

ENDODONTIC MANAGEMENT OF THE ENDODONTIC-PERIODONTAL LESION

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Abstract

Occasionally periradicular lesions of endodontic origin may be radiographically indistinguishable from periodontal disease. Infected pulpal tissue and microbial by-products may move through accessory and furcal canals and cause loss of attachment in those areas. Accurate diagnosis may be particularly difficult when a sinus tract originating from the endodontic lesion drains along the periodontal ligament space, giving the appearance of periodontal disease. Thorough diagnostic testing to confirm pulp necrosis or periodontal disease becomes critical when attempting to diagnose the specific disease entity accurately and then deliver suitable treatment. In both clinical cases presented in this paper, diagnosis of the etiology of the pathosis was more difficult since there was extensive deep probe depths in more than one site. However, successful healing was obtained after thorough disinfection and sealing of the root canal system.

The relationship between the pulp tooth and the attachment apparatus of a tooth has been widely documented (Simon et al, 1972; Paul B, Hutter JW, 1997; American Association of Endodontists newsletter, 2001). Most of the time periodontal inflammation due to pulp space toxins occurs in the apical region and thus can readily be distinguished from a periodontal pocket (Figure 1).

However, occasionally necrotic infected tissue by-products move through accessory or furcal canals, producing inflammation that is indistinguishable from periodontal disease. The amount of tissue destruction is directly correlated with the total microbial content in the root canal system (Bystrom et al, 1987) and to the length of time these tissues are exposed to the infecting organism (Korzen et al, 1974). Differential diagnosis is particularly difficult when a sinus tract originating from the endodontic lesion may drain along the periodontal ligament, giving the appearance of periodontal breakdown (Simring M, Goldberg M, 1964; Seltzer et al, 1967). Yamasaki et al (1994) have reported that periradicular lesions may initially expand horizontally through cancellous bone and then proceed

vertically. Analyzing a series of retrospective studies, Blomlof et al (1993) concluded that endodontic infection promotes periodontal pocket formation and should be regarded as a risk factor in periodontitis progression. Therefore, a primary endodontic lesion draining through the attachment apparatus should be treated initially by endodontic therapy (Zehnder M, Hasselgren G, 2002). This must be confirmed by accurate diagnostic tests to confirm pulp necrosis and diagnostic probing (usually a precipitous drop in probe depth is detected around a tooth) (Harrington, 1979).

Periodontal health should be reassessed only after one to two months (Paul B, Hutter JW, 1997) since aggressive removal of periodontal ligament and underlying cementum during interim endodontic therapy adversely affects periodontal healing (Blomlof et al, 1993). As such, scaling should not be done in these cases. If a vertical fracture has been ruled out, and the standard of endodontic care rendered well done, healing should be expected in the vast majority of cases (Figures 2a-e). In both cases presented, diagnosis of the etiology of the pathosis was more difficult since there was extensive deep probing depths in more than one site. However, successful healing was obtained after thorough disinfection and sealing of the root canals.

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Case report one

A 45-year-old female patient presented with swelling of the soft tissues distal to tooth 47. Probe depths were within normal limits in the area except for 13mm both on the distal and the lingual aspect. No mobility was determined and the tooth was non responsive to a cold test (Endo Ice, Hygenic). All other teeth in the area tested within normal limits to thermal challenge. Pus drained

through the sulcus and no indication of fracture was detected. Radiographic examination demonstrated severe bone loss around the distal root, the furcation area and the apex of the mesial root of tooth 47 (Figure 3a). The C-shaped root canal was instrumented to completion using nickel titanium rotary instruments, irrigation was performed with sodium hypochlorite 5.25% and REDTA (Roth Intl) (Figure 3b), all canals were dried and an interappointment dressing with calcium hydroxide was placed.

Two weeks later, soft tissues looked normal, there was no pus draining through the sulcus and the tooth was asymptomatic. After rubber dam isolation, the calcium hydroxide was removed from the canals using EDTA and sodium hypochlorite 5.25%, and the canals were dried and filled with lateral condensation of gutta percha and sealer. An IRM (Dentsply) temporary restoration was placed in the access cavity and the patient was referred back to her dentist for a final restoration.

