnancies and was first described by Pinard in 1877.[53] It is characterized by redness, swelling, gum enlargement, and increased bleeding tendency. Although histologically similar to classic gingivitis, its etiological factors differ.[54] Periodontal health status prior to pregnancy may be a determining factor in the rate and severity of the disease. While the anterior regions and interproximal areas are more frequently affected, increased swelling can lead to increased pocket depth and tooth mobility. The gums are the area most affected by this process, followed by the tongue, cheek mucosa, and palate, which may also show changes.[53]

Pregnancy tumor: Pyogenic granuloma, also known as pregnancy tumor, pregnancy epulis, or granuloma gravidarum, occurs in 0.2% to 9.6% of pregnancies, usually during the second and third months of pregnancy.[53] This condition develops as a result of hormonal changes that occur during pregnancy. The effect of estrogen on macrophages, leading to an increase in vascular endothelial growth factor, local irritants, and bacterial factors cause this vascular lesion to grow.[55] Histologically, it is a vascular lesion resembling granulation tissue and exhibiting high proliferative properties.[56]

Pyogenic granulomas seen during pregnancy usually shrink and disappear spontaneously after birth as hormonal balance returns to normal. In this respect, they differ from other pyogenic granulomas.[57]

The lesion may be flat or lobulated, with an exophytic structure; it may be erythematous, pedunculated, or sessile. Although it is mostly localized in the gingiva, it can also develop on the tongue, palate, and buccal mucosa.[58] It has a fragile surface prone to bleeding and may be pink, red, or purple in color. This color variation may differ depending on the age of the lesion, the level of vascularization, and venous circulation.[58] Bone loss is not usually observed in pregnancy-related pyogenic granuloma.[53]

If the lesion is not bleeding, does not affect chewing function, and gradually shrinks after birth, only regular follow-up is recommended instead of surgical intervention.[55] However, pyogenic granulomas removed surgically may recur due to incomplete surgery or poor oral hygiene.[57]

Clinical and microbial changes in periodontal tissues during pregnancy:

- Increased gingival probing depths
- Increased gingival inflammation
- Increased gingival crevicular fluid
- Increased bleeding on probing
- Increased tooth mobility
- Increased incidence of pyogenic granulomas
- Increased number of periodontal pathogens, particularly *P. gingivalis* and *P. intermedia*.[59]

Effects of Periodontal Disease on Pregnancy

Numerous epidemiological studies examining the relationship between periodontal infections and adverse pregnancy outcomes have demonstrated a statistically significant association between the two conditions.[60] However, some studies have reached opposite conclusions, suggesting that periodontal disease has no significant effect on pregnancy outcomes. Although the exact cause of these conflicting findings is unclear, genetic and environmental factors that vary across populations are thought to be one of the key determinants of susceptibility to adverse pregnancy outcomes. Adverse pregnancy outcomes include preterm birth, very preterm birth, low birth weight, very low birth weight, miscarriage, and preeclampsia.[61]

Preterm birth is defined as birth occurring before the 37th week of pregnancy and is seen in approximately 10-15% of pregnancies.[62] According to World Health Organization (WHO) data, the preterm birth rate was reported as 9.6% between 1997 and 2007.

Births occurring before the 32nd week of pregnancy are referred to as very preterm births.[63] Low birth weight was defined by the WHO in 1976 as a birth weight of less than 2500 g. A weight of less than 1500 g is referred to as "very low birth weight".[64]

Infection is reported to be one of the main factors in 30-50% of cases of preterm birth and low birth weight. Infections occurring in the cervical region of the uterus, in particular, significantly increase these risks.[49] Periodontal disease is an infectious condition caused by gram-negative anaerobic bacteria.[61]

Colins et al.[65] reported that periodontal infections may lead to low birth weight by triggering cellular immune mechanisms that produce cytokines such as interleukin-1 beta (IL-1β), TNF-α, and prostaglandin E (PGE_2) through bacterial activation.

The relationship between periodontal disease, preterm birth, and low birth weight can be explained by three mechanisms:

- Dissemination of inflammatory products into the bloodstream
- The immune response of the mother/fetus to oral pathogens
- Dissemination of oral bacteria through the bloodstream.[66]

In some cases, it has been observed that amniotic fluid infected by oral microorganisms can trigger premature birth. In this context, it is noted that bacteria such as *Streptococcus* species, *E. corrodens*, *F. nucleatum*, and *P. gingivalis* may play a role.[49]

Clinical approach to the pregnant patient: An important component of periodontal examination is taking a detailed medical history.

Immunological changes that occur during pregnancy can cause an increase in blood pressure. In addition, considering the physiological interactions between the fetus and the mother, the physician must carefully evaluate the patient.[46]

The medical history should focus particularly on:

- Pregnancy-related complications
- Previous miscarriages
- Cramps, vomiting
- Vaginal bleeding in the form of spotting or staining.[46]

To accurately determine the patient's periodontal and dental treatment needs, communication with the obstetrician is essential, and information about the patient's general health status should be obtained.

It should be explained in detail that pregnant individuals are more prone to gingival inflammation, effective oral hygiene techniques should be taught, and they should be encouraged to seek regular care.

During pregnancy, tartar removal, polishing, and root planing procedures can be performed when necessary. When mouthwash use is recommended, alcohol-free formulations should be preferred.[46]

Elective dental treatments should be limited to maintaining good oral hygiene and should be postponed as much as possible during the first and second half of the third trimester of pregnancy.

The first trimester is a period when the fetus is extremely sensitive to environmental factors due to the organogenesis process. In the latter half of the third trimester, the uterus becomes sensitive to external stimuli and the risk of premature birth may increase. The patient should not be seated for long periods of time because supine hypotensive syndrome may occur. The pressure exerted by the pregnant uterus on the inferior vena cava and pelvic veins causes a slowdown in venous return and a decrease in cardiac output, leading to hypotension. Women lying supine may experience dizziness, paleness, tachycardia, sweating, and nausea. In a semi-recumbent or supine position, the inferior vena cava is compressed by the weight of the uterus.

