Endodontic–Periodontal Lesion:  
A Case Report

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Abstract  The report case of 36 years old male was referred to have an endodontic treatment of #36. Examination showed endodontic-periodontal lesion which had large destruction of bone around tooth including furcation involvement. It was diagnosed as a non-vital tooth from both endodontic and periodontal origin. Treatment was first done with conservative endodontic method by using calcium hydroxide to eliminate the microorganism. The periodic recall were performed until there were the sign of bone regeneration then filled root canals. Following that, the patient was referred to have periodontal treatment with periodontist. After treatment the results of re-evaluation periods were pleasant, the healing of periapical lesion were presented.

Key words: endodontic-periodontal lesion, endodontic treatment

Introduction

Lesions of the periodontal ligament and adjacent alveolar bone may originate form infections of the periodontium or tissues of the dental pulp. Therefore, the pathologic processes of the pulp can cause periodontal involvement, and similarly periodontal disease can cause alterations in pulp tissues.

The periodontium communicates with pulp tissues through many channels or pathways. Studies of human teeth have shown that lateral and accessory canals and their foramina are present in great
numbers, especially in the bifurcation and trifurcation regions of molars\textsuperscript{1,2}. There is a sample evidence to indicate that infection of the pulp can potentially communicate with the periodontium at location other than the apex of the tooth. However, it had been clearly shown that when apical granulomas that develop from necrotic pulps are extensive, the infected granulomatous tissue can be present along the lateral aspects of roots. This may cause extensive resorption of adjacent alveolar bone, involving the alveolar crest 1. The following anatomical entities or pathways have also been mentioned in the literature as possible causes of periodontally derived endodontic lesions: lingual grooves, root/tooth fracture, cemental agenesis/hypoplasia, root anomalies, intermediate bifurcation ridge, fibrinous communications, and trauma-induced root resorption\textsuperscript{3}. For example, in the absence of an intact enamel or cementum covering, the pulp may be considered exposed to the oral environment via the gingival sulcus or periodontal pocket. Experimental studies demonstrate that soluble material from bacterial plaque applied to dentin can cause pulpal inflammation\textsuperscript{4}. Noyes et al. report that 5\% to 10\% of human teeth display enamel-cemental dysfunction, exposing cervical dentinal tubules to the gingival sulcus\textsuperscript{5}. In addition, dentinal tubules may be exposed in developmental anomalies such as lingual grooves, enamel projections, and other conditions\textsuperscript{3,6}.

In general, pulpal infection has the potential to initiate inflammatory changes in the alveolus at both apical and non-apical area of teeth. On the other hand, periodontal disease is generally a slow process that can have a gradual atrophic effect on the dental pulp\textsuperscript{7}. However both ways can induce inflammatory response, in some cases developing and abscess. An abscess may arise every time that balance between microorganisms and the host breaks down. The radiographic aspect of the periodontal response to the pulp necrosis and the bacterial contamination consists of an area of radiolucency generally localized in the proximity of the apical foramen or sometimes the accessory lateral endodontic canals\textsuperscript{8}.

An endodontic-periodontal lesion, or endo-perio lesion for short, is a lesion that occurs as the result of the interaction between disease of endodontic and periodontal origin, on the same tooth, irrespective of the sequence in which they occur\textsuperscript{9}. A lesion can be of primary endodontic or primary periodontal origin, or stem from separate origins (meaning that both the endodontic lesion and the marginal periodontal lesion have developed independently)\textsuperscript{10}.

According to Simon et al.\textsuperscript{11}, classification of endo-perio lesions is based on aetiological factors. They separated the lesions involving both periodontal and pulpal tissues into the following groups:

- primary endodontic lesions with secondary periodontal involvement,
- primary periodontal lesions with secondary endodontic involvement, and,
- true combined lesions.

In most endo-perio cases, it will be possible to differentiate between lesions of endodontic and periodontal origin; but in some cases this may not be entirely clear. It is important to define whether the aetiology is endodontic, periodontal, or that combined lesions, because it will help operator to choose the most appropriate treatment modalities for his or her patients\textsuperscript{12}. For instance, if the origin of the lesion is truly endodontic, a proper endodontic treatment will lead to a complete healing of the periodontal tissues.

**Case report**

A 36-year-old patient was referred to dental department, Priest hospital for consultation and endodontic treatment of tooth 36. The patient’s dental history indicated that the 36 was restored many years ago. Last year, the tooth had abscessed and the pulp was extirpated, dressed with Ledemix
paste and temporalised with a Glass Ionomer restora-
tion. The patient had been asymptomatic during this period. One year later, the patient experienced a spontaneous dull pain and abscess started to develop and resolved many times, as it was treated with systemic antibiotics.

