CASE REPORT

Apparent periapical repair without operative intervention: a case report and discussion

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Abstract

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Case report A case is described where substantial reduction of an established periapical lesion appeared to take place in the absence of operative intervention, and as the crown of the tooth was progressively destroyed by dental caries. The case raises debate on the pathogenesis, diagnosis and monitoring of endodontic lesions, and may stimulate renewed research interest in these most fundamental elements of clinical endodontology.

Keywords: apical pathosis, healing, host/parasite relationship, imaging.

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Introduction

The endodontic literature contains a number of reports of periradicular lesions healing without the apparent need of a root filling (Walker 1984, Donnoley 1990). Such cases should not cause surprise. Numerous studies published during the last two decades have provided compelling evidence that periapical radiolucencies of endodontic origin are established and propagated by microorganisms residing in the pulp canal space (Fabricius *et al.* 1982a, Korzen *et al.* 1974, Moller *et al.* 1981, Sundqvist 1976), and that only by eliminating this infection can healing be anticipated. Healing is therefore expected when the canal system of a tooth has been properly instrumented, medicated, and temporized against coronal reinfection, even if the canals were not filled.

This report describes an unusual and perplexing case of apical periodontitis, in which significant bony healing appeared to take place without any operative intervention, and in the presence of long-term dental neglect. Presentation of this case serves to stimulate debate on the pathogenesis of endodontic lesions, and the imaging techniques we use to diagnose and monitor them.

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Case report

A 35-year-old female Caucasian attended Newcastle Dental Hospital complaining of pain on biting from tooth 36 (FDI). There was no significant medical history. Clinical examination revealed a large carious lesion breaching the distal marginal ridge of tooth 36, and pulp stimulation with ethyl chloride and an electronic pulp tester yielded no response. Associated buccal soft tissues were erythematous and slightly swollen, with no evidence of discharge through a fistula, or through the intact periodontium. The tooth was moderately tender to percussion. A periapical radiograph (Fig. 1) identified the presence of a large carious lesion involving the pulp, and of 5 mm diameter radiolucencies affecting both mesial and distal roots. A diagnosis of pulp necrosis, with an acute exacerbation of chronic apical periodontitis was made for tooth 36.

The patient expressed her desire to save the tooth and was referred for caries control, and to initiate root canal therapy. However, she left the building without any treatment.

Thirty-one months later, the patient returned to the Newcastle Dental Hospital, this time complaining of lingual discomfort from abrasion against the sharp lingual wall of tooth 36. She had received no dental care in the interim, and gave no history of antibiotic use.

Tooth 36 was found to be unrestorably carious, with sharp spurs of enamel remaining mesio-lingually. There

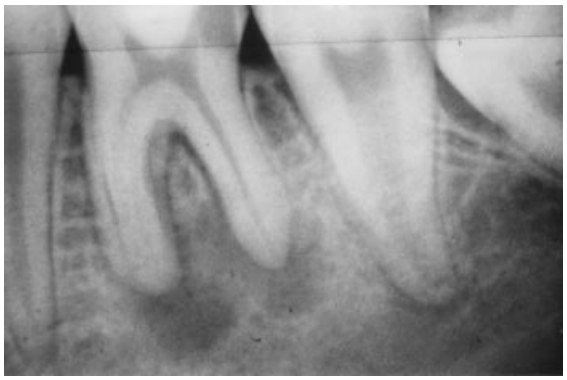

Figure 1 Tooth 36 at presentation with 5 mm apical lucencies on mesial and distal roots.

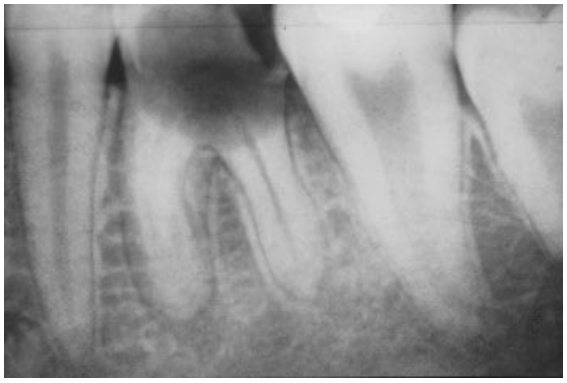

Figure 2 Tooth 36 31 months later with coronal destruction and apparent periapical improvement.

was no discomfort from the tooth itself, and the associated soft tissues appeared healthy. A periapical radiograph (Fig. 2) revealed massive caries affecting the whole of the crown and involving the root bifurcation. The periapical lesion affecting the distal root appeared to have completely healed with the re-establishment of a normal periodontal ligament shadow. The mesial lesion had also substantially reduced in size radiographically.

Tooth 36 was unrestorable, and was extracted. No histological or microbiological investigations were undertaken.

Discussion

There is a substantial, and seemingly irrefutable body of evidence to suggest that lesions of endodontic origin are manifestations of microbial infection (Kettering & Torabinejad 1998). An equally strong body of evidence supports the view that control of intracanal infection is a fundamental prerequisite for periapical healing to take place (Maalouf & Gutmann 1994). It is therefore perplexing when a lesion of endodontic origin is found to show signs of healing without any effort to control or even upset the ecological balance of the microbial flora of the affected tooth (Sundqvist 1992). Far from managing infection by careful operative management, the tooth described had been open to the mouth for many months through a progressively enlarging carious lesion. Although no microbiological sampling was undertaken, it is difficult to conceive that the canal system had been sterile during its time of exposure to advancing caries, infected saliva and foodstuffs.

