

Pulpal changes associated with advanced periodontal disease: A histopathological study

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Abstract

Introduction: Over the past century, the dental literature has consistently reflected a controversy related to the effect of periodontal disease on the dental pulp. Nonetheless, practitioners are of the opinion that teeth having deep periodontal pockets show variable pulpal response, which may necessitate root canal treatment. Thus, this study aimed to evaluate the changes in pulp due to advanced periodontal disease.

Materials and Methods: Forty caries-free teeth affected with severe periodontitis were collected from patients aged between 18 and 55 years. The collected teeth were stored in formalin for 24 h and were then decalcified and examined histologically after staining with hematoxylin and eosin to note the changes that occurred in pulp.

Results: Pulpal calcification (52.62%) and partial necrosis of pulp (52.62%) were found to be the most common findings. Inflammation, which was found in 47.38% of the cases, ranged from mild to severe in most sections and was always chronic. Pulp with complete necrosis was seen in 26.32% of cases. Fibrosis and pulpal edema were seen in 36.84% of cases.

Conclusion: In the presence of moderate to severe chronic periodontitis, degenerative changes such as inflammation, fibrosis, edema, calcification and necrosis were observed to variable degree.

Keywords: Endo-perio lesion, dental pulp, histopathological study

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INTRODUCTION

The pulp and periodontal tissues share close embryologic, anatomic and functional interrelationship.^[1] Although communication pathways of the developmental origin, pathological origin and iatrogenic origin have been suggested, the most primitive and demonstrable relationship between the two tissues is through 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 consensus that pulpal disease can initiate or perpetuate periodontal disease through the apical foramen. However, periodontal disease causing pulpal disease remains a topic of debate. It has been suggested in the past that since inflammation follows venous drainage and venous blood flows outward from pulp into periodontium, periodontal disease cannot affect pulp.^[3] However, later studies demonstrated that teeth affected with chronic periodontitis showed pathologic changes in pulp in the form of inflammatory alterations,

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localized necrosis, calcification, root resorption and deposition of secondary dentin. These changes occurred because of spread of noxious inflammatory substances in a reverse direction through the lateral and accessory canals.^[4] Czarnecki and Schilder^[5] and Torabinejad and Kiger^[6] in their study disapproved this correlation between periodontal pathology and changes in pulp. The variation of opinions is due to differences in periodontal diagnostic criteria, difficulties in pulpal tissue fixation or lack of clear histologic criteria for definition of observations. With this regard, the study was undertaken to address the controversy surrounding the relationship between periodontal disease and pulpal tissue changes. To examine the degree to which pulpal pathosis is associated with periodontal disease, we evaluated the pulp of teeth affected by moderate to severe chronic periodontitis.

Objective of the study

The aim of this study was to evaluate the histopathological changes in pulp due to moderate to severe chronic periodontitis.

MATERIALS AND METHODS

The present study was carried out in the Department of Periodontics, V. S. Dental College and Hospital, Bengaluru, and was approved by the Institutional Ethics Committee. Patients were included in the study after signing informed consent form. Forty human teeth affected with moderate to severe chronic periodontitis were extracted from patients with age range of 18–55 years. Chronic periodontitis was diagnosed based on the criteria of the American Academy of Periodontology (1999).

Teeth were extracted in the Department of Oral and Maxillofacial Surgery of the same institution. Before extraction, clinical examination was carried out and radiographs were taken as needed. Patients providing teeth for the study were selected based on the following criteria:

Inclusion criteria

- Patients' age (whose teeth were extracted) between 18 and 55 years
- Grade III Mobile teeth
- Noncarious and nonattrited teeth
- Single or multirooted teeth
- Pocket probing depth of more than 6 mm.

Exclusion criteria

- Adolescents (11–15 years) and older patients (>55 years) were excluded

- Immunocompromised patients
- Restored teeth
- Teeth with developmental disorders (amelogenesis imperfecta, dentinogenesis imperfecta, dentin dysplasia, regional odontodysplasia, etc.)
- Teeth with a history of trauma, bruxism or clenching
- Orthodontically treated teeth
- Teeth having any history of periodontal treatment.

Using local anesthesia, the teeth were extracted as atraumatically as possible to prevent the histological sequel of traumatic extraction.

Processing of sample for histological examination

Immediately following extraction, the apical 2–3 mm of the roots were sectioned with a straight fissure bur and kept in 10% neutral buffered formalin solution for a week. The sectioning of apical 2–3 mm of roots provided a clear accessibility for the solution to fix the pulp.

