**ENDO-PERIO LESION: AN INTERDISCIPLINARY APPROACH**

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**ABSTRACT**

The relationship between pulpal and periodontal disease was first described by Simring and Goldberg in 1964. Since then, the term endo-perio lesions have been used to describe lesions due to inflammatory products found in varying degrees in both pulp and periodontal tissues. The pulp and periodontium have embryonic, anatomic and functional interrelationships. As the tooth matures, and the root is formed, three main avenues are created between pulp and periodontal ligament, i.e., dentinal tubules, apical foramen, lateral, and accessory canals. These are the pathways that may provide a means by which pathological agents pass between the pulp and periodontium, thereby creating the endo-perio lesion. Lateral canals play an important role in the spread of infection from the pulp to the periodontium. This paper reports the management of a 35-year-old patient with a combined endo-perio lesion.

Key words: Endo-Perio lesion; Pulp necrosis; Periapical abscess; Secondary periodontitis.

**Introduction**

The relationship between pulpal and periodontal disease was first described by Simring and Goldberg in 1964. Since then, the term endo-perio lesions have been used to describe lesions due to inflammatory products found in varying degrees in both pulp and periodontal tissues. The pulp and periodontium have embryonic, anatomic and functional interrelationships. They are ectomesenchymal in origin. The pulp originates from the dental papilla and periodontal ligament from the dental follicle. They are separated by the formation of the tooth bud. As the tooth matures, and the root is formed, three main avenues are created between pulp and periodontal ligament, i.e., (a) dentinal tubules, (b) lateral and accessory canals, (c) apical foramen. These are the pathways that may provide a means by which pathological agents pass between the pulp and periodontium, thereby creating the periendo lesion.

The pulpal inflammation and necrosis are initiated by dental caries, restorative procedures, trauma, and chemical irritation. The oral bacteria and their components can arrive at the pulp via dental microtubules. Often in these cases, some typical signs of periodontal involvement can be observed. Clinically symptoms such as thermal hypersensitivity, pain on percussion and radiographically a slight enlargement of the periapical periodontal space can be detected. In such cases if pulp does not undergoes necrosis, it reacts by producing reparative dentin formation. After removal of etiological factors, the symptoms and signs of periapical radiolucencies should disappear. Pulp necrosis is always associated with periapical response and according to studies it is of microbiological in nature. After the initial phase where the pathological phenomenon expands from the apical part of the pulp to the periapical tissue, the second phase involves either of the possible two ways: (1) the formation of an abscess or (2) establishment of the balance between host response and bacterial challenge. In case of abscess formation, inflammation spreads through the periodontium. The abscess may drain through a fistula or via the periodontal ligament, with ligament and adjacent bone destruction, which can involve the entire root length.

When balance establishes between the host response and bacterial challenge, there will be formation of a richly vascularized granulation tissue infiltrated by different inflammatory cells. The granulation tissue may increase in size or become cystic lesion overtime. The radiographic aspect of the periodontal response to pulp necrosis consists of an area of radiolucencies localized in the proximity of the apical foramen or sometimes the accessory lateral canals. The lateral canals contain vessels and connective tissue. Since the width of lateral canals is reduced by continuous deposition of dentin and root cementum, the apical foramen remains the main pathway through which the inflammation can spread to the periodontal ligament in permanent teeth.

In periapical abscess, the lesion may perforate the cortical bone close to the apex, elevate the peristemeum and overlying soft tissues, and drain into the gingival sulcus. Meanwhile, the drainage may also tract along the periodontal ligament and into the gingival sulcus or in multirooted teeth into the furcation. The endo-perio lesion presents challenge to the clinicians as far as diagnosis and prognosis of involved teeth are concerned. Proper diagnosis of the lesion is essential for appropriate treatment This paper reports the management of a 35-year-old patient with a combined endo-perio lesion.