In supine hypotensive syndrome, turning the patient onto her left side is usually sufficient (**Figure 2**). This relieves pressure on the inferior vena cava, allowing blood to return from the pelvic area and lower extremities. A soft cushion approximately 15 cm thick (a rolled towel may be used) should be placed under the patient's right side when she is reclined for treatment.[46]

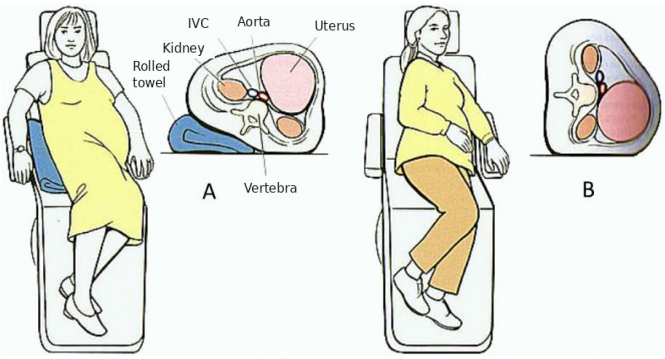

Figure 2. Patient position applied to prevent supine hypotensive syndrome[46]

The second trimester is considered the most appropriate time for routine dental treatment in pregnant patients. During this period, the goal is to control existing diseases and prevent potential problems that may arise in the later stages of pregnancy.

Major oral and periodontal surgeries should be postponed until after delivery. However, in cases of pregnancy tumors causing pain, difficulty chewing, or suppuration with bleeding, a biopsy or excision of the lesion may be necessary before delivery if deemed necessary.[46]

THE EFFECT OF ORAL CONTRACEPTIVE USE ON PERIODONTAL TISSUES

Steroid-containing medications taken orally by women to prevent pregnancy are called oral contraceptives. These medications contain estrogen and progestin hormones and prevent pregnancy by temporarily suppressing fertility.[67] Combination oral contraceptives used for contraception are usually taken daily as a single dose from the 5th day of the cycle until the 25th day, with a total of 21 pills used per cycle. These pills contain a combination of estrogen and progestin. Considered one of the most effective methods of preventing pregnancy, these drugs should ideally contain the lowest amount of hormones necessary to prevent pregnancy.[68]

Hormonal changes can increase susceptibility to gingivitis by affecting the host's biological environment.[69] For example, during pregnancy, a higher-than-expected increase in plaque-related inflammation may be observed compared to the level of plaque accumulation.[70] In addition, it has been reported that the immune response to subgingival microbiota and bacterial antigens changes during pregnancy.[71]

Along with the rise in hormone levels, there is also an increase in the proportion of bacteria such as *P. intermedia*, which can use estrogen and progesterone instead of vitamin K. During this process, an increase in clinical susceptibility to gingivitis is observed, while these changes generally return to normal after delivery.[72]

Clinical studies have shown that women using hormonal contraceptive drugs experience more inflammatory reactions than those who do not use them. However, it has not been definitively determined whether these drugs directly affect attachment levels. Gingivitis associated with oral contraceptive use shares similar characteristics with plaque-induced gingivitis and gingivitis observed during pregnancy. A significant increase in gingival crevicular fluid may be observed during this process. Additionally, it has been reported that the severity of gingivitis is related to the dose of the medication, with inflammatory reactions being milder at lower doses. In some cases, it may be necessary to change or discontinue the medication based on the severity of gum symptoms.

Clinical studies have shown that the use of oral contraceptive agents may increase gum changes during the premenstrual period but does not cause significant tissue growth.[74] Furthermore, it has been reported that reducing the dose of contraceptive drugs or discontinuing their use alleviates or prevents gum discomfort.[73] Each of these mechanisms contributes to a stronger inflammatory response in the gums to local irritants. Bacterial plaque accumulating at the gum line causes mild inflammation in individuals who do not use oral contraceptives, while the artificial increase in systemic progesterone levels in individuals who use oral contraceptives may cause this inflammatory response to become more pronounced.[74]

Changes observed in the periodontium with oral contraceptive use:

- Increased inflammation against local irritants
- Increased plaque levels, pocket formation, bleeding, and attachment loss
- Increased numbers of *Candida*, *P. gingivalis*, *P. intermedia*, and *A.a* species in periodontal pockets.[74]

Consequently, the use of oral contraceptives containing estrogen and progesterone may cause an increase in the incidence of gingivitis by inducing effects similar to the hormonal changes seen during pregnancy. Furthermore, long-term use of hormonal contraceptives has been reported to cause progression of attachment loss in periodontal tissues.[74]

MENOPAUSE

The WHO defines menopause as "the permanent cessation of menstruation following the end of ovarian function".[75] Clinically, menopause is confirmed 12 months after the last menstrual cycle, while the menopausal transition period generally lasts 4 to 7 years.[76] Global data shows the average age of menopause to be 51.[77] It has been reported that this range varies between 49.3 and 51.4 years in developed countries and between 43.5 and 49.4 years in developing countries.[78] According to a study conducted in Turkiye, the average age of menopause onset was 35, while the highest average age was recorded as 55. According to data from the 2013 Turkish Population and Health Survey, 49% of women aged 48-49 had entered menopause.[79]

Katz and Epstein[80] suggested that androgens, together with estrogens, may play a fundamental role in maintaining bone health. They attributed this to the inhibitory effect of estrogens on osteoclastic activity. In the postmenopausal period, symptoms such as increased risk of osteoporotic fractures, myocardial infarction, menstrual irregularities, hot flashes, and night sweats may be observed.