Despite numerous reminders, the patient did not return for reassessment until eight months later. At this visit there was no permanent restoration in the tooth; however, probing depths were 3mm all around the tooth and soft tissues looked normal. The tooth was asymptomatic to percussion and radiographic examination revealed a dramatic regeneration of the periradicular tissues (Figure 3c). After removing the remaining IRM, there was no evidence of fracture or decay in the access cavity, which was disinfected with sodium hypochlorite 5.25%, dried and filled with a cotton pellet and IRM. The patient was sent directly to her dentist who placed a ceramic onlay on the tooth. The patient returned six years and six months after the placement of the permanent restoration and tooth 47 was asymptomatic, probing depths were still 3mm all around the tooth and soft tissues appeared normal. Radiographic examination revealed complete regeneration of the periradicular tissues (Figure 3d).

Case report two

A 58-year-old male presented with swelling on the buccal mucosa of tooth 21, which was restored with porcelain fused to metal crown. Probing depths were 16mm over all the buccal and the mesial surfaces of the tooth. Pus was draining through the sulcus, however the tooth was not mobile. The rest of the dentition demonstrated a stable periodontal condition.

Radiographic examination revealed severe bone loss on the mesial, apical and distal surface (Figures 4a and 4b) and the tooth was non responsive to a cold test (applied to exposed dentin on the lingual surface)(Endo Ice). A cavity test was performed but no reaction was reported by the patient upon entrance into the dentin. The root canal was instrumented to completion using nickel titanium rotary instrumentation with sodium hypochlorite 5.25% and REDTA as irrigants. Aqueous

chlorhexidine 2% (Alphadental Products) was placed in the canal and activated with a passive ultrasonic tip for one minute. As the canal could not be dried, calcium hydroxide was placed as an interappointment dressing and a temporary filling was placed in the access cavity (Provisit).



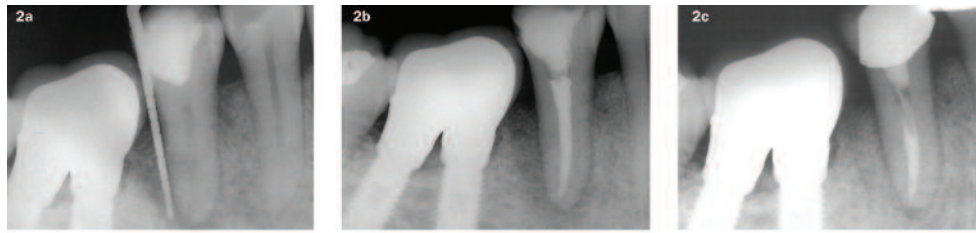
Figure 1: Apical periodontitis on the apical region of tooth 21.

When the patient returned after seven days, soft tissues appeared normal and the tooth was asymptomatic; no probing was attempted at this time. After isolation of the tooth and removal of the temporary filling, the calcium hydroxide was removed with copious irrigation of sodium hypochlorite 5.25%, REDTA and rotary instruments. The canal was dried and filled with lateral condensation of gutta percha followed by a down pack using the Buchanan pluggers (SybronEndo) and the Touch 'n Heat (Analytic Endodontics) (Figure 4c). A follow-up examination was done one year and two months after the initial appointment. Probe depths were 3mm on all aspects of the tooth, which had remained symptom-free since completion of the root canal. Radiographic examination revealed dramatic regeneration of the periradicular tissues (Figure 4d).

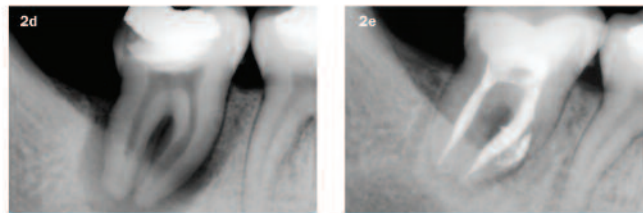
Discussion

In the preponderance of endodontic lesions, microflora is the etiologic vector that dictates the clinical course of the disease and therefore the treatment plan (Zehnder M, Hasselgren G, 2002). On occasion, a sinus tract originating from diseased apical tissues may drain alongside the periodontal ligament, giving the appearance of a periodontal pocket. After ruling out fracture as the etiology, careful examination with a periodontal probe should be done, not only at the site of the lesion but also in the rest of the mouth. In addition, a negative response to thermal challenge and lack of mobility of the tooth may indicate that the lesion is purely of endodontic origin. Thus root canal therapy should be performed and periodontal therapy avoided, or at least delayed, until one or two months after the root canal has been performed (Blomlof et al, 1993), and then only if the attachment apparatus does not seem to be improving. Follow-up examination is crucial when attempting to evaluate the prognosis of the treated tooth.

