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# *Original Research*

## Determination of pulpal changes in patients with periodontitis

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***ABSTRACT*:**

**Background:** Pulpal disease can initiate or perpetuate periodontal disease through the apical foramen. The Present study was conducted to determine pulpal changes in patients with periodontitis. **Materials & Methods:** The present study was conducted on fifty human teeth affected with moderate to severe chronic periodontitis extracted from both genders. All specimens were examined under electron microscope ×100 for inflammatory and degenerative changes such as fibrosis, calcification and necrosis. **Results:** Pulpal changes were inflammation which was mild in 10, moderate in 15 and severe in 20 teeth. Fibrosis was present in 23 and absent in 27 teeth, edema was present in 20 and absent in 30. Pulpal calcification was present in 21 and absent in 29, pulpal necrosis was partial in 22, complete in 16 and absent in 12 teeth. The difference was significant (P< 0.05). **Conclusion:** Authors found that chronic periodontitis leads to degenerative pulp changes such as inflammation, fibrosis, edema, calcification and pulpal necrosis.

**Key words:** calcification, edema, fibrosis

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## INTRODUCTION

Pulpal and periodontal problems are responsible for more than 50% of tooth mortality today. Dental biofilm on root surfaces following periodontal disease has the potential to induce pathologic changes in the pulp along the same pathways as an endodontic infection can affect the periodontium in the opposite direction.[1] Bacterial products and substances released by the inflammatory process in the periodontium may access to the pulp via exposed lateral canals, apical foramen or dental tubules. Reported outcomes show that involvement of the pulp function depends on the periodontal disease severity.[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.[3] 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. However, later studies demonstrated that teeth affected with chronic periodontitis showed pathologic changes in pulp in the form of inflammatory alterations 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. 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 observations.[4] The Present study was conducted to determine pulpal changes in patients with periodontitis.

## MATERIALS & METHODS

The present study was conducted in the department of Endodontics. It comprised of fifty human teeth affected with moderate to severe chronic periodontitis extracted from both genders. All patients were informed regarding

the study and written consent was obtained. Ethical approval was obtained from institutional ethical committee prior to the study.

The diagnosis of chronic periodontitis was based on the criteria of the American Academy of Periodontology. Before extraction, clinical examination was carried out and radiographs were taken as needed. 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 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 seconds each.

The decalcified specimens were embedded in paraffin wax and sectioned longitudinally mesiodistally using a microtome set at 5 μm thickness. The specimens were stained with hematoxylin and eosin stain. All specimens were examined under electron microscope ×100 for inflammatory and degenerative changes such as fibrosis, calcification and necrosis. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

## RESULTS

Table I, Graph I shows that pulpal changes were inflammation which was mild in 10, moderate in 15 and severe in 20 teeth. Fibrosis was present in 23 and absent in 27 teeth, edema was present in 20 and absent in 30.

**Table I Assessment of pathologic changes in pulp**

| Groups | Number | P value |
|---|---|---|
| **Inflammation** | | |
| **Mild** | 10 | 0.05 |
| **Moderate** | 15 | |
| **Severe** | 20 | |
| **Fibrosis** | | |
| **Present** | 23 | 0.91 |
| **Absent** | 27 | |
| **Edema** | | |
| **Present** | 20 | 0.04 |
| **Absent** | 30 | |

Table II, graph II shows that pulpal calcification was present in 21 and absent in 29, pulpal necrosis was partial in 22, complete in 16 and absent in 12 teeth. The difference was significant (P< 0.05).

## DISCUSSION

The possibility that periodontal disease might be related to, or cause, pulpal disease was reported by Colyer and Cahn in the 1920s.[5] The most demonstrable relationship between the two tissues 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. There is general agreement that pulpal disease can initiate or perpetuate periodontal disease; the opposite theory is controversial. Some have found a strong association between periodontal disease and inflammatory and degenerative pulp changes. Still others have not found this association.[6] The Present study was conducted to determine pulpal changes in patients with periodontitis.

