Original Article

Study of Grades 2–3 Endoperiodontal Lesions Treated Non-surgically in Periodontitis Patients

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Abstract

Background: Endoperiodontal lesions, caused by bacteria, are a kind of infectious illness that may affect either the periodontal or pulp tissues and has a dismal prognosis in both cases. Their prognosis is difficult for doctors to assess. The aim of this study evaluates the Grades 2–3 endoperiodontal lesions treated non-surgically in periodontitis patients. **Materials and Methods:** The sample size for this research was 100 teeth (50 cases and 50 controls). One hundred people were utilized in the final analysis of this retrospective case–control study. Patients with periodontitis who had endoperiodontal lesions were included in the research if they had completed treatment for both endodontic and periodontal components. **Results:** Root canal therapy was associated with statistically significant changes in tooth mobility (TM), periapical index (PAI), and chewing discomfort scores from baseline, and periodontal therapy reduced probing depth (PD), clinical attachment level (CAL), sulcus bleeding index (SBI), TM, Simplified Oral Hygiene Index (OHI-S), full-mouth periodontitis severity, PAI, and chewing discomfort scores from baseline by a similar amount within 6 months. Six months following periodontal treatment resulted in significantly different values for PD (P 0.01), CAL (P 0.01), SBI (P 0.01), TM (P 0.003), OHI-S (P 0.007), full-mouth periodontitis severity (P 0.031), PAI (P 0.036), and pain during chewing (P 0.007). There were no statistically significant differences between the high and low responder groups with regard to SBI, OHI-S, or the kind of alveolar resorption of the affected teeth. **Conclusion:** We find that the prognosis of nonsurgical treatment of Grades 2–3 endoperiodontal lesions in patients with periodontitis is affected by smoking, PD, CAL, the severity of full-mouth periodontitis, and the number of root canals.

Keywords: Endoperiodontal lesions, Non-surgically, Periodontitis.

Introduction

Bacterial infectious diseases known as endoperiodontal lesions are responsible for extensive loss of periodontal tissue and pulp inflammation or necrosis.[1] The periodontal and endodontic tissues of the same tooth might show signs of these diseases.[2] Particularly in individuals with periodontitis, the situation gets more difficult when endodontic and periodontal diseases are combined. Endoperiodontal lesions remain a challenge for dentists and oral surgeons. During tooth growth and root canal construction, the apical foramen, lateral and auxiliary canals, and dentinal tubules are produced as main routes of communication between periodontal and endodontic tissues.[3] Due to their similar anatomical make-up, periodontal and endodontic tissues are closely interconnected, enabling infection to spread from one to the other in the event of pathological changes in the former. The bacterial flora associated with endodontic and periodontal illnesses has been shown to have commonalities in terms of both quantity and structure, suggesting that periodontal and endodontic tissues interact.[4-6]

Root canal therapy and early periodontal therapy are both necessary for treating endoperiodontal disorders. Some cases need periodontal and/or apical surgeries[7-9] or possible extraction because to the poor prognosis. When endoperiodontal lesions are detected in a tooth, it is crucial

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for dentists to assess the tooth's prognosis to come up with a treatment plan that has a good chance of success[10-13] Early judgments on the prognosis of endoperiodontal lesions are now made by clinicians based on their own practical experience, which is inadequate and may result in wasted treatment for some hopeless teeth, wasting both time and money. Therefore, it is up to the discretion of the treating physician to choose whether or not to retain an impacted tooth.

In 2018, the American Academy of Periodontology (AAP) published a new categorization for endoperiodontal lesions. Grades 2–3 endo-periodontal lesions are characterized by the presence of either one or more deep periodontal pockets on several tooth surfaces.[10] It has a wide variety of potential causes, and its prognostic variables, which include factors such as the severity of periodontal and endodontic lesions and the patients' underlying medical conditions, are similarly intricate. Prognosis of endoperiodontal lesions is not well understood. Very few studies have been undertaken that are well constructed to examine endoperiodontal lesions, and none of these studies have used multivariate models. Therefore, we sought to conduct a retrospective case–control study using a multivariable model to better understand the factors affecting the prognosis of teeth with Grades 2–3 endoperiodontal lesions in patients with non-surgical periodontitis and to provide a reference for clinicians to use when making decisions regarding teeth involved in endoperiodontal lesions. The aim of this study evaluates the Grades 2–3 endoperiodontal lesions treated non-surgically in periodontitis patients.

