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ORIGINAL ARTICLE

Histopathological Investigation of Dental Pulp Reactions Related to Periodontitis

Mohammad SABETI, Hossein TAYEED, Gregori KURTZMAN, Fatemeh MASHHADIABBAS, Mohammadreza Talebi ARDAKANI

ABSTRACT

Objective: Since the 1960s, there has been contradictory evidence regarding the association between periodontal pathology and the status of the pulp. The purpose of this study was to evaluate the histopathological changes of pulp tissue with severe periodontal disease, including vertical bone loss involving the major apical foramen, and compared them with the histological pulpal status of teeth with healthy periodontium.

Methods: This case-controlled study included 35 intact teeth with severe periodontitis of hopeless prognosis (test group) and 35 teeth without periodontitis extracted for orthodontic reasons (control group). For each tooth, periodontal and endodontic parameters such as probing depth and pulpal vitality were recorded, and the pulp tissue was evaluated histologically. The data were analysed with a significance level of 0.05.

Results: Vital pulp was observed in all specimens of both groups (P=1). Pulpal inflammation in the apical portion was observed in 81.71% of the severe periodontitis group, whereas all teeth in the control group demonstrated no signs of pulpal inflammation. Dystrophic calcification and pulp stones were observed in 7.5% of the periodontitis group and 5.7% of the healthy group (P>0.05). Pulp fibrosis was observed in 22.8% of the periodontitis group and 2.8% of the control group (P=0.012). Pulpal necrosis was not noted in either group. In the periodontitis group, internal resorption was present in 22.8% of cases (P=0.005) and external resorption was present in 80% of cases (P<0.001). In the control group, no internal or external resorption was observed in any of the specimens. No differences were noted in the study patients with regard to sex or age.

Conclusion: Periodontal disease does not significantly affect pulp vitality and pulpal calcifications. However internal and/or external resorption was significantly different between the two groups as well as apical inflammation and pulp fibrosis.

Keywords: Aggressive periodontitis, calcification, chronic periodontitis, dental pulp, histology, perapical tissue, periodontium

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HIGHLIGHTS

- Severe periodontitis does not to lead to pulpal necrosis.
- Severe periodontitis does not significantly affect pulp vitality and pulpal calcifications.
- Severe periodontitis seems to lead to internal/external resorption as well as apical inflammation and pulp fibrosis.
- Morphological pulpal degeneration of periodontal origins seem to take place mostly in the radicular portion.

INTRODUCTION

In the absence of coronal caries, it may be challenging to determine where an infection has initiated and whether the infection spread from periodontitis to the pulp or vice versa (1). Dental pulpal tissue communicates with the surrounding periodontal tissues through various paths such as the apical foramen and lateral or accessory root canals (2). It is not uncommon for bacteria to originate in the pulp and spread through these pathways to initiate a periodontal lesion (3). Previous clinical and animal studies have reported that different pulpal pathologies may cause alterations in the periodontal tissue (4-6). The impact of periodontitis on the dental pulp is a little more debatable and has been studied for the past decades (7). The bacterial pathway from a periodontally diseased tooth

to the pulpal tissue has been reported to be either through the canal anatomy directly connecting the pulp and periodontal ligament (PDL) or via the radicular dentinal tubules (8-10).

For pathologic changes of endodontic origin, appropriate endodontic therapy, such as root canal therapy, usually resolves the periodontal defect as well (10, 11). When it comes to pulpal pathology of periodontal origins, the matter is more controversial (12-14). Dentinal tubule penetration by bacteria from the periodontium is dependent on cementum coverage of the root, with the cementum acting as a physical barrier to prevent or limit bacterial invasion into the root canal system (15). Periodontal disease progresses from the crest apically along the root surface and causes loss of cementum as the crestal bone continues to be lost. This loss of the cementum barrier may exposes the dentinal tubules and allow bacterial entrance into the tooth, which results in endodontic involvement in some cases (16, 17). As a result, patients with severe periodontitis may present with pulpal involvement (18, 19). Previous studies have shown calcifications in the pulp chamber and root canal system with fibrosis of the pulpal tissue (20). The literature presents conflicting evidence on whether periodontal destruction can alter the pulpal tissue and result in pulpal necrosis and dystrophic changes. However, most previous investigations were conducted with methodological issues. For instance, in a 1978 study by Mazur, the authors fail to include controls and excluded teeth with periodontal destruction involving the major apical foramen (21).