After 7 days, teeth were decalcified with 6%–8% nitric acid in a microwave oven (LG, 700 watts, Model 1911HE) placed in an aluminum enclosure fitted with an exhaust fan and vent. A beaker containing 6%–8% nitric acid with teeth was placed in oven and irradiated for 5 cycles of 30 s each (at 1 h intervals) per day. This made the temperature of decalcifying solution to be around 41–43°C. The decalcifying solution was changed every day. Decalcification of teeth was completed by the end of 4 days.

The decalcified specimens were embedded in paraffin wax and sectioned longitudinally mesiodistally using a microtome (Leica RM2245, Germany) set at 5 µm thickness. The specimens were stained with hematoxylin and eosin stain. From the forty samples, two specimens were excluded as pulpal tissue was lost during processing and only 38 teeth were available for histological evaluation.

All specimens were examined under compound light microscope (model Magnus MLX) using magnification of ×10, ×40 and ×100 for inflammatory and degenerative changes (fibrosis, calcification and necrosis). The relative degree of inflammation was graded as mild, moderate and severe. Fibrosis was defined as an increased fibroblast and collagen fiber concentration. It was noted as present or absent. Edema was defined as accumulation of interstitial fluids in pulp and its presence or absence was recorded.^[7] Pulpal calcification was also noted. Necrotic specimens were categorized by whether the necrosis was partial or complete. All the parameters were checked and commented upon by a single experienced oral pathologist.

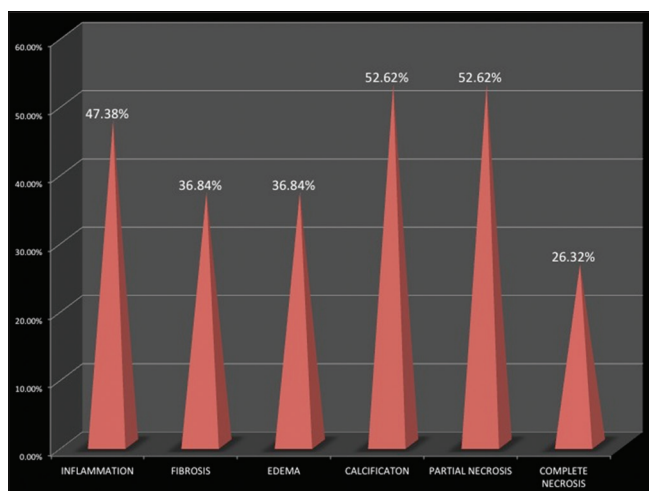
RESULTS

The mean age of participants in the study was 36.5 years. Histological sections were evaluated by microscopy, regardless of clinical results. Several microscopic sections indicated that the pulp could range from intact to necrotic in different sections of the same tooth [Table 1 and Graph 1].

- Pulpal calcification (52.62%) and partial necrosis of pulp (52.62%) were found to be the most common finding [Figures 1 and 2]
- Inflammation, which was found in 47.38% of the cases, ranged from mild to severe in most sections and was always chronic [Figure 3]
- Pulp with complete necrosis was seen in 26.32% of cases [Figure 4]
- Fibrosis [Figure 5] and pulpal edema was seen in 36.84% of cases.

Table 1: Percentage distribution of various pathologic changes in pulp

Parameter	Grade	n (%)
Inflammation	Mild	5 (13.16)
	Moderate	7 (18.42)
	Severe	6 (15.80)
	Total	18 (47.38)
Fibrosis	Present	14 (36.84)
	Absent	24 (63.16)
	Total	38 (100)
Edema	Present	14 (36.84)
	Absent	24 (63.16)
	Total	38 (100)
Pulpal calcification	Present	20 (52.62)
	Absent	18 (47.38)
	Total	38 (100)
Pulpal necrosis	Partial necrosis	20 (52.62)
	Complete necrosis	10 (26.32)
	Absent	8 (21.06)
	Total	38 (100)



Graph 1: Percentage distribution of various pathological changes in pulp

Table 2 shows samples of pulpal inflammation, which exhibited calcification and partial necrosis:

- Of 18 samples of inflammation, nine had calcification (50%) and 13 had partial necrosis (72.22%).

