EVALUATION OF HISTOPATHOLOGIC CHANGES OF DENTAL PULP IN ADVANCED PERIODONTAL DISEASES

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Abstract- The adverse effects of periodontal disease on dental pulp have been debated for many years. This case-control study was performed to assess the possible effects of advanced periodontal disease on the structure of dental pulp. Fifty-two permanent teeth extracted because of advanced periodontitis with ≥ 5 mm attachment loss and grade III mobility were compared to 52 control teeth, obtained from systemically healthy adults. Two groups were matched for age and teeth types. Inflammation, fibrosis, calcification and necrosis were observed in the 27.8- 40%, 0-59.4%, 0-26.4% and 0-20.9% of the different sections of the study group, and 0%, 9.7-50%, 0-11.6% and 0% of the control group ($P < 0.05$). Abnormal pulp tissue was observed in the 33.3-88.1% and 12.9-50.5% of different sections of the study and control groups respectively ($P < 0.05$). Complete necrosis of dental pulp occurred only when depth of adjacent periodontal pocket reached the apical third of the root. There was an increase in frequency of pathologic changes as the depth of periodontal pocket increased ($P = 0.00$). We conclude that advanced periodontal disease can affect the dental pulp, although not necessarily leading to complete pulp disintegration. Careful consideration of diagnostic and treatment planning in patients with endodontic-periodontal involvement is recommended. Fibrosis and diffuse calcification of dental pulp in teeth with advanced periodontal involvement may endanger root canal therapy, if needed.

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INTRODUCTION

The possibility that periodontal disease might be related to, or cause, pulpal disease was reported by Colyer and Cahn in the 1920s (1). The most demonstrable relationship between the two tissue is via vascular system in the presence of the apical foramen and aberrant, accessory communications. These channels, when patent, may serve as potential routes of inflammatory interchange (2). There is general agreement that pulpal disease can initiate or perpetuate periodontal disease; the opposite theory is controversial (3). Some have found a strong association between periodontal disease and inflammatory and degenerative pulp changes (4, 5). Still others have not found this association (3, 6).

Variation of opinions may be due to differences in periodontal diagnostic criteria, difficulties in pulpal tissue fixation, lack of healthy controls, or lack of clear histologic criteria for definition of

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observations (3, 7). We designed this case-control histopathologic study to assess pulp changes in advanced periodontal disease. We think may be this is the first well matched case-control study of these changes.

MATERIALS AND METHODS

Fifty-two teeth in the study group were obtained from the periodontics department of the Faculty of Dentistry of Tehran University of Medical Sciences and some private offices. Fifty-two teeth in the control group were obtained from the Oral and Maxillofacial Surgery Department of the same faculty and some private offices. Two groups were matched for age and tooth type. Study group included teeth with advanced periodontitis and questionable or hopeless prognosis which had 5 mm or more attachment loss and grade III mobility and lack of gingival recession or hypertrophy. Teeth should had at least one of these criteria:

a) advanced bone resorption to 2/3 of root length, proved by radiography,

b) grade II or III furcation involvement,

c) inaccessible areas,

d) non maintainable areas.

Control group included teeth without any periodontal involvement which were extracted for orthodontic or prosthetic reasons. Exclusion criteria were history of systemic diseases (such as rickets, diabetes, hypertension), long term use of any drugs, previous periodontal therapies (such as scaling or root planning), and history of significant dental trauma, bruxism or clenching.

Before extraction, oral physical examinations were done and radiographies were taken as needed and only teeth with intact crowns and no restorations were selected.

Teeth were extracted gently, under complete local anesthesia. The pulp was exposed by making a deep hole, at the roof of the pulp chamber, with a fissure bur (SR-11, MANI-DIA BURS). Immediately then teeth were immersed in 10% formaldehyde solution for at least 10 days for complete fixation. After that, teeth were cleaned with a sponge and root lengths were measured from cemento- enamel junction (CEJ) to apex.

Decalcification was performed with immersion of teeth in 10% formic acid for 3 months. Radiographies were performed for proving complete decalcification, at the end of 3 months. Crowns were cut at the CEJ. Two mm transverse sections of roots were performed and multiple samples of each section were examined by light microscope (Olympus B×40F4) after H & E staining.