Materials And Methods

The sample size for this research was 100 teeth (50 cases and 50 controls). One hundred people were utilized in the final analysis of this retrospective case–control study. Patients with periodontitis who had endoperiodontal lesions were included in the research if they had completed treatment for both endodontic and periodontal components. This comprised nonsurgical root canal therapy of an acceptable quality and periodontal initial therapy. Patients with periodontal or periapical surgery; patients with systemic diseases such as hypertension, diabetes, heart disease, liver disease, or kidney disease; patients with incomplete medical records; and patients with root fracture or cracking, root canal, pulp chamber perforation, or external root resorption were excluded from this study.

The AAP case definitions were used to diagnose and categorize patients with periodontitis.[14] The following criteria were used for making a diagnosis of endoperiodontal lesions in teeth. The patient's awareness of pulp symptoms, such as a history of spontaneous pain or abnormal results on pulp vitality tests, is also a major diagnostic criterion. In addition to the major diagnostic criteria, secondary criteria include: (1) Red and swollen gums, (2) bleeding on probing, (3) radiographic examination revealing varying degrees of alveolar bone resorption, (4) occlusal discomfort, (5) tooth mobility (TM), (6) discoloration due to pulp necrosis, (7) purulent exudate, and (8) sinus tract.[15-17]

Following the 2017 international workshop on the classification of periodontal and peri-implant diseases and disorders, a new classification system was approved. The consensus report distinguished between endoperiodontal lesions with and without root damage when classifying endoperiodontal conditions. In addition, endoperiodontal lesions without root damage were classified as either occurring in patients with periodontitis or in patients without periodontitis. There were three tiers in each class. Teeth were collected from individuals with endoperiodontal lesions of Grades 2–3.[18] The medical records were reviewed to obtain demographic information such as age, gender, and smoking status as well as clinical and radiographic characteristics and clinical symptoms such as the presence or absence of occlusal discomfort, swollen gums, and a history of spontaneous pain. In this study, patients were classified as either current smokers or non-smokers (including ex-smokers).[19] Those who either had not stopped smoking at the time of diagnosis or who had quit within the previous 5 years were considered smokers. According to the definition, non-smokers are either those who have never smoked or people who have stopped smoking at least 5 years ago.[20]

At baseline, before periodontal therapy, and 6 months after periodontal therapy, patients' clinical parameters were recorded, including probing depth (PD), clinical attachment level (CAL), sulcus bleeding index (SBI, scored from 0 to 5),[21] Simplified Oral Hygiene Index (OHI-S, scored from 0 to 6),[22] TM (scored from I to III),[23] and alveolar resorption (horizontal and angular resorption)[24] of the involved teeth. Sites with an even radiographic appearance of an interdental alveolar ridge paralleling an imaginary line between the cementoenamel joints of neighboring teeth, or with an intrabony component 2 mm, were considered to have horizontal resorption.[24] Locations with radiographic evidence of increased bone resorption on the mesial or distal aspect of an interdental alveolar ridge and where the bottom of the oblique radiolucency was less than 2 mm apical to the most coronal level of the interproximal alveolar bone were classified as having angular resorption.[24] The severity of full-mouth periodontitis (mild, moderate, or severe) was determined by recording the values of the PD, CAL, and SBI.[14] With the use of a manual periodontal probe, we measured the clinical periodontal parameters (PD, CAL, and SBI) of each tooth in six different locations.

At baseline, before periodontal treatment, and 6 months following periodontal therapy, periapical radiographs were acquired using an intraoral X-ray equipment with an exposure of 10 mA and 70 kVp. The radiographic examinations also used a similar method, with the Rinn XCP Dental X-ray Positioning system ensuring that all X-rays were taken at consistent angles. Visualization programs were used to examine the data. The radiographic measurements included the clinical crown-root ratio (CR), the periapical index (PAI, scored from 1 to 4),[25] and the alveolar resorption type (horizontal and angular resorption).

During endodontic therapy, periapical radiographs were used to determine the number of root canals in each affected tooth.