Menopause not only causes systemic changes but also affects oral tissues. Changes in the mouth generally occur as a result of the aging process and decreased estrogen levels. Decreased estrogen levels cause reduced epithelial keratinization and decreased salivary flow rate.[81] During this period, some women may experience symptoms such as menopausal gingivostomatitis, dry mouth, pale or red gums, and bleeding during probing and brushing.[82]

Various changes in oral health may occur in women during menopause. Burning sensation in the mouth, dryness, and impaired taste are among the common complaints during this period. A burning sensation felt in the normal-looking oral mucosa is called "burning mouth syndrome" and is common in postmenopausal women. The symptoms of this syndrome can range from mild discomfort to severe pain. Some oral diseases, such as lichen planus, candidiasis, and viral infections, can cause similar symptoms, but no significant pathological changes are observed on the mucosal surface in burning mouth syndrome.[83]

Wardrop et al.[84] investigated the relationship between menopause and oral disorders in a study of 149 women. According to the study results, the prevalence of oral disorders was 43% in perimenopausal and postmenopausal women, while this rate was found to be 6% in premenopausal women.

One of the most significant health problems encountered during menopause is osteoporosis. Osteoporosis is characterized by a decrease in bone density, which leads to an increased risk of fractures. Both osteoporosis and periodontal diseases are among the major health problems in older individuals. Studies have suggested that ovarian dysfunction and increased incidence of periodontal disease may be associated with a decrease in mandibular bone density. Osteoporosis leads to a decrease in alveolar bone volume in the crest region, and this bone loss becomes even more pronounced in the presence of periodontal disease.[85]

The fact that a large proportion of patients with desquamative lesions are middle-aged and predominantly female (80%), along with the higher prevalence of diseases such as benign mucous membrane pemphigoid and lichen planus in women, suggests that sex steroid hormones may be associated with certain desquamative lesions. It has been reported that such lesions can be successfully managed with exogenous estrogen therapy.[23]

Clinical and Microbiological Changes in the Periodontium During Menopause

In recent years, hormone replacement therapy (HRT) has been recognized as an effective method for managing symptoms associated with menopause. This treatment plays an important role in preventing postmenopausal osteoporosis by reducing bone mass loss. Furthermore, it has been suggested that HRT may contribute to the preservation of periodontal health in postmenopausal women.[86]

Clinical symptoms associated with menopause:

- Decreased epithelial keratinization
- Reduced salivary flow, burning sensation in the mouth, dry mouth, and bad taste
- Increased bleeding during probing and brushing
- Increased alveolar bone loss due to increased osteoporosis
- Increased incidence of desquamative lesions has been reported.[86]

HRT may contribute to the preservation of periodontal health during the postmenopausal period.[87] It has been suggested that estrogen supplementation may regulate periodontal tissue destruction by helping to reduce matrix metalloproteinases such as MMP-8 and MMP-9, as well as cytokines involved in bone resorption.[88] Some clinical studies have shown that HRT has positive effects on tooth preservation and alveolar bone density.[89] In addition, it has been noted that the incidence of gingival bleeding is lower in women using HRT.[90]

CONCLUSION

This review aims to examine the effects of hormonal changes observed in women on periodontal tissues. Numerous studies have shown that hormonal fluctuations during adolescence, the menstrual cycle, pregnancy, oral contraceptive use, and menopause affect the course of periodontal disease by increasing gingival inflammation. The literature has concluded that there is an increase in the frequency of conditions such as gingivitis, periodontitis, and pyogenic granuloma during hormonal periods. Furthermore, it has been emphasized that periodontal diseases during pregnancy may be associated with adverse pregnancy outcomes such as preterm birth and low birth weight. It is necessary to consider hormonal changes and raise awareness in this regard for the preservation of periodontal health and management of treatment processes in female patients. These findings are expected to contribute to the development of more effective and individualized treatment strategies for female patients in clinical practice.

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Review

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The relationship between stress distribution and adhesive failure in non-carious cervical lesions (NCCL): a review

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ABSTRACT

Non-carious cervical lesions (NCCLs) are lesions observed in dental hard tissues and have a multifactorial etiology. It is believed that surface loss processes such as abrasion and erosion, together with the biomechanical effects caused by occlusal stress, play a role in the development of these lesions. The structural characteristics of the cervical region and the distribution of stress within this area influence both lesion formation and the success of restorative treatments. The aim of this review is to evaluate the relationship between the etiology of NCCLs, biomechanical effects, and mechanisms of adhesive failure and to outline clinical approaches and restorative strategies accordingly. Current findings suggest that stress accumulation in the cervical region may adversely affect the adhesive interface, leading to loss of retention and marginal breakdown. Consequently, to achieve successful outcomes in the treatment of NCCLs, it is necessary to evaluate biomechanical factors and clinical variables together and to adopt a personalized treatment approach.

Keywords: Non-carious cervical lesions, NCCL, biomechanical stress, adhesive failure, occlusal loading, restorative treatment

INTRODUCTION

Non-carious cervical lesions (NCCL) are lesions frequently encountered in clinical practice and are recognized as a significant oral health problem. These lesions are characterized by non-carious hard tissue loss in the cervical region of the tooth, at the level of the cementoenamel junction (CEJ).[1] With increasing life expectancy, individuals are able to preserve their natural teeth for longer, which has led to NCCLs becoming a more frequently observed finding during clinical examinations.[2] These lesions not only result in permanent tissue loss but can also lead to clinical problems such as dentine hypersensitivity, changes in pulp vitality, and increased plaque retention. Clinically, NCCLs can present with various morphological appearances, including superficial, concave, notched, or wedge-shaped lesions.[3]

NCCLs are defined as multifactorial lesions in which surface loss processes such as abrasion and erosion interact with the biomechanical effects caused by occlusal stress.[4] Furthermore, factors such as poor oral hygiene habits, parafunctional activities, low buffering capacity of saliva, and an acidic pH in the oral environment may also contribute to the formation of these lesions. The reduction in enamel thickness in the cervical region and the relatively weak enamel-dentin junction render this area more susceptible to stress and wear factors.[5] However, there is still no consensus in the literature regarding which material is more suitable for NCCL restorations and when restorative treatment should be initiated. In particular, the effect of occlusal stress distribution on the adhesive interface and its relationship with restoration failure has not been sufficiently elucidated. Therefore, the aim of this review is to evaluate the effect of stress distribution on adhesive failure in NCCL restorations and to discuss clinical approaches in line with the current literature.