Inflammation was graded as none, mild, moderate and severe, according to Yaltirik *et al.* (8). Fibrosis was defined as increased fibroblasts and collagen fibers. Calcification was graded as single, multifocal and diffuse. Necrosis was graded as partial or complete. If none of the above mentioned findings were seen, dental pulp was considered normal. A single experienced oral pathologist reviewed the slides. In the case group, teeth (roots) were then subcategorized according to depth of the adjacent periodontal pocket: 1) group 1: coronal third of the root, 2) group 2: middle third of the root, 3) group 3: apical third of the root.

Data were analyzed using Chi square, Fisher exact, Mann-Whitney, Kruskal Wallis and *t* test. A P value ≤ 0.05 was considered statistically significant.

RESULTS

Mean age of study and control groups were 37.8 (±11.8) years and 35.0 (±18.6) years, respectively ($P = 0.63$).

Totally, 548 slides (276 and 272 in the study and control groups, respectively) were examined. Inflammation was observed in the 27.8-40% (mean, 33%) of different sections of study group and none of sections of the control group (Table 1, Fig. 1). Inflammation was mild in most sections and always chronic. Fibrosis was seen in the 0-59.4% (mean, 45.3%) of the study group sections and 9.7-50% (mean, 30.3%) of the control group sections ($P < 0.05$ in 4 to 12 mm sections from the apex) (Table 2, Fig. 2). Calcification was seen in the (0-26.4%, mean: 18.4) of the study group sections and 0-11.6% (mean, 5.65%) of the control group sections ($P < 0.05$ in 2 to 12 mm sections) (Table 2, Fig. 3).

Table 1. Prevalence of inflammation in the "case group"

Distance from apex (in millimeters)	No inflammation	Mild inflammation	Moderate inflammation	Severe inflammation	Inflammation
2	72.3%	14.9%	9.9%	3%	27.8%
4	69.5%	19%	7.6%	3.8%	30.4%
6	67.9%	19.8%	7.5%	4.7%	32%
8	68.3%	20.8%	5.9%	5%	31.7%
10	69.7%	19.1%	6.7%	4.5%	30.3%
12	67.4%	23.3%	9.3%	0%	32.6%
14	60%	10%	30%	0%	40%
16	66.7%	33.3%	0%	0%	33.3%