The current study aimed to investigate the relationship between severe periodontitis and histopathological alterations in pulpal tissues by evaluating the pulp of periodontally hopeless teeth affected by severe periodontitis with bone loss involving the major apical foramen.

MATERIALS AND METHODS

Patients and specimen collection

All patients signed an informed consent form before initiation of any treatment and evaluation. The study was approved by the Institution Review Board in Shahid Beheshti University of Medical Sciences (Iran). Clinical endodontic and periodontal data, including pulp vitality, percussion, palpation, pocket depth (PD), degree of mobility, bleeding on probing (BOP), and attachment level (AL) were collected for each patient at the baseline visit. The degree of bone loss was evaluated both radiographically (Fig. 1) and clinically before extraction. Teeth that had advanced periodontal disease with inadequate osseous support were included in the study.

The followings were the inclusion criteria:

- Patients 35 to 60 years of age
- Posterior teeth with attachment loss
- Teeth with BOP indicative of active periodontal disease
- Teeth with bone loss greater than 75%
- Teeth with grade 3 mobility
- Endodontically asymptomatic teeth

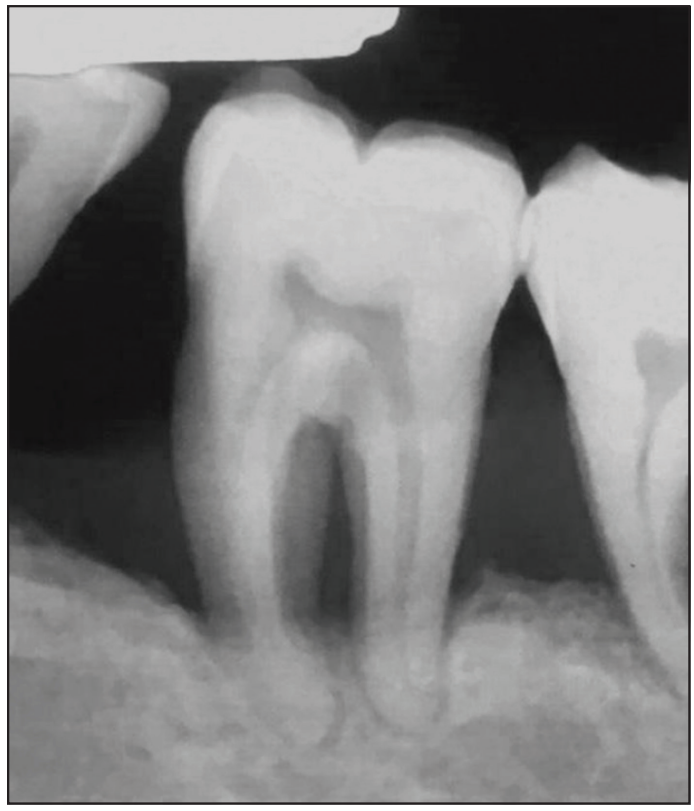

Figure 1. Radiograph of a hopeless mandibular left first molar from 59-year-old woman with severe chronic periodontitis

- Teeth free of restorations and decay
- Patients with no recent history of periodontal therapy for one year before the study assessment.
- Healthy patients without any systemic disease
- Non-smokers
- Patients without any current or previous medication use associated with pulpal calcifications such as statins

The test group had 35 structurally intact teeth (without restorations or caries) with severe periodontitis and a hopeless prognosis. The control group included 35 teeth with healthy periodontium (absence of periodontal disease) extracted for orthodontic reasons. A sample size of 35 for each group was selected based on power analysis calculations with an error of alpha=0.05 and a power of 0.8. All patients were non-smokers and systemically healthy. The average age of the patients was 46.32±9.45 years for the test group and 34.02±10.31 years for the control group. The test group consisted of 12 males and 23 females, whereas the control group had 16 males and 19 females.