At the outset, patients were evaluated with a series of clinical and imaging tests. The procedure to remove the infected root canal was split over two appointments. Root canal therapy begins with the placement of a rubber dam around the offending tooth. Using drills, the coronal section of the root canal was removed, and the working length was established with the aid of an electronic apex finder and afterward confirmed radiographically. Using a crown-down approach and rotational nickel-titanium devices, the canals of patients' teeth were reshaped. We irrigated the root canals with solutions of 3% sodium hypochlorite and 17% EDTA. After the paper points were removed from the root canals, calcium hydroxide paste was used to seal them. A temporary filling was made out of an intermediate restorative substance. After around 7 days, patients were contacted to return for the last stage of root canal therapy. Root canal irrigants include 3% sodium hypochlorite and 17% EDTA were used to remove the paste and clean the canals. A vertical compaction method was used to obturate the root canals with gutta-percha and zinc oxide-eugenol after they were dried with paper points. Root canal therapy's success was verified by an intraoral X-ray examination of the treated canals. The canal openings were then sealed using a flowable resin composite to a depth of at least 2 mm. Composite resin was used for the remainder of the crown repair (3M ESPE, St. Paul, Minnesota). If the composite resin was not holding on its own, a fiber post (3M ESPE, St. Paul, Minnesota) was glued into the canal. After 1–2 months, an ultrasonic scaler was used to do supragingival scaling on the whole mouth, and subsequent subgingival scaling and root planning occurred over the course of two appointments separated by 1 week.[26,27] Patients were returned to the clinic for reevaluation before periodontal treatment and again 6 months following periodontal therapy. Patients' understanding of proper oral hygiene practices, such as brushing, flossing, and using interdental brushes, was re-enforced at each appointment. Oral prophylaxis was also performed professionally, focusing on the area above the gum line. At 6 months, patients' clinical symptoms and parameters were used to determine the treatment's curative efficacy Common Benchmark.[28,29] We classified these teeth into high and poor responder groups [30,31] based on clinical symptoms and parameters 6 months after the combination periodontal-endodontic therapy and how they fared in comparison to the baseline. No pain, improved masticatory function, no signs of redness, swelling, or inflammation of the gums on clinical examination, probing pocket depth (PD) 5 mm or at least a 2 mm reduction in probing pocket depth, sensitivity (SBI) 2, descending and invariable CAL and TM, absence of sinus tract, and no increase in PAI all characterize the high responder group. Disease progression, patients with self-conscious symptoms or impaired chewing function, red and swollen gums on clinical exam, probing pocket depth (PD) >5 mm or <2 mm reduction, SBI 3, increased CAL or TM, sinus tract present, pain (+), or increased PAI are all indicators of being in the low responder group. Statistical programs were used for all analyses (SPSS, version 25.0).

Results

Clinical parameters and symptoms were compared between the start of treatment (1–2 months after root canal procedure), throughout treatment, and 6 months thereafter (Table 3). Root canal therapy was associated with statistically significant changes in TM, PAI, and chewing discomfort scores from baseline, and periodontal therapy reduced PD, CAL, SBI, TM, OHI-S, full-mouth periodontitis severity, PAI, and chewing discomfort scores from baseline by a similar amount within 6 months. Six months following periodontal treatment resulted in significantly different values for PD (P 0.01), CAL (P 0.01), SBI (P 0.01), TM (P 0.003), OHI-S (P 0.007), full-mouth periodontitis severity (P 0.031), PAI (P 0.036), and pain during chewing (P 0.007) (Table 3).

Based on their ability to heal, all the teeth were separated into two groups. There were a total of 50 teeth in the high responder group, with a mean patient age of 47.89 (split evenly between 30 male and 20 female teeth). There were a total of 50 teeth in the low responder group, with an average patient age of 48.98. There were 28 male and 22 female patients represented, for a male-to-female ratio of 1.28:1. When comparing the high and low responder groups, neither age nor gender was found to be significantly different ($P > 0.05$). High responders were more likely to be smokers than low responders, with a 17:33 ratio compared to a 30:20 ratio. There were more smokers among the poor responders (P 0.05, Table 1) than among the high responders.

Clinical criteria for high and low responders are shown in Table 2. Univariate analysis indicated that high and low responders were significantly different on measures of PD (P 0.001), CAL (P 0.001), TM (P 0.003), full-mouth periodontitis severity (P 0.003), CR (P 0.019), PAI (P 0.003), and root canals (P 0.001). There were no statistically significant differences between the high and low responder groups with regard to SBI, OHI-S, or the kind of alveolar resorption of the affected teeth.