In both cases presented here, there were deep probe depths



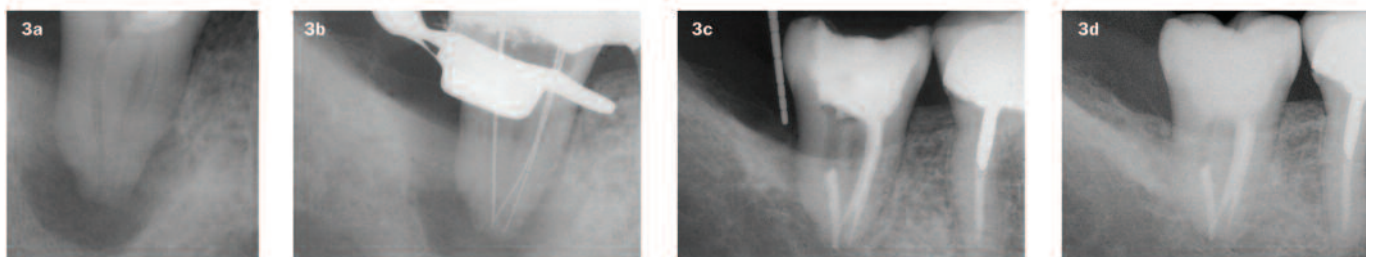
Figures 2a-e: (a) Probing depth 13mm on distal area of tooth 45; (b) Immediately after filling; (c) 10 months postoperatively, 3mm probing depth all around the tooth; (d) Preoperative radiograph of tooth 47 with extensive periradicular bone loss; (e) One year and eight months control X-ray film showing considerable bone apposition, probing depths were of no more than 3mm all around the tooth.



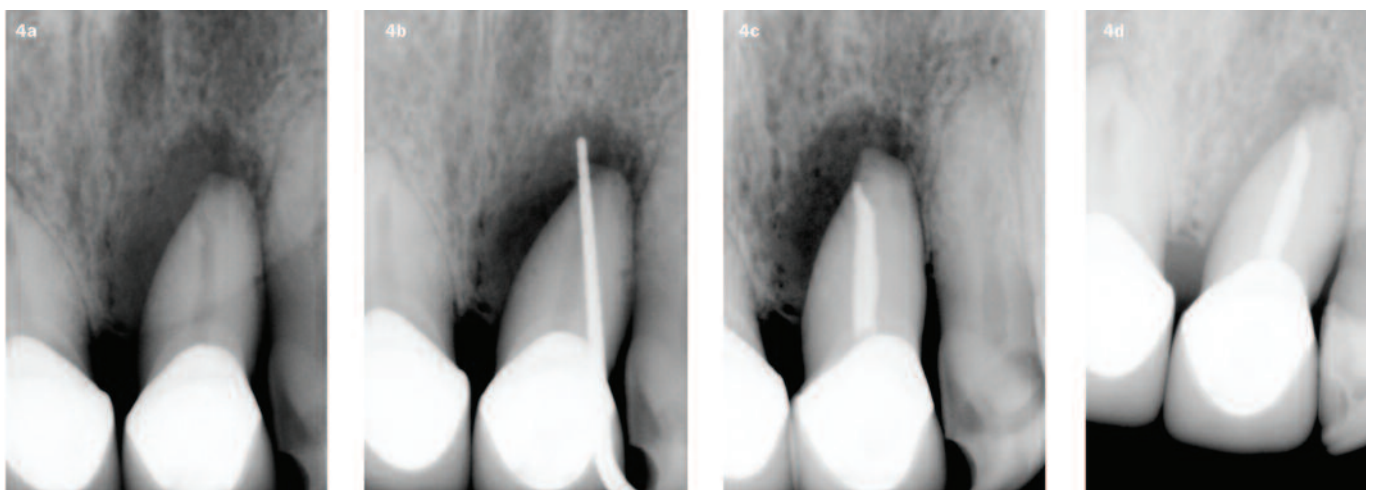
along more than one surface of the tooth. Radiographically there was extensive bone loss, however successful disinfection and filling of the root canal system of both teeth led to regeneration of the attachment apparatus without further periodontal therapy, which in these cases could have worsened the prognosis of the teeth. In fact, there is evidence that proper root canal treatment can heal sinus tracts originating from an endodontic lesion even if they have been present for a long time