Specimen processing

All teeth were extracted under local anesthesia with 2% Lidocaine with 1:100.000 epinephrine (Darou Pakhsh Pharmaceutical MFG, Tehran, Iran). Immediately after extraction, the teeth were immersed in a 10 % formalin solution for 3 days. All specimens were then decalcified in ethylenediaminetetraacetic acid (EDTA) and embedded in paraffin. Serial sections of the embedded specimens were made in 5-micrometre thickness,

taken longitudinally to the tooth's long axis and stained with hematoxylin and eosin (H&E) (Beijing Ding Guo Biotechnology Company, Beijing, China). Light microscopic (Olympus, Tokyo, Japan) evaluation of the sections was performed by two independent pathologists blinded to the clinical data.

Histological evaluation of dental pulp

All specimens were examined under a compound light microscope (Magnus MLX, Olympus Laboratory, Chennai, India), using a magnification of 10x, 40x and 100x, for inflammatory and degenerative changes (fibrosis, calcification, and necrosis). The relative degree of inflammation was scored (PPF X200) as follows: score 0: absent/slight (0-10%); score 1: mild (10%-30%); score 2: moderate (30%-50%); score 3: severe (>50%). The presence of fibrosis, oedema, and calcification was recorded. Fibrosis was defined as increased fibroblast and collagen fibers concentrations as observed visually. Edema was defined as the accumulation of interstitial fluids within the pulp, and necrotic specimens were categorised according to whether the necrosis was partial (areas of vital pulpal tissue interspersed with necrotic tissue) or complete (absence of vital pulpal tissue). Internal and external resorption were identified by the presence of large multinucleated odontoclasts occupying resorption lacunae on the canal walls or root surface. All parameters were checked and recorded by a single experienced oral pathologist.

Statistical analysis

The Mann-Whitney U rank-sum test was used to compare histological changes within the dental pulp of teeth with different grades of attachment loss and different tooth types. A contingency coefficient was calculated to analyse the interrelationship between the severity of periodontitis and histological changes in the pulp. The contingency coefficient was also used to analyse the interrelationship between different grades of attachment loss and histological changes in the pulp. All statistical analyses were performed with a two-tailed chi-square test and t-test test at a significance level of 0.05, using SPSS 13.0 software (SPSS Inc., Chicago, IL, USA).

RESULTS

Longitudinal histological sections were evaluated by light microscopy, and the inflammatory infiltrate was scored (PPF X200). In Table 1 the frequencies of various histopathologic characteristics of dental pulp among study groups has been

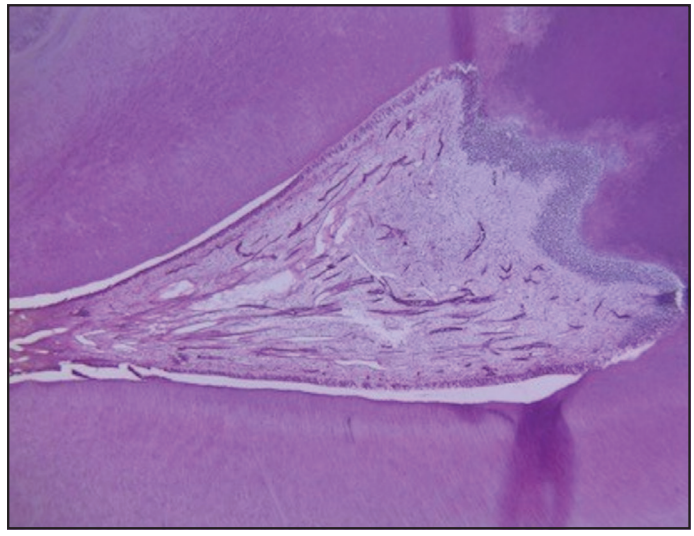

Figure 2. Representative section of pulp tissue from a tooth with severe periodontitis that shows vital coronal pulp (H and E staining, ×40)

