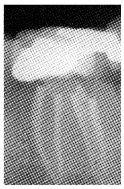

CASE REPORT

Calculus-like deposit on the apical external root surface of teeth with post-treatment apical periodontitis: report of two cases

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Abstract

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Aim To report two cases in which calculus-like material was found on external root surfaces of (i) an extracted root and (ii) an apicected part of a root, both of which were removed due to post-treatment refractory apical periodontitis.

Summary In each case, there was a fistulous tract, which did not heal after conventional root canal treatment. The first case did not heal even after apical surgery, and subsequent tooth extraction revealed calculus-like material on a root surface of complex anatomy. The second case showed radiographic signs of healing after apicectomy. Histology of the apical biopsy revealed a calculus-like material on the external surface of the root apex. It is suggested that the presence of calculus on the root surfaces of teeth with periapical lesions may contribute towards the aetiology of failure.

Key learning points

• Biofilm on the external root surface has been implicated in the failure of apical periodontitis to heal, despite adequate root canal treatment.

• Calculus-like material was found, in two cases, on the root surface of teeth with post-treatment apical periodontitis, where the only communication externally was a sinus tract.

Keywords: apical periodontitis, calculus, endodontic failure, extraradicular infection.

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Introduction

The major aetiological factor for the development and the maintenance of apical periodontitis is the colonization of microorganisms (bacteria and fungi) in the root canal

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system (Kakehashi *et al.* 1965, Sundqvist 1976). Elimination or substantial reduction of microorganisms from the root canal system is therefore crucial for the resolution of apical periodontitis.

However, because of the complexity of the root canal system, complete elimination of endodontic infection cannot always be achieved. Bacteria located in isthmuses, ramifications, dentinal tubules and irregularities of the root canal walls may be unaffected by endodontic disinfection procedures. Bacterial antigens egressing into the periapical area maintain the periapical inflammation, and ultimately cause endodontic treatment failures (Nair *et al.* 1990, 1999, Lin *et al.* 1991, Siqueira 2000).

It has been reported that certain microorganisms may survive outside the root canal and maintain the inflammatory disease in periapical tissues. This has been suggested to happen either by microbes adhering to the apical root surface, forming biofilm-like structures (Tronstad *et al.* 1987, 1990, Noiri *et al.* 2002), or by their existing within the inflamed periapical tissues, usually as cohesive colonies, becoming independent of the root canal infection. Bacteria of the genera *Actinomyces* and *Arachnia* may prevent periapical healing in this way by establishing themselves in the periapical tissues (Happonen *et al.* 1985, Happonen 1986, Byström *et al.* 1987, Sjögren *et al.* 1988, Figdor *et al.* 1992). The presence of bacteria on the root surface in failed endodontic treatment has also been reported in histomorphological studies (Bergenholtz *et al.* 1983). However, descriptions of calculus found on the external surface of root tips of teeth with post-treatment apical periodontitis are sparse. Harn *et al.* (1998) described a case of periapical lesion with a fistulous tract, which did not resolve after conventional root canal treatment. During surgical treatment of the tooth a calculus-like deposit was observed on the apical root surface. This deposit was removed with the lesion. Thereafter, the sinus tract disappeared and radiographic healing was observed at 9 months. According to the histopathological report, the diagnosis of the periapical lesion was that of a radicular cyst.

Histological demonstrations of calculus associated with endodontic failures are few. In a study of 66 endodontic failures, Andreasen & Rud (1972) described a case in which, at the surgical operation, 'it was noted that the apex was covered with concrements and these were removed'. The treatment failed, and histobacteriological analysis demonstrated bacteria within clefts of the cementum.

Rud & Andreasen (1972) described two cases in which a 'filamentous material, similar to dental calculus' was found covering the root surface. Deposits of a similar structure had also been found and carefully removed some years previously when the cases were initially operated on.

The purpose of this communication is to report two cases in which calculus-like material was found on the external surfaces of (i) an extracted root and (ii) an apicected part of a root, both of which were removed due to post-treatment apical periodontitis. In each case there was a sinus tract which did not resolve after conventional endodontic treatment.