**Case Report**

A 35-year-old male patient reported to the Department of Periodontics, HKE Dental Hospital, India, with a chief complaint of mild pain in the lower left back region of the jaw for the last fifteen days. There was no history of trauma in the region. On intraoral examination, there were stains and calculus covering the crown of lower and upper anterior teeth lingual surfaces as well as in the upper first molar region. On probing there was a pocket of 9mm at mesio-buccal aspect of 36, with furcation involvement. Except the mandibular Left permanent first molar, none of the tooth revealed a probing depth of more than 5mm. On palpation, 36 was neither mobile nor tender on percussion. The caries associated with that 36 was obturated. An Intraoral Periapical Radiograph with bisecting angle technique was taken in that region, which revealed a radiolucent area at the mesial as well as distal aspect of the mesial root of left first molar involving the furcation area. Also, the tooth was tested for pulp vitality, where we found that to be non-vital.
The clinical examination showed a pocket depth of 9mm. On radiological examination a radiolucent area was seen around the mesial root of 36 without any caries. The initial diagnosis was periodontal lesion. But after the pulp vitality testing we found that pulp was non-vital hence the second hypotheses were necrotic pulp of 36 with a secondary involvement of periodontal ligament space through lateral canals. In spite of unclear nature of this tooth, lesion was treated with scaling and polishing, followed by root canal treatment. Patient was recalled after three months for revaluation of periodontal status of 36. The probing depth had reduced from 9mm to 5mm (Figure 1). However, still the 5mm of probing depth was persistent on the mesio-buccal surface of 36, hence, through sub gingival scaling and root planning was performed and patient was recalled after three months. At the 6th month visit the patient showed a probing depth of 3mm at mesio-buccal aspect of 36 with subsiding of all symptoms of the patients (Figure 2).

**Discussion**

It was a case of primary endodontic lesion with a secondary involvement of periodontal tissue. The case was reported six months back with mild pain in the 36 region, associated with pocket of 9mm. When radiograph was taken, it showed radiolucency in the mesial as well as distal aspect of the mesial root involving the furcation. Tooth was tested for vitality of the pulp where it was found to be negative. Hence, the clinical examination was very similar to periodontal lesion involving the first molar, it was thought to be periodontitis. But as the radiograph and pulp vitality was done, it was clearly showing an endodontic lesion. Hence, scaling was performed, and endodontic treatment was completed. Then the patient was recalled after 3 months for reevaluation. During the 3rd month evaluation there was a persistent pocket of 5mm in the mesial aspect of 36, even after root canal treatment. This may be due to secondary involvement of periodontal tissue after the endodontic treatment.

The lateral canals play an important role in the spread of the infection from the pulp to periodontal tissues. There are many studies where they have shown that accessory canals in the root are more common in molars. Patient canals are especially common in furcation areas, where they have been found in between 20% to 60% of examined teeth. These accessory canals are one of the causes for periodontal involvement in endodontically involved tooth. Since there was a persistent pocket sub gingival scaling and root planning was performed, and patient is recalled after third month.

During the 6th month visit of the patient there were no symptoms of any pain or discomfort. On examination, the pocket was reduced from 5mm to 3mm and on radiograph we could see some amount of bone fill in the apical portion of initial defect seen around the mesial root. Hence, it subsided with a non-surgical treatment by scaling and root planning. In primary endodontic disease with secondary periodontal involvement should first be treated with endodontic therapy. Treatment results should be evaluated in 2-3 months and only then should periodontal treatment be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition. In this case, we observed a reduction in probing depth from 9mm to 3mm in six months with a radiographic bone fill after six months.

**Conclusion**

The presentation of the case with mild pain associated with a deep pocket of 9mm around a non-caries tooth 36, with a radiolucent area around the mesial root of 36 was typically pointing towards periodontal lesion. But the pulp vitality test, which showed the non-vital nature of the tooth, was a pivoting founding towards the endodontic nature of the lesion. So following endodontic treatment of the tooth the periodontal lesion did reduce, but did not subside completely. This showed a secondary involvement of the periodontal lesion along with endodontic origin. Hence, it was treated with sub-gingival scaling and root planning which helped in reducing the probing depth to 3mm. Hence, this case report demonstrates the nature of the periodontal lesion, as a secondary involvement to an originally endodontic lesion involving the tooth. It demonstrates that the lateral canal can be one of important criteria deciding for the periodontal involvement of periodontal tissue in endodontic lesion, and in turn on prognosis of the tooth.

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