TABLE 1. Frequencies of various histopathologic characteristics of dental pulp among study groups

Histopathologic characteristics	Group				
	Severe periodontitis		Control		
	Number	Percentage	Number	Percentage	P value*
Vitality					
-	0	0	0	0.0%	0.001
+	35	100%	35	100.0%	
Pulpal inflammation					
-	6	17.1%	32	91.4%	0.031
0-10%	23	65.71%	3	8.5%	
10-30 %	2	5.7%	0	2.8%	
30-50 %	1	2.8%	0	0.0%	
>50 %	3	7.5%	0	0.0%	
Pulp stone					
-	32	92.5%	33	94.2%	>0.05
+	3	7.5%	2	5.7%	
Fibrosis					
-	27	77.1%	34	97.1%	0.012
+	8	22.8%	1	2.8%	
Internal Resorption					
-	27	77.1%	35	100.0%	0.005
+	8	22.8%	0	0.0%	
External Resorption					
-	7	20.0%	35	100.0%	<0.001
+	28	80.0%	0	0.0%	

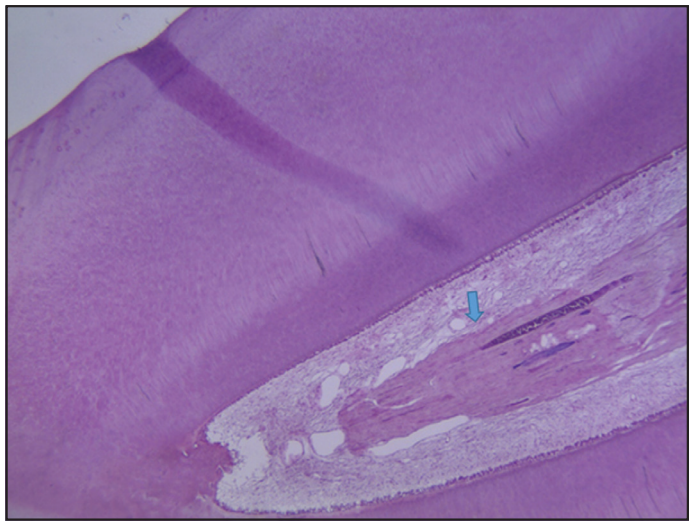

Figure 3. Representative section of vital pulp tissue from a tooth with severe periodontitis that shows dystrophic calcification in apical area (H and E staining, ×40)

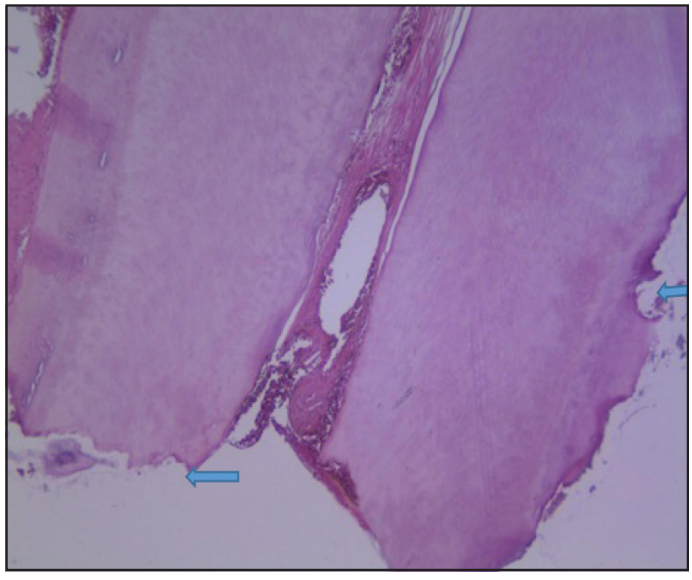

Figure 4. Representative section of pulp tissue from a tooth with severe periodontitis that shows internal and external resorption in periapical area (H and E staining, ×40)