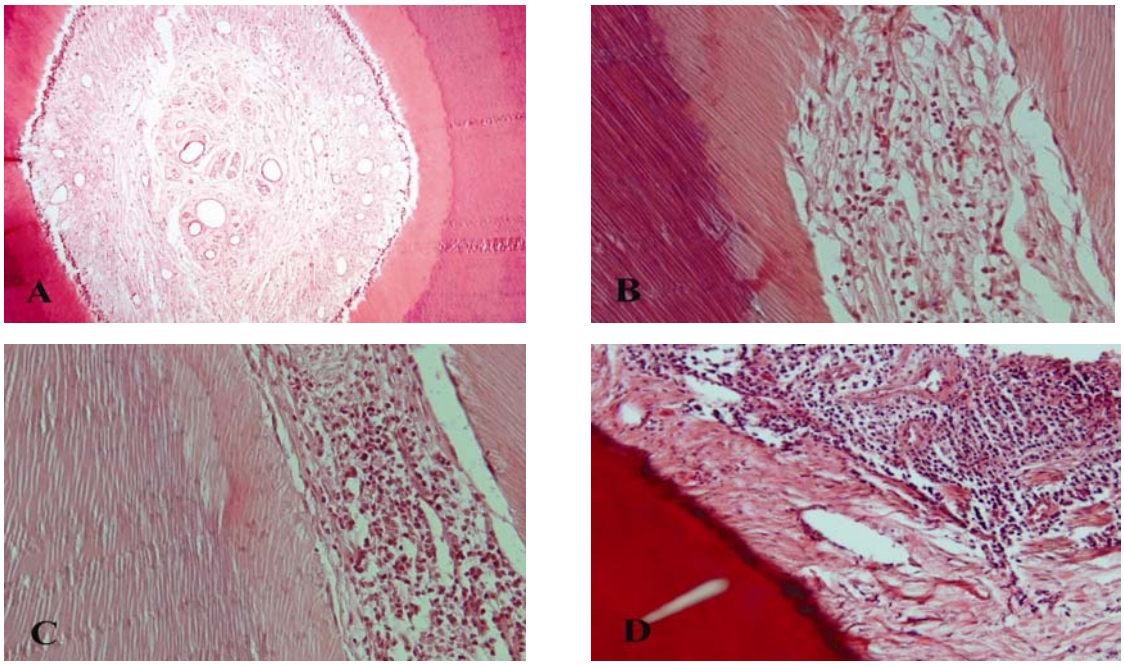

Fig. 1. A: Histologic view of an uninflamed pulp in control group (× 100). B: Histologic view of a pulp affected by mild inflammation in case group (× 400). C: Histologic view of a pulp affected by moderate in inflammation in case group (× 200). D: Histologic view of a pulp affected by severe inflammation in case group (× 200).

Table 2. Comparative percent of calcification and its different patterns in histologic sections of two groups*

Distance from apex (mm)	Fibrosis		Total calcification		Single calcification		Multiple calcification		Diffuse calcification	
	case group	control group	case group	control group	case group	control group	case group	control group	case group	control group
2	59.4%	49.5%	20.8%	6.3%	3%	5.3%	6.9%	0%	10.9%	1.1%
4	59%	40%	22.9%	11.6%	2.9%	9.5%	8.6%	1.1%	11.4%	1.1%
6	56.6%	30.3%	26.4%	11.1%	4.7%	6.1%	9.4%	4%	12.3%	1%
8	52.5%	23.5%	25.7%	8.2%	3%	3.1%	12.9%	4.1%	9.9%	1%
10	50.6%	14.5%	25.8%	4.8%	3.4%	2.4%	12.4%	1.2%	10.1%	1.2%
12	44.2%	9.7%	25.6%	3.2%	0%	0%	16.3%	0%	9.3%	3.2%
14	40%	25%	0	0%	0%	0%	0%	0%	0%	0%
16	0%	50%	0	0%	0%	0%	0%	0%	0%	0%

*Data are given as percent.

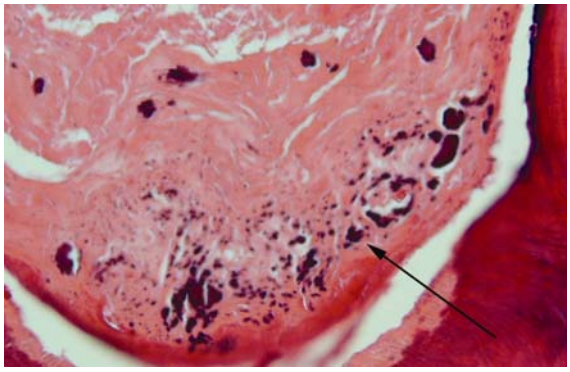

Fig. 2. Histologic view of a pulp affected by fibrosis in case group (diffuse calcification in also shown by arrow) (×200).

This difference was related to multiple and diffuse calcifications; and single calcifications were not different between groups. Necrosis was seen in the 0-20.9% (mean, 14.4%) of the study group sections and none of the control group sections ($P < 0.05$ in 2 to 12 mm sections from the apex) (Table 3, Fig. 4). Complete necrosis was seen more frequently than partial necrosis.

Abnormal pulp tissue (defined as presence of either inflammation, fibrosis, calcification, necrosis or any combinations of them) was seen in 33.3-88.1% (mean, 77.8%) of the study group sections and in 12.9-50.5% (mean, 32.5%) of the control group sections ($P < 0.05$) (Table 4).

Sections of the study group were further subcategorized according to depth of the adjacent pocket into coronal third, middle third and apical third. 3.6% of sections belonged to first group, 55.4% of sections belonged to second group and 41% of sections belonged to the third group. Prevalence of inflammation was not different in 3 groups ($P = 0.2$). Prevalence of fibrosis was inversely related to depth of the pocket ($P = 0.00$).

Calcification in the third group was more prevalent ($P = 0.02$). Necrosis was more prevalent in the third group ($P = 0.00$). Complete necrosis was seen only in the third group. Abnormal pulp tissue was seen in 65%, 70.2% and 98.7% of the sections of the three groups (1, 2 and 3 respectively) ($P = 0.00$).