Periodontists have long debated the concept of a 'healthy' periodontal flora, and have described specific and nonspecific plaque hypotheses in the pathogenesis of periodontal diseases. The specific plaque hypothesis (Loesche 1979) nominates certain organisms as putative periodontopathogens, and therefore acknowledges that other strains may be host-compatible and nonpathogenic. The nonspecific hypothesis (Theilade 1986), on the other hand, describes a critical biomass of any microorganism as being capable of inducing adverse tissue reactions. Subthreshold levels of organisms are therefore nonpathogenic.

Specific and nonspecific plaque hypotheses have been discussed less by endodontists.

Korzen *et al.* (1974) observed that the severity of periapical inflammatory response was related to the *quantity* of microorganisms within the root canal, and the length of time the tissues were exposed to them. They did, however, note that monoinfections tended to produce less severe reactions than mixed infections. This tendency was corroborated by Fabricius *et al.* (1982b), who showed that some combinations of bacteria were more potent in inducing apical periodontitis than other single strains, and that anaerobes were significantly associated with apical periodontitis. But they were also clear (Fabricius *et al.* 1982a) that it had not been definitely proven whether certain species were more responsible than others for the induction of apical periodontitis. Watts & Paterson (1992), for example, have shown that even monoinfection of the pulp by mutans streptococci could induce apical periodontitis in animals.

But as anaerobic culturing and diagnostic techniques have developed, convincing specific microbial associations have emerged. Gram negatives, especially the black pigmented prevotella and porphyromonas species have been especially recognized in the pathogenesis of periapical inflammatory lesions (Trowbridge & Stevens 1992, Yoshida *et al.* 1987, Griffe *et al.* 1980).

And more recently, Gomes *et al.* (1994) have added further weight to the specific microbial case by showing significant associations between certain bacteria, and combinations of bacteria and specific endodontic signs and symptoms.

It is therefore conceivable that changes in the redox potential, or nutritional status within tooth 36 as it continued to be opened by caries may have provided selective pressures for a change in the microbial flora of the canal system to a less pathogenic one, incapable of sustaining chronic inflammatory change.

But to focus entirely on the microbial contents of the root canal system is to disregard the possible role of changes in the host response. Fabricius *et al.* (1982b) raised the question whether, when an infection has been in progress for a long time, the host can mobilize defense factors against bacteria in root canals and/or their products, and to what extent such factors may influence the host parasite relationship during the development of apical periodontitis. Molander *et al.* (1998) also drew attention to the possibility of periapical tissue healing in the presence of continued infection. Whilst it is common to witness small lesions of endodontic origin that remain unchanged for many years, indicating some balance between host and microbial flora (Van Nieuwenhuysen *et al.* 1994), it is far less common to see a large, established lesion dramatically reduce in size, as if by the switching on of enhanced host defenses after a period of antigenic challenge. But again, this eventuality cannot be ruled out in the case described.

An additional consideration surrounds the initial diagnosis of the periapical lucencies affecting tooth 36. Radiographic differentiation of periapical cysts and granulomas is notoriously difficult (Whaites 1996a), and even routine histopathological investigation can produce erronious diagnosis (Nair 1998). Traditionally, clinicians have diagnosed lesions of more than 1 cm in diameter, and with well-corticated margins as cystic (McIvor 1986). But this rule of thumb approach may result in the misdiagnosis of many lesions greater or smaller, and it is uncertain what the true nature of the lucencies affecting the reported tooth were. Decompression is a well established method of initiating cystic healing (Hoen *et al.* 1990, Neaverth & Burg 1982), since the expansion of both true and pocket cysts is commonly held to be pressure-related. It is possible that the lesions were in fact periapical pocket cysts, and that coronal breakdown, possibly in combination with apical inflammatory root resorption, may have opened a free pathway for drainage and decompression. Radiographic signs of healing may have then followed epithelial disruption and breakdown, despite the continued presence of intracanal infection.

Diagnostic issues are further clouded by problems of imaging and image interpretation. The success of root canal treatment is usually assessed from plain radiographs because no other method is either reliable or practical (Pitt Ford 1984). But the reliability of routine radiographs is questioned by the widely held view that cortical erosion or perforation is required for the detection of periapical lesions (Bender & Seltzer 1961, Regan & Mitchell 1963, Wengraf 1964, Schwartz & Foster 1971). Slight decompression and shrinkage of a periapical lesion and consequent regeneration of cortical plate may therefore have given the impression of repair in the presence of a still substantial lesion. This position is not, however, unchallenged. At least two studies have indicated that lesions confined to the cancellous space can be imaged by routine methods (Pitt Ford 1984, Lee & Messer 1986). Whilst more sophisticated techniques, such as tomography and magnetic resonance imaging may provide the potential for better intrabony imaging (Whaites 1996b), they have their radiation, financial and logistic costs, and cannot be considered for routine endodontic imaging in their present form.

Conclusion

This report documented a case in which substantial healing of an established periapical lesion appeared to take place without operative intervention, and in the presence of dental neglect. The case raises debate on the pathogenesis, diagnosis and monitoring of endodontic lesions, and may stimulate renewed research interest in these most fundamental elements of clinical endodontology.

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