THE ETIOLOGY AND BIOMECHANICAL BASIS OF NCCL

The accurate identification and differentiation of NCCLs is of great importance for treatment planning. A careful clinical assessment is required, particularly in early-stage lesions, to distinguish between different etiological factors.[4]

Attrition refers to physical wear observed as a result of contact between tooth surfaces during normal masticatory (functional) or habit-related (parafunctional) movements. This condition is generally observed on occlusal and approximal surfaces and may lead to an enlargement of the interproximal contact areas over time. In advanced cases, changes in occlusal relationships may also occur.[4]

Abrasion is defined as wear in dental hard tissues resulting from external mechanical forces.[6] Incorrect brushing techniques, excessive pressure, the use of brushes with stiff bristles, and toothpastes with high abrasive properties play a significant role in this process. These lesions are typically sharp-edged and wedge-shaped.[7]

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Erosion is the dissolution of dental hard tissues resulting from a chemical process caused by acids, independent of bacterial influence. This process is accelerated particularly by the consumption of acidic foods and drinks, the reflux of stomach acid into the mouth (gastric reflux), and environments with low pH levels. Furthermore, it is suggested that the buffering capacity of the environment may be a more significant factor in the progression of erosion than the pH value alone.[8]

Abfraction, on the other hand, is associated with a concentration of stress in the cervical region of the tooth resulting from flexure caused by occlusal forces, particularly their oblique and lateral components. Lesions formed via this mechanism are typically wedge-shaped, with sharp internal angles, and tend to progress towards the CEJ.[9]

While it is known that the development of NCCL is multifactorial, it has been suggested in recent years that biomechanical factors also play a significant role. In particular, the distribution of stress on the tooth caused by occlusal forces can concentrate in the cervical region, potentially leading to tissue loss.[10] From a biomechanical perspective, occlusal forces, particularly those in the lateral direction, lead to stress concentration in the cervical region of the tooth. This accumulation of stress can cause micro-level flexures in the cervical region, leading to weakening of the enamel and dentine structure and, over time, tissue loss. The reduction in enamel thickness and the weaker enamel-dentine junction in the cervical region make this area more susceptible to the effects of stress.[11] However, it is thought that the effect of repetitive loads, rather than the one-off impact of occlusal forces, plays a significant role in the development of NCCL. The stress and flexion generated in the cervical region as a result of occlusal loads can lead to the formation of microcracks in the enamel and dentine structure; over time, this can create a fatigue-like effect, resulting in tissue loss.[12]

MECHANISMS OF ADHESIVE FAILURE IN NCCL

The number of studies evaluating the clinical efficacy of universal adhesive systems has increased recently. The clinical performance of these systems is generally assessed through randomized controlled trials, and the retention of the restorative material is considered a key indicator of long-term success. However, parameters such as marginal degradation and marginal discoloration are also important criteria in assessing adhesive success in NCCL restorations.[13]

The morphological and histological structure of the cervical region plays a critical role in adhesive failure. Thinning of the enamel layer and changes in the structure of the enamel prisms in this region can adversely affect the quality of the adhesive bond. Furthermore, the fact that dentine frequently exhibits sclerotic characteristics and that the tubules are obstructed by mineral deposits can hinder the penetration of adhesive systems into the dentine, thereby reducing bond strength.[14] In NCCL restorations, adhesive failure is predominantly associated with defects occurring at the bond interface. The inability of adhesive systems to provide sufficient infiltration into the dentine surface and the degradation of the hybrid layer over time can lead to a reduction in bond strength. Systematic reviews and meta-analyses incorporating randomized clinical trials evaluating the efficacy of adhesive strategies in NCCL restorations have shown no significant difference between self-etch and etch-and-rinse approaches in terms of retention, marginal adaptation, and staining. However, it is stated that restoration failures most commonly manifest as a loss of retention and that this is associated with the weakening of the adhesive interface over time.[15]

Due to the cervical region's proximity to the gingiva, controlling moisture is difficult, which increases the risk of contamination during adhesive application and may adversely affect the quality of the bond. These circumstances pave the way for clinical failures, particularly marginal detachment, microleakage, and loss of retention.[16]

The long-term success of NCCL restorations remains limited, and the mechanism of failure has not yet been fully elucidated. Restoration performance depends on factors such as the material's elastic properties, the durability of the adhesive interface, and wear resistance; it has been demonstrated that these properties degrade over time. Furthermore, the higher incidence of marginal defects in the cervical region is associated with the biomechanical and biological characteristics of this area. It has been reported that restoration failure is a process resulting from the combined influence of numerous factors, including occlusal stress, the location of the lesion, the presence of sclerotic dentine, and material properties.

THE RELATIONSHIP BETWEEN STRESS DISTRIBUTION AND ADHESIVE FAILURE

In NCCLs, stress distribution is concentrated in the cervical region, particularly depending on the direction and magnitude of occlusal forces. Elastic deformation of the teeth under oblique forces leads to an increase in tensile stresses in the cervical region. Finite element analyses show that these stresses accumulate most around the enamel-dentin junction and that this region is biomechanically more sensitive. Furthermore, the direction and magnitude of the load, as well as tooth morphology, directly influence the distribution of stress, potentially increasing stress concentration in specific areas.[17] The effect of stress distribution in NCCL restorations on adhesive failure is explained, in particular, by the changes in stress occurring at the bonding interface. It has been reported that stresses generated in the cervical region cause different deformations between the restoration and the tooth structure and that this mismatch leads to stress accumulation at the adhesive interface.[18,19] This accumulation of stress causes the adhesive layer to remain under constant load and may lead to a reduction in bond strength over time. In particular, the difference in behavior between dental tissue and restorative materials, which possess different elastic properties, can increase stress concentration at the adhesive interface, thereby adversely affecting bond integrity.[19] Long-term clinical follow-up studies have reported that NCCL progression is associated with occlusal stress and that lesion progression is more pronounced in teeth subjected to high occlusal loads. This suggests that stress may play a role not only in lesion formation but also in post-restoration adhesive failure.[20]