Case 1

A 22-year-old man sought treatment of large carious lesions in both maxillary left premolars. The first premolar had a carious cavity mesially, but the pulp responded sensitive to cold, hot and electrical tests. The second premolar exhibited extensive carious destruction and the pulp was not sensitive to pulp tests. There was a sinus tract exiting buccally in the apical area. The tooth was tender to percussion, presented no mobility, and meticulous periodontal probing revealed all pockets to be less than 2 mm in depth. A periapical radiograph of the second premolar confirmed the extensive coronal carious lesion involving the pulp and revealed a large periapical lesion (Fig. 1a).

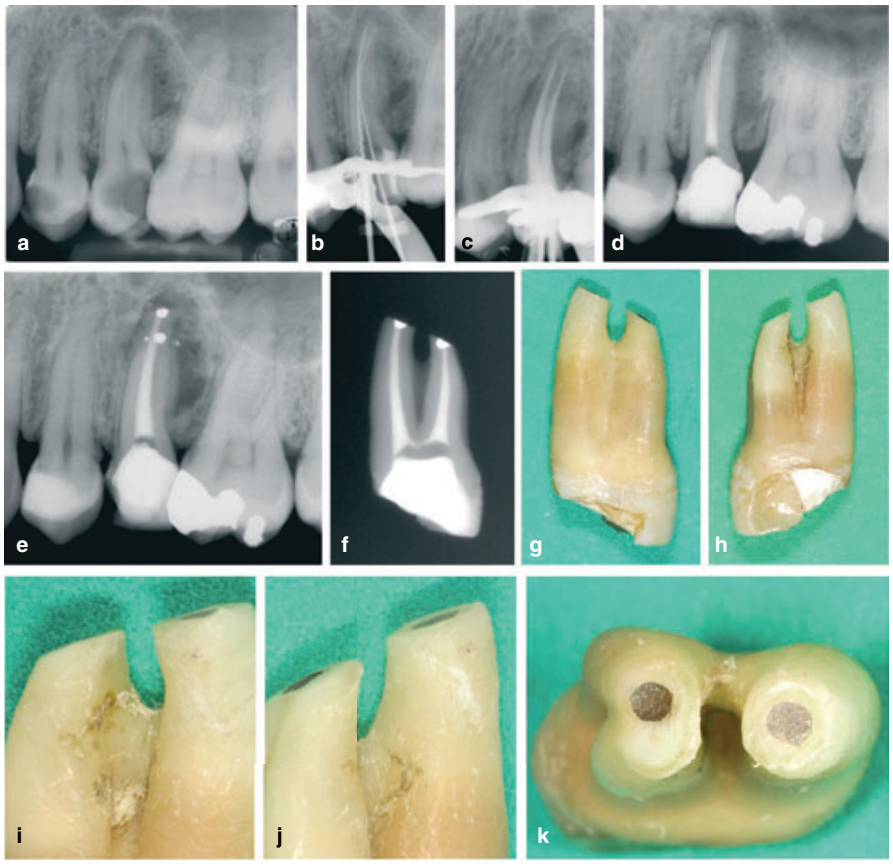

Figure 1 (a) Preoperative radiograph. (b) Working length radiograph. (c, d) Obturation of canals. (e) Apicectomy. (f) Radiograph of extracted tooth, taken at a 90° angle. (g) Mesial aspect of extracted tooth. (h–j) Distal aspect, showing grey and brown calculus-like deposits. (k) Apical view of tooth anatomy.

Root canal treatment was scheduled. After rubber dam isolation, removal of all carious tissue and access cavity preparation, a working length radiograph was taken (Fig. 1b). During canal preparation, abundant irrigation with 1% sodium hypochlorite was carried out after each instrument. The canals were dried and filled with chemically pure calcium hydroxide mixed with sterile saline. The sinus tract was still present after 3 weeks. The root canals were re-opened, irrigated and packed with calcium hydroxide. This procedure was repeated three times at intervals of approximately 3 weeks. The canals were then filled with laterally condensed gutta-percha and a sealer (Fig. 1c,d).