Clinical examination revealed the fractured large occluso-buccal glass ionomer restoration in the 36. Coronal leakage could be detected around the restorations. There was swelling at the apical and mesial marginal region of the tooth, gingival inflammation, and attachment loss. A type II furcation involvement was detected. The periodontal probing depths were about 8 mm on both buccal and lingual side of the tooth. The tooth was tender to percussion and palpation, and had a sinus track opening at furcation area of buccal ginvia. The tooth displayed second degree mobility. No pulp sensibility tests were carried out as the 36 had previously been pulp extirpated. It was also noted that there was a premature contact affecting this tooth.

Radiographic examination revealed large radiolucency at furcation area, of approximately 4 mm in diameter, there was a periapical radiolucency around both mesial and distal roots of the tooth.

A diagnosis was made that an endodontic-periodontal lesion which the true combine type was associated with the 36 that already had been pulp extirpated. The tooth also displayed acute periodontitis and furcation involvement.

Conventional root canal treatment was initiated using a crown-down instrumentation technique with combined rotary (Protaper files, Dentsply Maillefer, USA) and hand files (K-files; Halas Dental limited, USA). The root canals were biomechanically cleaned and irrigated with Milton’s solution (1% NaOCl) and EDTA. The prepared root canals were then dressed with Ledermix paste. The access cavities were filled with Cavit (ESPE, Dental AG Norristown, PA, USA) and Fuji IX (Kerr Corp, CA, USA). Occlusal adjustment was performed by selective grinding of cusps.

At the second appointment, fourteen days later, patient reported that the pain was relieved, and gingival inflammation was significant reduced. But the tooth was still slightly tender to percussion and palpation. At this appointment the tooth was dressed with calcium hydroxide paste (Calypxyl, OCO Praparate GMBH, Dirmstein, Germany). The patient was scheduled to have the calcium hydroxide dressing changed every three months.

Unfortunately, the next appointment was made in four months later. At this appointment, clinical examination with the probe indicated some furcation healing and an absence of bleeding. Radiograph examination showed regeneration of bone, especially around the distal root and mesial aspect of mesial root.

As a consequence, root canal obturation was performed. A lateral condensation technique with gutta percha and AH26 sealer (Densply, Detrey, Konstanz, GMBH, Germany) was used. Following that, the patient was referred to have periodontal treatment with periodontist. Periodontal treatment including radicular scaling and debridement of tooth 36 was performed. The patient have the recall appointments in every three months.

Discussion

Communication allowing the spread of bacteria between pulpal and periodontal tissues is possible via various pathways. Pulpal disease may cause periodontal changes through lateral canals, accessory canals, accessory and apical foramina, and gingival extension of endodontic lesions. Periodontal disease may cause pulpal changes through communications between the pulp and the root surface via internal and accessory canals, accessory foramina and dentinal tubules where cementum have been lost.

Both endodontic and periodontal disease are caused by microbial infection. When classifying microorganisms by morphological criteria with
interference microscopy, no significant difference in microbial flora was found between infected root canals and adjacent periodontal pockets\textsuperscript{16}. It has also been noted that the endodontic flora can appear in clusters of mixed bacterial content similar to the arrangement seen in subgingival plaque\textsuperscript{17}. This was confirmed by more recent studies that suggested the microorganisms of an acute dentoalveolar abscess are usually polymicrobial, similar to those associated with periodontal disease. Moreover, exacerbations of periapical lesions appear to be linked to the black-pigmented, gram-negative anaerobic rods in the root canal system\textsuperscript{18,1,20}. However the microorganisms in necrotic pulps are not as complex as those in deep periodontal pockets\textsuperscript{19,20,21}.

There are some different characteristics between endodontic and periodontal lesions. In contrast to superficial periodontal lesions extending from gingivitis, pulpal disease have the potential to initiate inflammatory changes deep in the alveolus at both apical and non-apical locations of involved teeth.

So, the diagnosis of periodontal lesion can be reinforced by the typical morphology of the defect, the wide extension of the probing site along the buccal and distal aspect of the tooth, the absence of deep caries, and a positive response of pulp sensibility test and cavity test. On the other hand, periapical acute manifestations, due to root canal infections, can increase in size, causing a progressive destruction of the periodontal apparatus. If this lesion evolves into an abscess, the suppurative exudates try to find drainage to the gingival sulcus (or periodontal pocket), generally through the periodontal ligament or perforating the bony wall and periodontal ligament. This produces the typical narrow and deep probing site can be detected\textsuperscript{8}.