Furthermore, the fact that the stress generated in this region is not merely static but exhibits a repetitive nature during functional and parafunctional movements may lead to the weakening of the adhesive bond through a fatigue-like process. This study explains how interface disruptions, which

initially occur at a microscopic level, progress over time to result in clinically observable loss of retention and marginal defects.[4]

In NCCL restorations, adhesive failure is not only related to material or technical factors but also to the dynamic effects of stress on the adhesive interface. This situation sets the stage for the adhesive bond to weaken over time, leading to clinical loss of retention and marginal deterioration.[16]

CLINICAL APPROACHES AND RESTORATIVE STRATEGIES

In the management of NCCLs, non-restorative approaches should be considered before proceeding to restorative treatment. Particularly in cases where dentine hypersensitivity is the primary concern, the use of desensitizing agents, fluoride varnishes, and products containing potassium nitrate can be effective in controlling symptoms. Furthermore, controlling the etiological factors can help slow the progression of the lesion, thereby delaying the need for restorative intervention.[5]

The clinical approach to the treatment of NCCLs should not be reduced solely to material selection. A successful restorative strategy must be determined by evaluating the depth and morphology of the lesion, the presence of dentine hypersensitivity, aesthetic expectations, occlusal loading, parafunctional habits, and the gingival margin. In particular, the presence of sclerotic dentine in the cervical region, high marginal stress, and the tendency of the adhesive interface to degrade over time make NCCL restorations more difficult to predict.[16]

In terms of the choice of restorative materials, there is no clear superiority between resin composites and glass ionomer-based materials. While glass ionomer-based materials offer advantages, particularly in cases where moisture control is difficult and the root surface is more extensively involved, resin composites have a wider range of applications due to their aesthetic and mechanical properties.[21,22] However, marginal leakage issues with resin composites and the degradation of the adhesive over time are among the key factors limiting clinical success. Consequently, the choice of an adhesive system and application technique play a critical role in the long-term success of the restoration.[23]

A three-year randomized clinical trial compared different adhesive strategies (universal adhesive, glass ionomer, and EDTA pretreatment). While similar success rates were achieved across all groups, it was reported that marginal defects were observed more frequently in resin composite restorations during the early period.[24]

In a randomized clinical trial comparing flowable composite with nano-filled composite, it was reported that the restorations were applied over the NCCL and that both materials demonstrated similar clinical performance in the short term.[25] In a 3-year study comparing high-filler flowable composite with conventional composite, it was reported that retention rates were similar but that the flowable composite yielded better results in terms of marginal adaptation.[26]

Current evidence regarding adhesive strategies indicates that there is no significant difference in clinical success between self-etch and etch-and-rinse approaches. In NCCL restorations using universal adhesive systems, no significant superiority has been found between different protocols in terms of retention, marginal adaptation, and staining.[27-29] However, the limited presence of enamel in the cervical region, the absence of macro-mechanical retention, and the highly sclerotic nature of dentine are significant factors that complicate adhesive bonding. Furthermore, the heterogeneous structure of the bonding surface may adversely affect hybrid layer formation.[23,24] For this reason, it has been suggested that, in some cases, the application of phosphoric acid prior to self-etch adhesive systems may enhance restoration retention.[30] This situation indicates that, in the clinical decision-making process, product-based evaluation is more important than material classification.[23]

In NCCL restorations, lesion morphology is also an important clinical variable. While stress concentration and increased tension at the adhesive interface are more pronounced in deep, wedge-shaped lesions, a more homogeneous stress distribution may be observed in superficial and broad lesions. This indicates that the success of a restoration depends not only on the material but also on the shape of the lesion.[4]

In NCCL cases accompanied by parafunctional habits, a restorative approach alone may not be sufficient. Occlusal adjustments and the use of a night guard (occlusal splint) to reduce occlusal stress may help to control the stresses arising in the cervical region. It has been reported that this approach may indirectly enhance the long-term success of restorations.[17]

In cases of NCCL presenting with gingival recession, restorative treatment and periodontal surgery may need to be planned in conjunction. In such situations, the restoration stabilizes the root surface, while periodontal surgery supports aesthetic and functional improvement. It has been reported that the combined approach yields more successful results, particularly in the aesthetic zone.[31]

CONCLUSION

Non-carious cervical lesions are complex lesions with a multifactorial nature in terms of their formation mechanism and clinical behavior and cannot be explained by a single etiological factor. Success in the restorative treatment of these lesions depends not only on the material or adhesive system used but also on the combined assessment of stress distribution, dentine properties, lesion morphology, and patient-related factors. The current literature indicates that there is no single material or adhesive strategy that can be universally regarded as superior in NCCL restorations. Therefore, the clinical approach must be based on individualized treatment planning rather than standard protocols. Consequently, to achieve successful and long-term clinical outcomes in the management of NCCLs, the lesion must be assessed not only from a restorative perspective but also in conjunction with its biomechanical and biological environment, and the treatment plan must be formulated accordingly.

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Case Report

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Oral and dental rehabilitation under general anesthesia in a pediatric patient with Smith-Magenis syndrome: first case from Turkiye

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ABSTRACT

Smith-Magenis syndrome (SMS) is a rare, genetically-based syndrome affecting multiple systems. It is characterized by developmental delay, behavioral disorders, and characteristic facial features. While various descriptions of the craniofacial and oral findings of SMS exist in the literature, studies involving dental treatment requirements are quite limited. This study aims to present the oral and dental rehabilitation performed for the second time under general anesthesia in a 9-year-old pediatric patient diagnosed with SMS during the mixed dentition period, who presented to our clinic with pain complaints, and to discuss the challenges of dental management of this rare syndrome in light of the literature.