**Graph I Assessment of pathologic changes in pulp**

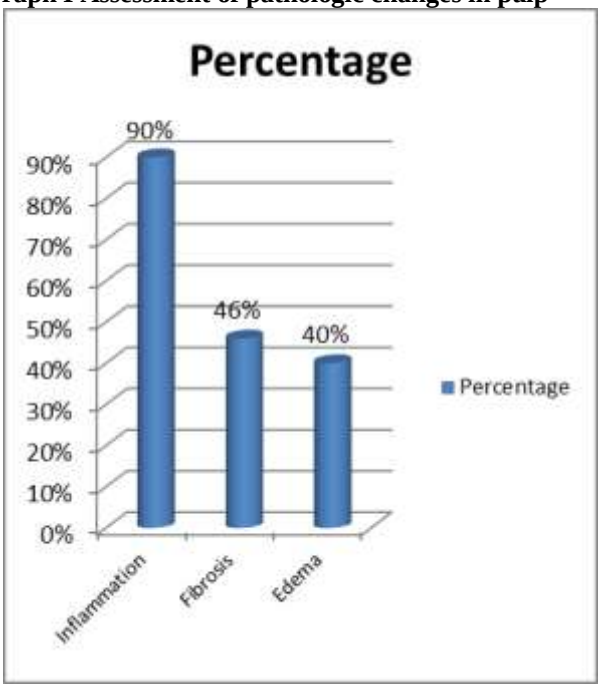
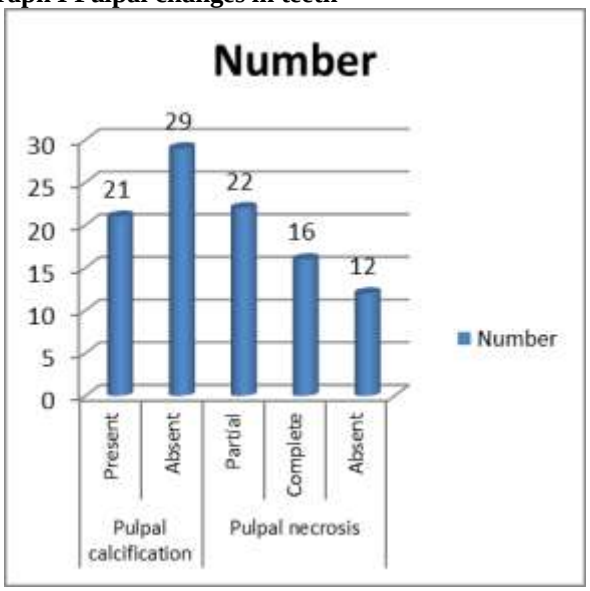



**Table II Pulpal changes in teeth**

| Parameters | Changes | Number | P value |
|---|---|---|---|
| **Pulpal calcification** | Present | 21 | 0.12 |
| | Absent | 29 | |
| **Pulpal necrosis** | Partial | 22 | 0.04 |
| | Complete | 16 | |
| | Absent | 12 | |

In present study, fifty human teeth affected with moderate to severe chronic periodontitis extracted from both genders. Gautam et al[7] found that 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. 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.

**Graph I Pulpal changes in teeth**




We found that pulpal changes were inflammation which was mild in 10, moderate in 15 and severe in 20 teeth. Fibrosis was present in 23 and absent in 27 teeth, edema was present in 20 and absent in 30. Pulpal calcification was present in 21 and absent in 29, pulpal necrosis was partial in 22, complete in 16 and absent in 12 teeth. Pressure from the mobility of the periodontally involved teeth may underlie the observed changes. The greatest reactions in the pulp to inflammatory or atrophic changes appear to be related to the presence of excessive numbers of accessory and lateral canals. Therefore, these canals seem to have an effect on the status of the pulp in periodontally involved teeth. The interference of the blood supply through the lateral canals along the root sides may mediate the observed inflammation and pulp necrosis. Toxic products entering through the lateral canals can also produce inflammatory responses in the pulp. Microorganisms present in the periodontal lesions may injure the pulp cells through their metabolic products.[8]

Seltzer et al[9] 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.

Fatemi et al[10] in their study twenty hopeless permanent teeth were extracted from systemically healthy adults because of moderate to advanced chronic periodontitis, with a bone loss of >6 mm and a mobility of grade 2 or 3. Upon extraction, the apical 2 to 3 mm of the roots were immediately sectioned. Four to five sections were mounted on each slide, and every third slide was stained with hematoxylin and eosin. The specimens were histologically processed and examined by an oral pathologist. Authors found that non-inflamed pulp, with partial or complete necrosis in some sections and several non-necrotic sections, was found in only 6.3% of teeth. Most teeth (58.3%) displayed edematous pulps. Slightly fibrotic pulps were seen in 52.1% of sections. Odontoblastic integrity was seen in 31.3% of teeth. Most teeth (77.1%) displayed no pulp stones. In 43.8% of teeth, the pulp vessels displayed dilatation.

## CONCLUSION

Authors found that chronic periodontitis leads to degenerative pulp changes such as inflammation, fibrosis edema, calcification and pulpal necrosis.

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