One month later, the patient returned because the sinus tract had reappeared. Endodontic surgery was scheduled. A full-thickness gingival flap was raised, an apicectomy was performed, and apical root-end cavities were prepared and filled with amalgam (Fig. 1e). The pathological periapical tissue was removed intact and as thoroughly as possible. The patient returned 3 weeks post-surgery with pain and the sinus tract unhealed. Extraction of the tooth was recommended to which the patient consented.

A radiograph of the extracted tooth was taken at a 90° angle (Fig. 1f) to observe the centricity and homogeneity of the endodontic obturation. Soft tissue remnants were removed from the root surface by immersion of the root in 5% NaOCl for approximately 15 min and photographs were taken from the mesial and distal aspects. The mesial aspect

(Fig. 1g) exhibited two fused roots with a shallow concavity running longitudinally and a bifurcation in the apical third. The distal aspect (Fig. 1h) presented a deep concavity separating the two partially bifurcated roots, partly covered by grey and brown calculus-like deposits (Fig. 1i,j) that had not been detected during the surgical procedure (Fig. 1k).

Case 2

A 51-year-old woman was referred by her dentist for endodontic evaluation of the maxillary right quadrant, which presented a sinus tract between the first and the second premolar. The first premolar had been root filled 2 years previously. The patient had noticed the sinus tract about 1 month before, but did not have any pain.

The second premolar and first molar responded within normal limits to thermal tests and gave negative responses to percussion and palpation. The patient was unable to give clear information to thermal testing of the canine, which was slightly sensitive to percussion. The first premolar, on the other hand, gave a very positive response to percussion and palpation tests. There was no swelling and meticulous periodontal probing revealed no pockets of more than 2 mm deep.

A radiograph, taken with a gutta-percha tracer in the sinus tract (Fig. 2a), demonstrated a large periapical radiolucency extending from the mesial radicular aspect of the second premolar to the distal radicular aspect of the canine, and the gutta-percha cone was pointing between the premolars. Root canal re-treatment of the first premolar was scheduled. After access cavity preparation and removal of the existing obturation material, a working length radiograph was taken (Fig. 2b), the file lengths adjusted accordingly and the canals manually re-instrumented, thoroughly irrigating with 1% NaOCl solution. The canals were then filled with pure calcium hydroxide powder mixed with saline.

After 30 days the sinus tract had disappeared, the calcium hydroxide medication was removed and the root canals were filled with gutta-percha, laterally condensed with a sealer. The postoperative radiograph demonstrated that some sealer had extruded into the periapical tissue (Fig. 2c).

Six months later the patient returned because the sinus tract had reappeared. It was decided to retreat the root canals once more. After removal of gutta-percha, the canals were instrumented and packed with calcium hydroxide. After 2 months the sinus tract was still present (Fig. 2d). Apical surgery was recommended and the patient's consent obtained.

At this point the canine was tested again, and at this time gave a negative response to thermal tests and was positive to percussion. Although the canine clearly had a separate problem from that of the premolar, it was decided that there was no reason for further review and the canine should now also be root canal treated; pulp necrosis of the canine was confirmed during the treatment. The two teeth were obturated by lateral condensation of gutta-percha and AH26 sealer (Fig. 2e). The surgery was carried out 2 days later, for patient convenience (Fig. 2f). Approximately 3 mm of the apex of the premolar were removed using a diamond fissure bur. Cold burnishing of gutta-percha filling was performed. It was noted that the lesion did not affect the canine apex, where sound bone was present, confirming the premolar as having been the cause of the lesion. At a 9-month review, there were no signs or symptoms; the tooth was still temporized with IRM (Caulk, Milford, DE, USA) and glass–ionomer cement. The radiograph demonstrated a reduction in the size of the lesion (Fig. 2g).

The root apex, together with several fragments of the pathological periapical tissue that were not obtained *in toto* with the apex in their original relationships, were immediately immersed in 10% neutral formalin. The root apex was then decalcified in a solution of 22.5% (v/v) formic acid and 10% (w/v) sodium citrate for a period of 3 weeks under