Keywords: Smith-Magenis syndrome, chromosome deletion (17p11.2), general anesthesia, oral and dental rehabilitation

INTRODUCTION

Smith-Magenis syndrome (SMS) is a complex neurodevelopmental disorder affecting multiple systems, occurring sporadically and with a frequency of approximately 1/15,000-25,000. It was first described in 1986 by Ann C. M. Smith and Ellen Magenis. This genetic disorder is mostly associated with a deletion on the short arm of chromosome 17 (17p11.2). The most common cause of SMS is the deletion of the RAI1 (Retinoic Acid Induced 1) gene.[1]

Behavioral problems such as self-harming behaviors, aggression, temper tantrums, and sleep disorders are common in individuals with this syndrome. Sleep disorders are prevalent and can exacerbate other clinical symptoms. Hearing loss may be detected in some individuals. Skeletal anomalies, particularly scoliosis and other bone development disorders, are also reported. Intellectual disability is generally mild to moderate. Delays in communication skills significantly affect social interaction and learning processes.[2-5]

The facial appearance of patients with SMS is often unique. The main craniofacial findings are brachycephaly, frontal bossing, hypertelorism, fissure midline hypoplasia, tent lip (upward retraction of the midline of the upper lip), and micrognathia, which can develop into relative prognathism in later years.[6,7]

Oral findings of the syndrome have been reported as weakness of the tongue muscles, inadequate lip closure, prominent jaw structure, and abnormal palate morphology. Cleft lip and palate are rarely seen. Dental findings reported in SMS include micrognathia, hypodontia, microdontia, taurodontism, delayed eruption, persistent primary teeth, ectopic eruption, enamel hypoplasia/hypomineralization, bruxism, malocclusion, open bite, crowding, posterior crossbite, maxillary hypoplasia, relative prognathism, macroglossia, mouth breathing, and drooling. Dental anomalies, particularly agenesis of the lower second premolar teeth, taurodontism, and root anomalies, are frequently reported. The incidence of caries and gingivitis increases with age.[4,5,8] Due to behavioral and cognitive difficulties, non-pharmacological behavioral guidance techniques are not feasible for dental treatment in this patient group. Alternative treatment approaches such as conscious sedation or general anesthesia are needed.[9-11]

This case report presents the comprehensive dental treatment process and clinical management of a 9-year-old pediatric patient diagnosed with SMS, performed under general anesthesia. While the dental management of a limited number of pediatric cases with this syndrome has been reported worldwide,[3,12] this presentation is the first case reported from Turkiye.

CASE

A 9-year-old child with special needs presented to the Department of Pediatric Dentistry at Dicle University Faculty of Dentistry in Diyarbakır in 2025 due to aesthetic and pain problems caused by decayed teeth. The patient's medical history revealed a diagnosis of SMS. Necessary informed consent was obtained from the patient's parents.

Medical Anamnesis

The patient was born via cesarean section in 2016, with a recorded birth weight of 3.5 kg. Family history revealed consanguinity between the parents, the patient having one

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sibling, and no other family members diagnosed with SMS. The mother's medical history did not reveal any known teratogenic factors such as smoking during pregnancy or X-Ray exposure. Clinical examination revealed postural abnormalities, overweight, facial asymmetry, and prognathism. The patient's height (128 cm) and weight (35 kg) were determined to be developmentally advanced compared to peers. It was learned that the patient regularly used neurological medications. Based on the anamnesis obtained from the parents, it was reported that the characteristic findings of SMS included prominent self-harming behaviors, severe temper tantrums, and sleep disturbances. It was stated that the patient was able to self-harm to the point of causing bleeding by banging his head against the wall, and that these behaviors did not stop without family intervention. It was also noted that he exhibited repetitive self-harming behaviors such as throwing objects and biting his hand. It was learned that the patient regularly used Arislow ER oral tablets containing guanfacine hydrochloride and Neurodol oral drops containing haloperidol to control his temper tantrums. It was learned that 1 mg/ml Risperidone syrup (0.3 ml before bedtime) and 3 mg/5 ml Melatonin syrup (5 ml before bedtime) were prescribed to regulate sleep patterns, but due to insufficient clinical response after a certain period despite long-term and regular use, the treatment was discontinued by the parents 6 months ago. Regarding the feeding history, it was reported that the patient's eating behavior was uncontrolled, limited to the amount given by the family, and that they could not spontaneously terminate the feeling of fullness. No hearing loss was detected, and it was reported that intellectual development was behind that of their peers. The patient's history revealed that renal and cardiac anomalies and scoliosis, which are frequently reported in SMS, were not present.

Dental Anamnesis

The patient's history revealed that dental treatments under general anesthesia were performed at an external center 3 years prior. However, due to continued inadequate oral hygiene, secondary caries developed in the existing restorations, along with the formation of new carious lesions. Extraoral examination revealed brachycephaly, short stature with central obesity, midfacial hypoplasia, flat nasal bone structure, and mandibular prognathism. Occlusion assessment showed class III malocclusion according to Angle classification, along with antero-posterior crossbite. Macroglossia was also observed. Facial symmetry was preserved, and the facial profile was assessed as convex. The upper lip appeared full, and no cervical lymphadenopathy was found (**Figure 1a, b**). The clinical findings to be considered before dental treatment in this very rare patient are presented in the table (**Table**).

Figure 1. a) Extraoral view of the patient, **b)** Extraoral view of the patient

Teeth were identified using the FDI two-digit tooth numbering system (ISO 3950).[13] Intraoral examination revealed caries and mobility in teeth 52 and 62. Mobility was also detected in tooth 82. An enamel fracture was observed in tooth 31, and excessive substance loss due to old restorations and secondary caries was detected in teeth 74 and 75. Deep caries were observed in teeth 16, 26, and 73, while secondary caries beneath existing restorations were detected in teeth 36, 46, and 85 (**Figure 2a-d**).

In addition to the clinical diagnosis of caries, panoramic and periapical radiographic evaluation revealed the absence of the permanent tooth germ of tooth 45 (**Figure 3, 4**).