Discussion

This study found that root canal therapy alone cannot completely cure Grades 2–3 endoperiodontal lesions in

Table 1: Demographic data of the patients in the high and low responder groups at baseline

	High responder	%	Low responder	%	*P* value
Gender					
Male	30	60	28	56	0.32
Female	20	40	22	44	
Age	47.89		48.98		0.66
Smoking					
Yes	17	34	30	60	
No	33	66	20	40	

Table 2: Clinical parameters

Parameter	High responder	%	Low responder	%
PD	6.52		8.14	
CAL	6.11		8.25	
SBI				
I	6	12	5	10
II	15	30	10	20
III	20	40	15	30
IV	9	18	20	40
TM				
0	9	18	2	4
1	16	32	9	18
2	23	46	30	60
3	2	4	9	18
OHI-S	2.15		2.48	
Full-mouth periodontitis severity				
I	19	38	5	10
II	24	48	20	40
III	7	14	25	50
Alveolar resorption type				
Horizontal resorption	30	60	18	36
Vertical resorption	40	40	32	64
CR	0.74		0.87	
PAI				
I	16	32	7	14
II	20	40	15	30
III	13	26	23	46
IV		2		10
Number of root canals				
I	20	40	9	18
II	21	42	9	18
III	7	14	22	44
IV	2	4	10	20

PD: Probing depth, CAL: Clinical attachment level, SBI: Sulcus bleeding index, TM: Tooth mobility, OHI-S: Simplified Oral Hygiene Index, CR: Crown-root ratio, PAI: Periapical index

people with periodontitis, with only TM, PAI, and chewing discomfort improving after the procedure. To completely eliminate endodontic and periodontal germs, patients with endoperiodontal illnesses often need both root canal therapy and periodontal therapy.[17] Therefore, proper periodontal therapy is essential for periodontitis patients who have teeth with endoperiodontal lesions of Grades 2–3.[3]

Methods for assessing prognostic factors include cohort studies, randomized controlled trials, and retrospective analyses. In this study, we used a case–control design to look at data from the past. Exposure information for the case–control study was collected as part of a baseline phase, before participants were categorized into groups. If the research found an association between exposure and sickness or result, the association would have to have occurred in the order suggested by the chain of causation. In addition, the recall bias is either small or can be avoided, allowing for a potentially stronger inference of causality [Table 4].

Table 3: Changes in clinical parameters

Parameter	Before periodontal therapy	%	After periodontal therapy	%
PD	7.15		8.87	
CAL	7.55		8.19	
SBI				
I	5	10	5	10
II	8	16	6	12
III	21	42	18	36
IV	15	30	21	42
TM†				
0	6	12	5	10
I	10	20	10	20
II	30	60	32	64
III	4	8	8	16
OHI-S	2.15		2.48	
Full-mouth periodontitis severity				
I	13	26	13	26
II	25	50	25	50
III	12	24	12	24
Alveolar resorption type				
Horizontal resorption	20	40	20	40
Vertical resorption	30	60	30	60
CR	0.74		0.87	
PAI				
I	15	30	6	12
II	21	42	16	32
III	12	24	24	48
IV	2	4	4	8
Number of root canals				
I	19	38	10	20
II	20	40	10	20
III	5	10	20	40
IV	1	2	10	20

PD: Probing depth, CAL: Clinical attachment level, SBI: Sulcus bleeding index, TM: Tooth mobility, OHI-S: Simplified Oral Hygiene Index, CR: Crown-root ratio, PAI: Periapical index

Table 4: Logistic regression analyses

Parameter	OR (95% CI)	*P* value
PD	2.51 (1.31–4.74)	0.007
CAL	2.45 (1.41–4.31)	0.005
Smoking	4.21 (1.21–22.69)	0.021
Full-mouth periodontitis severity	2.95 (1.21–6.48)	0.031
Number of root canals	2.88 (1.31–5.98)	0.015

PD: Probing depth, CAL: Clinical attachment level

In this study, we looked at the prognosis of endoperiodontal lesions rather than the morbidity, and we did so by classifying patients into high and low responder groups according to clinical parameters at the 6-month follow-up and then conducting univariate and multivariate analyses of the baseline factors to determine the prognostic factors of non-surgically treated endoperiodontal lesions.