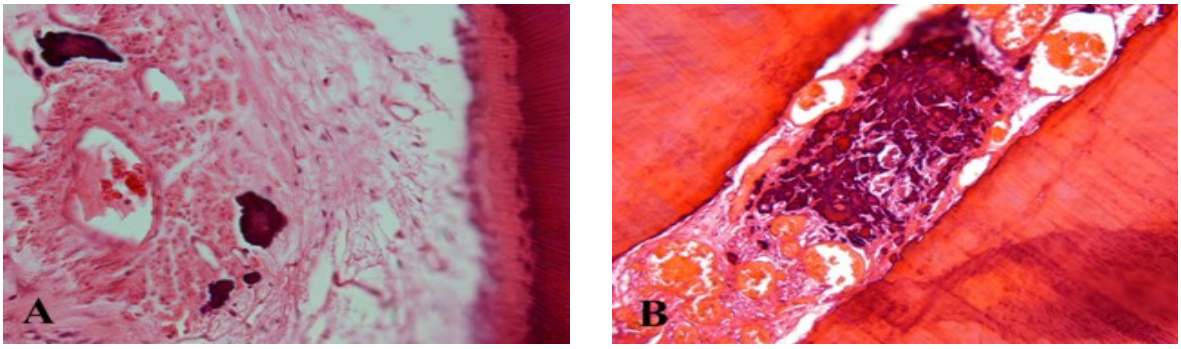

Fig. 3. A: Histologic view of a pulp affected by multiple calcification; in case group (× 400). B: Histologic view of pulp affected by diffuse calcification; in case group (×200).

Table 3. Comparative percent of necrosis in histologic sections of two groups*

Distance from apex (in millimeters)	Complete necrosis in case group	Partial necrosis in case group	No necrosis in case group	No necrosis in control group
2	19.8%	6.9%	73.3%	100%
4	21%	6.7%	72.4%	100%
6	18.9%	9.4%	71.7%	100%
8	17.8%	7.9%	74.3%	100%
10	16.9%	9%	74.2%	100%
12	20.9%	2.3%	76.7%	100%
14	0%	0%	100%	100%
16	0%	0%	100%	100%

*Data are given as percent.

Table 4. Comparative percent of abnormal pulp in histologic sections of two groups

Distance from apex (in millimeters)	Abnormal pulp in case group	Abnormal pulp in control group
2	88.1%	50.5%
4	87.6%	44.2%
6	84%	35.4%
8	79.2%	26.5%
10	75.3%	15.7%
12	76.7%	12.9%
14	50%	25%
16	33.3%	50%

DISCUSSION

There are many articles concerning pulp histology in periodontal disease (3, 4, 9). The criticism on many of these studies is the lack of well matched control groups. Only this form of study, if well designed, can show either pulp changes are the result of periodontal disease, or normal pulp changes with age or even result from technical difficulties of perfect pulp tissue fixation (2, 3). Some authors believe that history of periodontal treatments can induce histologic alterations in pulp (3); so the importance of case selection can not be over emphasized.

In the majority of previous studies, severity of periodontal disease was not very well defined or quantified (1, 3, 4). We did our best to define the inclusion criteria clearly and repeatable.

Adequate pulp fixation has always been a challenge, and artifacts resulting from inadequate fixation are described as evidence of pathosis. Fibrosis and reticular atrophy are classic examples of these artifacts (10).

Langeland's studies have demonstrated that merely dropping a tooth in a jar of formalin, even if done immediately after extraction, is inadequate to permit subsequent critical examination of the dental pulp (3). In a case report by Torabinejad and Kiger, the apical 2-3 mm of the roots was sectioned with a fissure bar and an opening was made into the pulp chamber with a round bur and fixed with 10% buffered formalin (9). This technique destroys the apical pulp tissue; so we preferred to make a coronal hole into the pulp chamber and delete coronal pulps from the study. Previous studies have used longitudinal sections of dental pulp (1, 3, 4, 9). We studied on transverse sections. This type of sectioning reduces the risk of pulp rupture and loss during tissue handling, because of smaller size of pulp tissue. Furthermore in this sectioning, evaluation of all pulp tissue in a discrete distance from apex becomes possible. It should be mentioned that, due to insufficient length of the roots, the sample size of 14 mm and 16mm sections did not meet the expectation for statistical analysis.