(Stromberg et al, 1972). Root canal disinfection is crucial when attempting to achieve regeneration of the periradicular tissues (Bystrom A, Sundqvist GT, 1983; Bystrom et al, 1985; Trope et al, 1999). Whether complete root canal disinfection can be achieved in one appointment is beyond the scope of this article.

In both these cases, calcium hydroxide was used as the intracanal inter-appointment dressing to disinfect the root canal system further and to evaluate the improvement of the



Figures 3a-d: (a) Tooth 47 presented with severe bone loss around the apical and distal root area; (b) Guide files film at the start of root canal therapy; (c) Eightmonth recall, tooth had not been restored, considerable bone apposition is seen in all periradicular area, probing depth was 3mm; (d) At six years and six months recall, probing depths were 3mm all around the tooth, which remained asymptomatic.



Figures 4a-d: (a) Tooth 21 with severe bone loss; (b) Probing depth was 16mm all over the buccal surface; (c) Post obturation X-ray film; (d) One year and three months recall, 3mm probing depth all around tooth structure. Almost complete healing of periradicular tissues is evident.

surrounding tissues at the second appointment, at which time it was decided to fill both teeth.

We can conclude that endodontic lesions with involvement of the attachment apparatus can be successfully healed by performing adequate root canal treatment with great emphasis on disinfection of the root canal system. Understanding the mechanisms of bone destruction in these types of lesions is of great importance when trying to achieve successful healing.

References

American Association of Endodontists (2001) Pulpal and Periodontal Relationships. ENDODONTICS: Colleagues for Excellence. Spring/Summer

Blomlof LB (1993) Relationship between periapical and periodontal status. J Clin Periodontol 20: 117-23

Bystrom A, Sundqvist G (1983) Bacteriologic evaluation of the effect of 0.5% sodium hypochlorite in endodontic therapy. Oral Surg Oral Med Oral Pathol 55: 307-12

Bystrom A, Claesson R, Sundqvist G (1985) The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals. Endo and Dent Traum 1: 170-175

Bystrom A, Happonen RP, Sjogren U, Sundqvist G (1987) Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis. Endo Dent Traumatol 3: 586-3

Harrington GW (1979) The perio-endo question: differential

diagnosis. Dent Clin North Am 236: 73-90

Korzen BH, Krakow AA, Green DB (1974) Pulpal and periapical tissue responses in conventional and monoinfected gnotobiotic rats. Oral Surg Oral Med Oral Pathol 37: 783-802

Paul B, Hutter, JW (1997) The endodontic-periodontal continuum revisited: new insights into etiology, diagnosis and treatment. JADA 128: 1541-48

Seltzer S, Bender IB, Nazimov H, Sinai I (1967) Pulpitis-induced interradicular periodontal changes in experimental animals. J Periodontol 38: 124-9

Simon JHS, Glick DH, Frank AL (1972) The relationship of endodontic-periodontic lesions. J Periodontol 43: 202-208

Simring M, Goldberg M (1964) The pulpal pocket approach: Retrograde periodontitis. J Periodontol 35: 22-48

Stromberg R, Hasselgren G, Bergsted H (1972) Endodontic treatment of resorptive periapical osteitis with fistula. A clinical and roentgenological follow-up study. Swedish Dent J 65: 457-465

Trope M, Delano O, Orstavik D (1999) Endodontic treatment of teeth with apical periodontitis: Single vs. multi-visit treatment. J Endod 25: 345-350

Yamasaki M, Kumazawa M, Kohsaka T, Nakamura H, Kameyama Y (1994) Pulpal and periapical tissue reactions after experimental pulpal exposure in rats. J Endod 20: 13-7

Zehnder M, Hasselgren G (2002) Pathologic interactions in pulpal and periodontal tissues. J Clin Periodontol 29: 663-71