The outcome of periodontal and endodontic diseases is impacted by smoking. Hyman and Reid[32] state that cigarette smokers have an increased risk of developing chronic periodontitis. Recent research by Bunaes *et al*.[33] has shown that compared to non-smokers, smokers had a negative response to periodontal therapy. There are three reasons for why cigarette smoking worsens the outlook for individuals with periodontitis. Beginning with the immune response, periodontal healing ability, and microbiota composition, smoking has a negative effect on periodontal tissue.[34-37] Second, nicotine reduces blood flow and metabolism because it narrows blood vessels in the mouth and throat.[38] Third, smoking inhibits the development and attachment of periodontal fibroblasts as well as osteoblast activity, which may reduce the formation and regeneration of new alveolar bone attachments.[39,40] In addition, smoking is associated with an increased risk of developing apical periodontitis, which may lead to the loss of a tooth that has already been treated with endodontics. Other studies, however, revealed no significant association between smoking and apical periodontitis after adjusting for age, marginal bone level, and quality of root canal fillings. More research is needed to understand how smoking contributes to apical periodontitis.[41-44]

Both the PD and CAL values of teeth with periodontitis represent the disease's severity. The initial periodontal therapy is less likely to be successful in teeth with advanced periodontitis. The periodontal condition of the impacted teeth may have an effect on how the pulp inflammation resolves. The periodontal health of the afflicted teeth was shown to be a predictor of success with nonsurgical root canal treatment in a 9-year retrospective research by Khalighinejad *et al*.[19]

The severity of full-mouth periodontitis was shown to be a significant predictor of endoperiodontal lesions in this study. Positive outcomes in periodontal health after root canal therapy have been related to healthy gums overall. Patients with periodontal disease had a 5.19-fold higher chance of developing apical periodontitis in endodontically treated teeth than those without periodontal disease, as shown by research by Ruiz *et al*.[45] It has been hypothesized that the prognosis of apical periodontitis in endodontically treated teeth is affected by the presence of dentinal tubules, which are exposed to the oral environment after the treatment of severe periodontitis.[45]

The number of root canals performed varied considerably between the high and low responder groups, as shown by the results of the logistic regression analysis. Root canal therapy is more challenging on multirooted teeth due to their smaller root canals. Root furcation and intricate root-canal connections hinder attempts to treat periodontal and endodontic inflammation. However, the clinical survival rate of multirooted teeth is higher than that of single-rooted teeth because not all roots in multirooted teeth suffer the same loss of supporting tissue and certain surgeries, such as root resection and root separation, may save the involved multirooted teeth.[45]

Determining the health of the suspect tooth's pulp was essential to this inquiry. We examined for teeth exhibiting symptoms of pulp inflammation, such as a patient's report of pain that came on suddenly, a negative result on a pulp viability test, a tooth's discoloration as a result of pulp necrosis, pain on palpation or percussion, or pain in the sinuses. During the root canal procedure, the pulp was examined for its health. Root canal treatment was suspected to be necessary after the history, clinical symptoms, and radiographic results all pointed to the presence of an infected root canal system. In patients with periodontitis, this is the first case–control study to use a multivariable model to examine the predictive aspects of endoperiodontal lesions of Grades 2 and 3, which are often treated non-surgically. The results showed that endodontic issues like PAI were not connected to the prognosis of the impacted teeth. These results may help clinicians determine the likely outcome of endoperiodontal lesions and provide individualized treatments that take into account each individual's preferences. Nonetheless, there are a few problems with this study. For one, the link between microorganisms and endoperiodontal disorders has not been definitively established. All of the teeth we collected displayed symptoms of both periodontitis and pulpitis, but we were unable to provide evidence through bacterial detection; periodontitis was diagnosed using the AAP case definitions, and pulpitis was determined by symptoms and clinical examinations, such as a spontaneous pain history, PAI, sinus tract, or having a negative or altered pulp vitality test. While this method may not capture all cases of endoperiodontal lesions in teeth when pulpitis symptoms are absent, all of the teeth included in the study did have to exhibit endoperiodontal lesions. Second, this study used conventional radiography, however, CBCT imaging might better aid clinicians in identifying bone resorption of afflicted teeth and counting root canals. In addition, well-designed follow-up studies, such as randomized controlled trials, are required to verify the findings of the present study.

Conclusion

We find that the prognosis of nonsurgical treatment of Grades 2–3 endoperiodontal lesions in patients with periodontitis is affected by smoking, PD, CAL, the severity of full-mouth periodontitis, and the number of root canals.

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