reported. Microscopic sections indicated that the pulp ranged from intact to pathological in different sections of the same tooth (Figs. 2-4). The mean pocket depths and standard deviations were 1.58±0.29 mm in the control group and 7.6±1.81 mm in the periodontitis groups, (P=0.001) (Fig. 5). The mean AL values and standard deviations were 0.21±0.31 mm in the control group and 6.88±2.31 mm in the periodontitis groups (P=0.001) (Fig. 5). All teeth were endodontically asymptomatic according to our inclusion criteria.

The specimens were histologically evaluated with microscopy. A vital pulp was observed in all specimens of both groups (P=1). Pulpal necrosis was not noted in either groups. Pulpal inflammation in the apical portion was observed in 81.71% of the severe periodontitis group, whereas only 11.3% of teeth in the control group demonstrated signs of pulpal inflammation

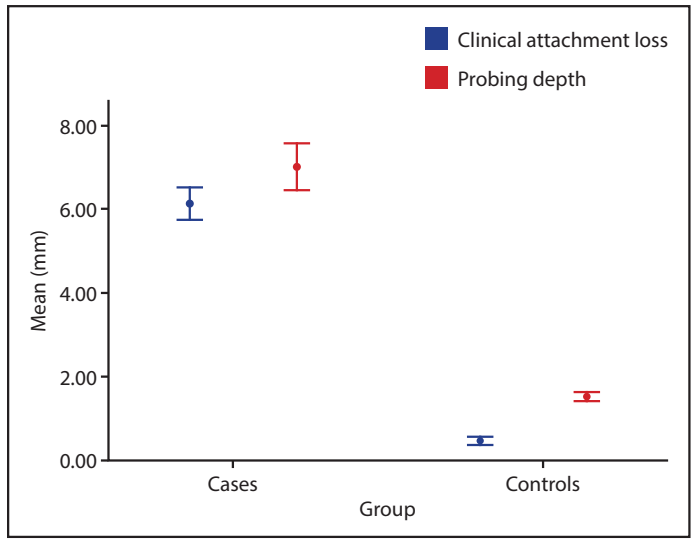

Figure 5. Boxplots of probing depth and clinical attachment loss means and standard deviations (mm) in case and control groups
*t test comparisons between two groups showed statistically significant differences in clinical attachment loss and probing depth (P=0.001)

(P=0.031). Inflammation was only present in the apical portion of the teeth with no inflammation observed in the coronal portion of either groups (Fig. 2). Dystrophic calcification and pulp stones were seen in 7.5 % of the periodontitis group and 5.7% of the healthy group, with no significant difference between the two groups (P>0.05). On the other hand, a significant difference was found in pulpal fibrosis, with 22.8% of the periodontitis group and 2.8% of the control group (P=0.012). Internal and/or external resorption was significantly different between the two groups. In the periodontitis group, internal resorption was present in 22.8% of cases (P=0.005) and external resorption was present in 80% of cases (P<0.001). In the control group, no internal or external resorption was observed in any of the specimens. No differences were noted in the study patients with regard to sex or age.

DISCUSSION

The current study evaluated the pulpal tissue alterations of teeth with severe periodontitis and compared them with periodontally healthy teeth. We found that teeth with severe periodontitis with a hopeless clinical prognosis could result in fibrosis, apical inflammation and internal/external resorption but did not lead to pulpal necrosis. This study was a case-control investigation and reaffirmed the previous studies' findings that inflammation and degenerative pulpal alterations such as fibrosis are the most frequent impact of periodontal disease on pulpal tissue (5, 22, 23). However, the previous studies utilised cross-sectional histological sections, whereas this study used longitudinal histological sections. This type of sectioning enabled assessment of the entire occlusal to the apical portion of each specimen with their accessory root canals observable in the histological slide.