Table. Specific findings observed in our patient's clinic

	Clinical findings	Specific features
Craniofacial (face)	Prominent facial phenotype	Square facial structure, broad forehead, flat nasal bone structure, "tent mouth" (upward curved upper lip), brachycephaly, midfacial hypoplasia
Behavioral	Severe maladaptive behaviors	Self-harm, sudden temper tantrums, impulsivity, throwing objects and biting one's hand, head banging against walls, uncontrolled eating, inability to spontaneously terminate satiety
Neurological/cognitive	Developmental delay	Mild to moderate intellectual disability, delayed speech and language development
Sleep pathology	Reverse melatonin cycle	Excessive daytime sleepiness, frequent nighttime awakenings and hyperactivity, irregular sleep patterns
Characteristic movements	Stereotypical behaviors	The "self-hug" gestures
Dental/oral	Orodental anomalies	Class III malocclusion, anterior-posterior crossbite, mandibular prognathism, macroglossia, hypodontia (missing teeth), bruxism (teeth grinding), and a tendency towards poor oral hygiene

Figure 2. a) Intraoral view of the patient before treatment (maxilla), **b)** Pre-treatment intraoral view of the patient (right lower mandibular quadrant), **c)** Pre-treatment intraoral view of the patient (left lower mandibular quadrant), **d)** Intraoral view of the patient before treatment

Figure 3. Panoramic X-Ray of the patient before treatment

Figure 4. Pre-treatment periapical X-Ray of tooth number 85

Treatments were planned under general anesthesia due to the patient's lack of communication skills. Necessary consent forms were obtained from the patient's parents. Preoperative examinations were performed. Anesthesia approval was obtained as a result of consultation with a general anesthesia specialist at the General Anesthesia and Reanimation Department of Dicle University Faculty of Medicine. It was determined as American Society of Anesthesiologists' (ASA) class II.[14] The anesthesia team evaluated the patient's anesthetic approach and determined monitoring methods. The pediatric patient, who had been fasting for 8 hours, was given intravenous midazolam (Dormicum, La Roche Ltd., Switzerland) at 0.5 mg/kg as premedication 15 minutes before the procedure. Non-invasive blood pressure, electrocardiogram, and peripheral pulse oximetry were used for standard anesthesia monitoring. Intravenous 0.09% NaCl infusion was administered. After 5 minutes of stabilization, the patient's pulse, systolic/diastolic arterial pressure, and mean arterial pressure were recorded as baseline vital signs. The patient's vital signs were closely monitored intraoperatively, and the anesthesia process was managed safely. Oral and extraoral hygiene was performed with 0.12% chlorhexidine, and a pharyngeal tampon was placed. A lip retractor was placed to provide better vision. In our patient, scaling was performed to control plaque.

Composite resin restorations (Palfique Estelite Paste®, Tokuyama Dental Corp., Tokyo, Japan) were placed under rubber dam isolation on teeth 16, 26, 73, and 31. Fissure sealants were applied to teeth 14 and 24 as a preventive measure (**Figure 5a, b**).

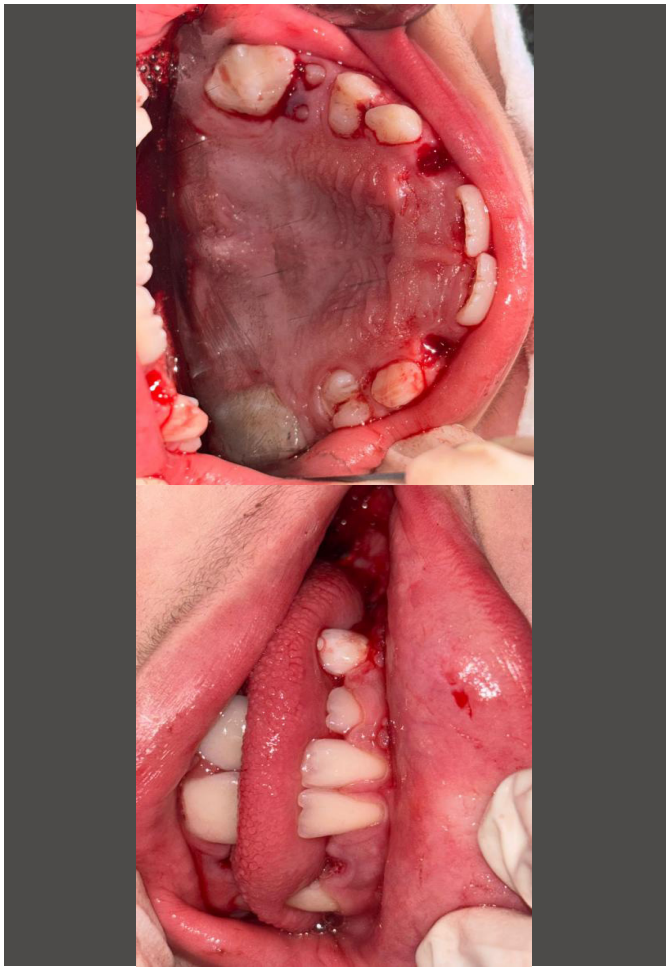

Figure 5. a) Post-operative immediate view (upper jaw), **b)** Post-operative immediate view (anterior)

Pulpotomy procedures were performed on teeth 36, 46, and 85 using mineral trioxide aggregate (MTA) (Angelus MTA®, Angelus Indústria de Produtos Odontológicos S/A, Londrina, PR, Brazil). A 2.5% sodium hypochlorite solution was used to control bleeding during the procedure. After completion of the composite restorations, stainless steel crowns (SSCs) were placed on the treated molars to ensure long-term durability.

Teeth 52, 62, 82, 74, and 75 were extracted under local anesthesia (**Figures 6a-d**). At the end of the procedure, the pharyngeal tampon was removed. Before extubation, intravenous paracetamol (10 mg/kg) (Parol Flakon®, MEFAR İlaç San., Turkiye) was administered for postoperative pain control. Following extubation, the patient was monitored in the recovery room. A liquid diet was initiated four hours after transfer to the ward.

Antibiotics and analgesics were prescribed to prevent post-operative infection risk following dental treatments. In the postoperative period, the patient received dietary recommendations to reduce caries-causing eating habits, and parents received oral hygiene education. The patient was discharged the same day. No new tooth decay was observed during the 6-month follow-up examination.