Radiographically, periodontal lesions generally show angular bone loss, extending roam the crown toward the root apex. In contrast, the

\begin{figure}[h]
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\includegraphics[width=\textwidth]{Fig_1}
\caption{Preoperative radiograph, showing the severe bone loss around the mesial root and in the furcation area.}
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\begin{figure}[h]
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\includegraphics[width=\textwidth]{Fig_2}
\caption{Radiograph showing the intervisit calcium hydroxide medication.}
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\begin{figure}[h]
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\includegraphics[width=\textwidth]{Fig_3}
\caption{Radiograph indicate repairing of bony lesion could be observed at the time of obturation (4 months later).}
\end{figure}
radiographic features of the periodontal response to the pulp necrosis and bacterial contamination consists of an area of radiolucency generally localized in the proximity of the apical foramen or sometimes the accessory lateral root canals.

The diagnosis of periodontal lesions associated with pulpal diseases maybe relatively simple if a patient has been monitored over a period of time and records are available. The diagnosis becomes more difficult when a complete history is unavailable. In general, it is easier to determine the origin of the lesion when a pulp sensibility test is obtained, because the test results usually will rule out an endodontic etiology. However, pulp test may not always be reliable. So location and extent of pockets, probing depth, a furcation invasions are also essential for the differential diagnosis.

The following table (adapted from Torabinejad and Trope, 1996 sited in Abbott 1998) outlines the common general findings when examining endoperio lesions.

It can be stated that in the vast majority of the lesions involving the pulpo-periodontal complex, the bacterial aetiology dictates both the clinical course of the disease and the subsequent treatment plan. Therefore, the primary goal of treatment must be to rid the patient of the infection.

In this particular case, the use of a biocompatible medicament was essential to promote periodontal healing. Ledemix paste was the first medicament that was selected. It has been shown to be effective as an anti-bacterial agent within the root canal and its anti-inflammatory action can reduce patient’s symptoms by decreasing the periodontal and periapical inflammation. After the symptoms were relieved, calcium hydroxide was used in the canals. It is also a very effective anti-bacterial agent within the root canal and it has been shown to stimulate hard tissue repair. Calcium hydroxide paste has a pH of 11-12 and low solubility in water. It releases calcium and hydroxyl ions. It can provide bactericidal activity by various mechanisms; calcium combines with carbon dioxide, the later being obligatory for anaerobic activity, forming calcium carbonate; the hydroxyl ions liberated produce an alkaline environment, which is inappropriate for survival of substrate for bacterial lodged within the canal and allows hydrolysis of bacterial lipopolysaccharied, which is responsible for inflammation, pain and bone resorption. The use of non-toxic intra-canal therapeutic medicaments is essential to destroy bacteria and to encourage tissue healing.

Following calcium hydroxide medicament, with the healing of bone demonstrated, the root canal treatment was performed and the healing

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<tr>
<th>Sign</th>
<th>Origin of Lesion</th>
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<tr>
<td></td>
<td>Endodontic</td>
</tr>
<tr>
<td>Localised to tooth</td>
<td>+</td>
</tr>
<tr>
<td>Extensive caries / restoration</td>
<td>+</td>
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<tr>
<td>Pulp sensibility tests</td>
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<td>Probing defect Narrow Wide Wide</td>
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<tr>
<td>Crestal bone less</td>
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<td>Percussion</td>
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<td>Palpation</td>
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potential of the alveolar bone then was allowed to continue.

However periodontal treatment will essential for continuing management of this tooth. Root scaling and polishing aids in reducing the number of anaerobic gram-negative bacteria present in the periodontal pocket, thus decontaminating the radicular surface. The use of specific curettes in the furcation region may have optimized treatment results. In particular, investigators have noted the importance of completing the indicated endodontic treatment first and then re-evaluating the periodontal status 2 to 3 months following completion\textsuperscript{32,33}. This allows sufficient time for initial healing events to present a more accurate picture of the status of periodontal tissue prior to developing the periodontal treatment plan.

**Conclusion**

The endodontic-periodontal lesion should be differentiated to endodontic origin, periodontic origin or true combined lesion by some different characteristics. This will help the operator to choose the proper treatment for the patient.

**References**