The mechanism of inflammation in dental pulp is the same as for other tissues. Based on the duration and intensity of the stimulant, the pulp responds differently, ranging from mild inflammation to complete necrosis (24). All of these processes

may happen without pain (25). Moreover, the pulp can react to irritation with a range of degenerative alterations, including fibrosis and calcifications (26). A plethora of nonspecific inflammatory mediators, including serotonin, bradykinin, histamine, arachidonic acid metabolites (prostaglandin E2) and various interleukins, are released in this process response to different stimuli such as trauma and bacterial contamination. The secretion of these mediators leads to vasodilatation and increases vascular permeability (26). The pulp then becomes oedematous due to increased serum protein filtration from the blood vessels (26). In addition, cellular metabolism is restricted due to hypoxia and ischemia, which eventually results in pulpal necrosis (26). Finally, the necrotic tissue becomes infected by various microorganisms penetrating the dentine-pulp complex through the exposed dentinal tubules (24). Necrosis usually does not occur if dentinal tubules are preserved by intact cementum, as cementum acts as a protective barrier to bacterial movement through the dentinal tubules (26).

The study by Seltzer et al. (8) was one of the first studies to emphasise the importance of lateral and accessory root canals in distributing noxious substances from the periodontal apparatus into the dental pulp. They found that severe periodontitis may lead to a greater incidence of inflammatory and degenerative alterations in the dental pulp. However, their conclusion was not supported by Mazur and Massler (21). Other studies also reported no significant relationship between periodontal disease and conditions of the dental pulpal tissue (27, 28). Interestingly, in our study inflammatory changes were observed only in the apical part of the pulp and no necrosis was observed in any of the specimens. This is in agreement with the histological study by Aguilar et al. (19), which indicated that morphological pulpal degeneration took place mostly in the radicular part of the tooth with no coronal changes during periodontal destruction involving the major apical foramen. In contrast to our findings, other histological studies found pulpal degeneration and pulpal necrosis of severely periodontally involved teeth (29, 30).

Previous studies have also examined the association between root planing and pulpal inflammation (31). Rotstein et al. (7) reported that root planing could result in severed blood vessels within lateral canals. Severed blood vessels allow microorganisms to invade the pulp, resulting in inflammation and necrosis. For this reason, the teeth in our study did not receive any periodontal treatment, including scaling and root planning for one year before the study assessment.

In the current study we also demonstrated that severe periodontits is associated with resorption. This findings are congruent with previous studies by Bergenholtz and Lindhe (28) and Wan et al. (32). In the animal study by Lindhe and Bergenholtz (28), the authors verified that periodontal disease could result in external and morphological alterations in the pulpal tissue. In addition, they indicated that fibrosis may occur due to the dynamic interaction between severe periodontitis and the pulpal tissues (28).

Some limitations were found in this present study. First, it did not compare the effects of various severities of periodontitis on pulpal tissues. In addition, although the frequency of degenerative alterations, such as pulpal calcification, usually increases in older individuals, our study included patients with severe periodontitis, ranging from 35-60 years old, and age was not found to be a contributing factor in the development of pulpal degeneration. Only asymptomatic cases with normal pulps were included in this study and this may explain our results where no teeth had pulpal necrosis. If symptomatic cases were to be included, there is a possibility that pulpal necrosis would have been observed due to an already compromised pulp.

CONCLUSION

Our study result reaffirmed the findings of previous studies that severe periodontitis involving the major apical foramen does not significantly affect pulpal vitality and inflammation of the coronal pulp, as well as pulpal calcifications. However, our study indicates that severe periodontitis can lead to apical pulpal inflammation, fibrosis and resorption. More case-controlled studies with larger sample sizes are required to further analyze to the relationship between periodontitis and its effects on the pulp.

Disclosures

Conflict of Interest: Authors deny any potential conflict of interest.

Ethics Committee Approval: The Institution Review Board in Shahid Beheshti University of Medical Sciences (Iran) approved the study. (July 2017)

Peer-review: Externally peer-reviewed.

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