At the follow-up visit, a topical remineralization agent containing casein phosphopeptide-amorphous calcium phosphate with fluoride (CPP-ACPF) was recommended to promote enamel remineralization.

Figure 6. a) Stainless steel crown application to tooth number 36, **b)** Stainless steel crown application to teeth 85 and 46, **c)** Periapical radiographs of teeth 85 and 46, **d)** Periapical radiograph of tooth number 36

DISCUSSION

SMS is a rare genetic disorder characterized by cognitive impairment, resulting from deletions in the chromosome 17p11.2 region or mutations in the RAI1 gene. It is noteworthy that clinical and medical case reports are quite limited in the literature.[4,15-17] The characteristic craniofacial findings in our case, such as mandibular prognathism, frontal prominence, midfacial hypoplasia (depression of the nose and cheekbone), and macroglossia, are in complete agreement with the SMS phenotype described in the literature.[6,7]

In the screenings conducted, it is estimated that the prevalence of SMS in Turkiye is between approximately 1/15.000 and 1/25.000, consistent with global data. When the Turkish literature is examined, it is seen that the studies on SMS mostly focus on genetic diagnosis, clinical phenotype and psychiatric findings.[18-20]

Studies addressing the dental management of patients with SMS are quite limited in the literature.[3,4,12] No case reports have yet been found regarding the dental management and oral health rehabilitation of individuals diagnosed with SMS in Turkiye. This situation is important because the case we present is the first study in Turkiye to address the dental approach in patients with SMS.

Tomona et al.[4] report that oral hygiene deteriorates with the transition from childhood to adolescence in individuals with SMS and that the prevalence of caries increases proportionally with age. The fact that our case presented with deep secondary lesions despite being treated under general anesthesia three years ago confirms this progressive picture in the literature.

In the dental management of patients with SMS, pharmacological agents used to treat sleep disorders are a critical risk factor. Soares and Kanungo[21] reported that the high sugar content of pediatric medications and side effects such as xerostomia (dry mouth) increase caries activity by reducing the protective effect of saliva. The patient's regular use of Risperidone and Melatonin, along with uncontrolled eating behavior, supports this pharmacological and dietary caries risk in the literature. In addition, behavioral problems such as temper tantrums, impulsivity, and attention deficit,[22] which are characteristic of the syndrome, accelerate destruction by making it difficult to maintain oral hygiene at home. Therefore, a multidisciplinary approach including the preference for sugar-free drug formulations, personalized hygiene education, and strict dietary control is essential for the success of restorative treatments in children with SMS.

The case of a 5-year-old child reported in Brazil by Ferreira et al.[3] shows a complete similarity to our case in terms of frontal prominence, prognathism, and high caries risk due to neurological drug use (Risperidone, Topiramate). While the case reported in Brazil only involved the primary dentition and followed a path focused on extraction and composite restoration; in our case, more comprehensive procedures such as Stainless-Steel Crown (SSC) and fissure sealant were applied to preserve the permanent teeth. Similarly, in the 3-year-old case in India, severe destruction of the primary dentition (13 extractions) necessitated a radical approach, and SSCs were used in the restored teeth. General anesthesia (GA) becomes a necessary option in such cases with physical or mental limitations where classical behavioral guidance methods are insufficient.[23-26] In both global examples, GA was preferred due to the difficulty of cooperation. However, the history of general anesthesia in our case proves the necessity of an individualized long-term follow-up strategy in patients with SMS, instead of focusing only on immediate treatment.

Dental rehabilitation in SMS patients requires advanced medical preparation encompassing the systemic, anatomical, and behavioral manifestations of the syndrome. The case of 3-year-old India,[12] while paralleling our case in terms of severe early childhood caries (ECC), highlights the broad phenotypic spectrum of the syndrome with its complex cardiac anomalies (PFO, tricuspid regurgitation) and hand-foot anomalies (brachydactyly, polydactyly). Furthermore, in terms of anesthesia management, the case of India highlighted the need to be prepared for anatomical anomalies (macroglossia, lymphoid hyperplasia, etc.) by reporting intubation difficulties due to airway obstruction caused by enlarged lingual tonsils. Although there was no cardiac pathology in our case, such complex cases prove the vital importance of preoperative multidisciplinary evaluation. In our case and in Ferreira et al.[3] Although nasotracheal intubation, preferred in study, provides surgical field comfort, airway anomalies should always be considered a risk factor in patients with SMS. In addition, the post-operative fever and need for intensive care observation observed in our case are consistent with the literature; confirming the necessity of close monitoring (PICU follow-up) after GA in these children.

Tomona et al.,[4] in their study examining 15 SMS cases aged between 4 and 19 years, found at least one dental anomaly in more than 90% of the patients and reported that this high prevalence could be a helpful clinical indicator in the diagnosis of SMS. In this presented case, congenital absence of the lower right second premolar tooth was detected. The prognathic appearance frequently observed in individuals with SMS is thought to develop as a result of a combination of factors such as maxillary hypoplasia, protrusion of the lower anterior teeth, increased chin size, and macroglossia, rather than excessive mandibular growth.[2] In this case, maxillary hypoplasia, class III malocclusion, anterior-posterior cross-bite, and macroglossia were observed, and it was observed that these morphological features contributed to the pronounced mandibular prognathic appearance in the patient.

ETHICAL CONSIDERATIONS

- Ethical approval was not required for this case report according to institutional policies.
- Written informed consent for publication was obtained from the patient's legal guardian.
- Patient confidentiality was strictly maintained.

CONCLUSION

This first reported case from Turkiye highlights the clinical necessity of comprehensive dental treatment under general anesthesia in patients with SMS and aims to be a unique reference from treatment planning to postoperative follow-up. Additionally, it emphasizes the importance of individualized treatment planning and multidisciplinary management to ensure successful dental rehabilitation in patients with complex syndromic conditions.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient's legal guardian included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

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Author Contributions

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