Inflammation was found in one third of the case group sections. It was usually mild to moderate and always chronic (Fig. 1). Seltzer and Bender have reported 37% pulpitis in their series, which is nearly similar to our study.

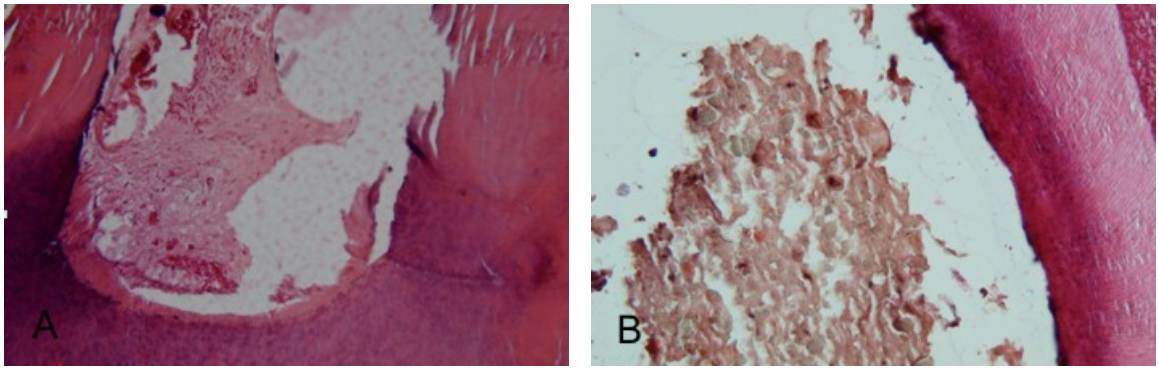

Fig. 4. A histologic view of partial (A; ×200) and complete (B; ×400) necrosis of the pulp tissue in the sections of case group.

They believe inflammatory lesions in the pulp, could be responses to toxic products entering through canal openings normally covered with bone and periodontal membrane, but now exposed to the oral fluids (4).

During evaluation of 2 mm level sections of both study and control groups, we found fibrosis in nearly half of sections (Fig. 2). This can be explained by high collagen fiber density in apical pulp (11). In 4 to 12 mm sections, there was a significant difference in prevalence of fibrosis between two groups; thus it could not be a fixation artifact, or a normal aging phenomenon in the pulps of periodontally affected teeth (3, 10).

Diffuse or multiple calcifications were more prevalent in our study group and can be considered the result of chronic hypoxia and cell death (4), not only a normal histologic finding or an aging phenomenon (3, 9) (Fig. 3).

Necrosis was an infrequent (less than 30%) finding and seen only in patients with deep pockets (middle and apical) (Fig. 4). Complete necrosis was seen only in the sections of roots adjacent to deep apical pockets. This finding shows that as long as the main canal is not seriously involved the entire pulp will not succumb despite that one or more lateral canals, or a number of dentinal tubules, are involved (1).

We showed that fibrosis and calcification (multiple and diffuse) are the most prevalent changes of the pulp; but inflammation and complete necrosis are infrequent and sometimes the result of apical involvement with periodontal disease. These are in agreement with some previous reports (1, 4); and in contrast with some others (3, 9). We found that in superficial (coronal) pockets, fibrosis is more prevalent than deep (apical) pockets. It can be explained that pulp reaction to superficial pockets is increased collagen production, and as the pocket deepens, the pulp reaction changes into dystrophic calcification and even complete necrosis as we showed.

Our data are in disagreement with a previous report which has claimed that there is no correlation between depth of periodontal pocket and pulp disease (5). In conclusion, advanced periodontal disease can affect pulp tissue. Fibrosis and calcification are the most prevalent changes. Necrosis of the pulp occurs infrequently and only in teeth with deep apical pockets. Pulp changes can jeopardize root canal therapy of these teeth. Careful consideration of diagnostic and treatment planning in patients with endodontic-periodontal involvement is recommended.

Conflict of interests

We have no conflict of interests.

Acknowledgment

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