DISCUSSION

A lot of speculation seems to exist with regard to potential or actual relationship between pulp and periodontal tissues. Since the effect of periodontal disease on pulp is unclear and controversial, this study examined the changes in dental pulp in teeth with moderate to advanced periodontitis. Seltzer *et al.*^[8] stressed the importance of accessory and lateral canals in spreading the noxious substance from periodontal tissue to pulp and concluded that periodontal disease could lead to a greater incidence of inflammatory and degenerative changes pulpal changes. On the other hand, Mazur and Massler^[9] criticized this study because of the lack of controlled specimens and contradicted the relationship between the severity of periodontal disease and pulpal changes. Later, other authors who conducted human and animal studies suggested that degenerative pulpal changes develop independent of periodontal disease.^[5,6,10,11]



Figure 1: (a) Histopathological image showing pulpal calcification (H & E stain) under x10. (b) Histopathological image showing pulpal calcification (H & E stain) under x40

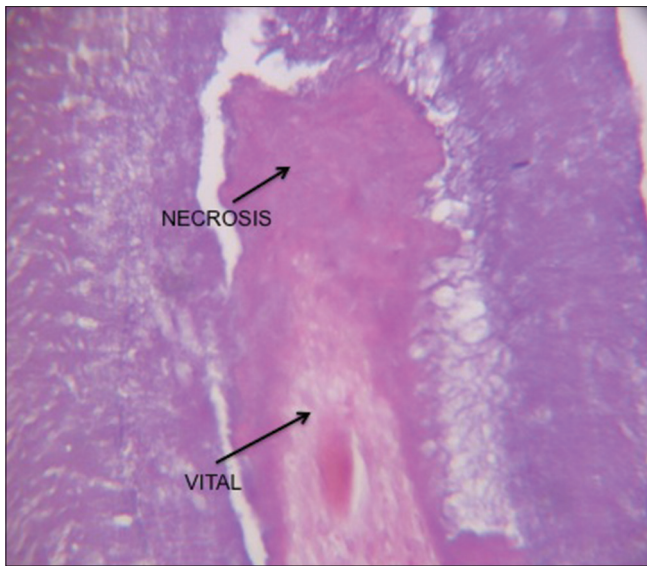


Figure 2: Histopathological image showing partial necrosis of pulp (H&E stain) under x10

Table 2: Pulpal inflammation samples showing calcification and partial necrosis

Inflammation	Number of cases	Pulpal calcification	Partial necrosis
Mild	5	3	3
Moderate	7	4	7
Severe	6	2	3
Total	18	9	13

This was an observational study. The most common pulpal changes reported in literature are inflammatory and degenerative changes in the form of fibrosis and calcifications and the results of our study are in agreement with earlier studies.^[7,12,13] These studies, however, employed cross-sectional specimens whereas we employed longitudinal sections. Although the chances of developing an artifact are more in longitudinal section, it allows to view the pulp entirely and also its relations to accessory and lateral canals. Instead of merely dropping the tooth in a formalin jar, the apical 1–2 mm of root was sectioned which provided a more prominent entry for formalin to fix the pulpal tissues as inadequate pulp fixation can result in artifacts.^[7] The microwave oven method of demineralization is more efficient as it reduces the time required for decalcification and is devoid of drawbacks related to the specimen preparation.

Table 2 shows that of 18 samples of inflammation, nine showed calcification and 13 showed partial necrosis. Inflammation probably occurred in response to toxic products from plaque bacteria colonizing the denuded root surface. The dynamics of pulp inflammation is not different to that of inflammation in the periapical and other tissues.

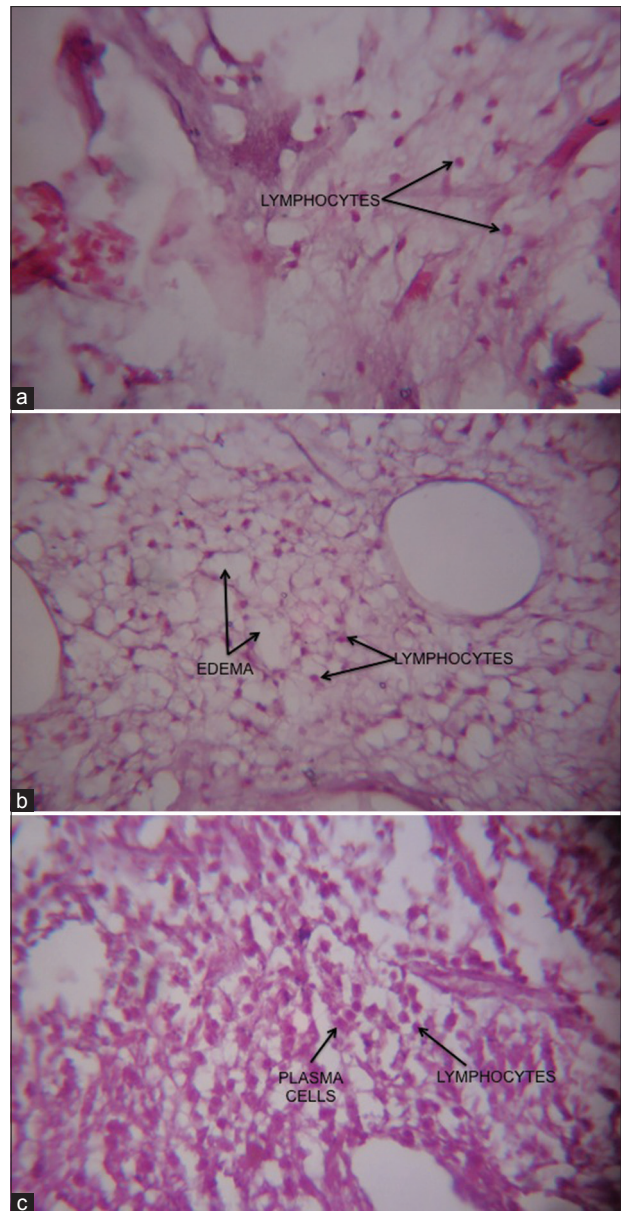


Figure 3: (a) Histopathological image showing mild inflammation of pulp (H&E stain) under x40. (b) Histopathological image showing moderate inflammation of pulp with edema (H&E stain) under x40. (c) Histopathological image showing severe inflammation of pulp (H&E stain) under x40

Depending on the severity and duration of the irritants, the pulp response ranges from reversible to irreversible pulpitis, then to partial necrosis which leads to complete necrosis. This may occur without pain.^[14] The dental pulp may also respond to irritation with a range of degenerative changes including fibrosis and calcification. A wide range of nonspecific mediators of inflammation such as histamine, bradykinin, serotonin, interleukins and arachidonic acid metabolites (PGE₂) are released in response to bacterial invasion and tissue injury. These cause vasodilatation and increased vascular permeability. The tissue becomes

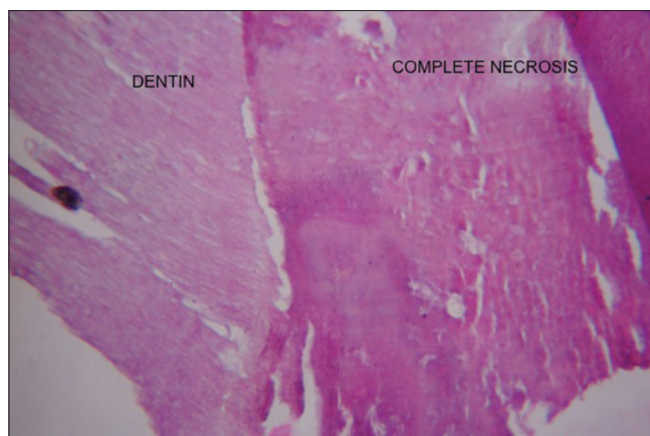


Figure 4: Histopathological image showing complete necrosis of pulp (H&E stain) under x40

edematous as a result of filtration of serum proteins and fluid from the vessels. The flow stasis causes an aggregation of red blood cells and an elevation of blood viscosity. It also produces tissue hypoxia or ischemia, which suppress cellular metabolism in the affected area of the pulp. This results in tissue necrosis. In addition, neutrophils in the area degenerate and release intracellular lysosomal enzymes to digest the surrounding tissue, forming necrotic tissue. As time progresses, necrotic pulp tissue becomes infected by oral microorganisms penetrating into the root canal system through exposed dentinal tubules and lateral/accessory canals.^[15] However, as long as the accessory canals are protected by sound cementum, necrosis usually does not occur. In addition, if the microvasculature of the apical foramen remains intact, the pulp will maintain its vitality.^[16] Thrombi in pulp blood vessels and collagen sheaths around vessel walls may become nidi for mineralization, resulting in pulp calcification. Pulp canal calcification is a protective mechanism. It may also be a normal physiologic response to aging and genetic predisposition may play a role.^[15,17]

This study is limited by the fact that no attempt was made to correlate the extent and severity of periodontal disease with pulpal changes. In the absence of age-matched controls, it is difficult to discuss whether changes observed are attributable to age alone. Degenerative changes, increased calcification and reduction in cellularity have been described with aging pulp. Over many decades, the enigma of periodontal disease affecting pulp has prevailed. More case-control studies with larger sample size are needed to resolve this controversy.

CONCLUSION

In the presence of moderate to severe chronic periodontitis, degenerative changes such as inflammation, fibrosis,



Figure 5: Histopathological image showing pulpal fibrosis (H&E stain) under x40

edema, calcification and pulpal necrosis were observed to a variable degree. It is not important what kind of degenerative changes develop in pulp, as the treatment modality remains the same for all of them. However, these pulpal changes may influence the outcome of root canal therapy.

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Conflicts of interest

There are no conflicts of interest.

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