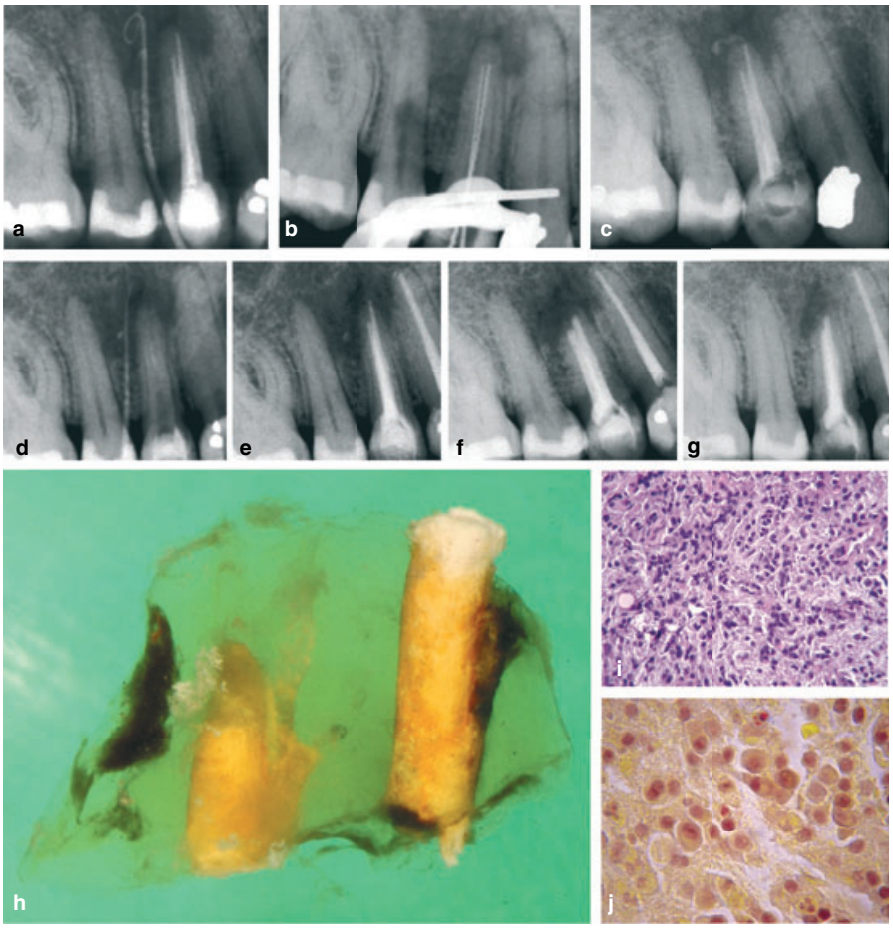

Figure 2 (a) Preoperative radiograph with gutta-percha tracer. (b) Canals were re-opened. (c) Post-obturation. (d) Sinus tract still present at 6 months; canals were re-opened and calcium hydroxide placed. (e, f) After 2 months, sinus tract still present, thus obturation of canals and apicectomy was performed. (g) 9 months later bone regeneration is appreciable. (h) Cleared root apex. (i) Histopathological view of periapical tissue, H & E stain demonstrating accumulation of chronic inflammatory cells. Original magnification 400×. (j) Taylor modified Brown & Brenn stain demonstrating no bacterial colonization of the tissue. Original magnification 1000×. (k) Section of root apex passing through buccal canal (see text). Taylor modified B & B stain, original magnification 25×. Original magnification of left inset 400×. Original magnification of right inset 1000×. (l) Section passing through the palatal canal (see text). (n) Bacterial aggregates in deeper part of amorphous material (rectangle in 2L). (m) Bacterial colonies between root canal wall and obturating material (arrow in 2L). Taylor modified B & B stain, original magnification 25× (L), 1000× (M and N).

constant agitation (Lipshaw bone decalcifier; Lipshaw MFG Co., Detroit, MI, USA). The decalcification process was controlled radiographically. A photograph of the root apex was taken at the end of decalcification, whilst immersed in the clearing agent (Fig. 2h). The cleared root apex showed gutta-percha extending beyond the foramen in one canal, whilst the obturation was short of the apex in the other. Apical ramifications were also present, but were free of obturating material.

The decalcified root apex and the fragments of periapical soft tissue were processed for routine histological examination. The specimens were embedded separately in paraffin blocks. Particular care was taken in embedding the root apex: the specimen was orientated parallel to the long axis of the two root canals, and serial sections in a

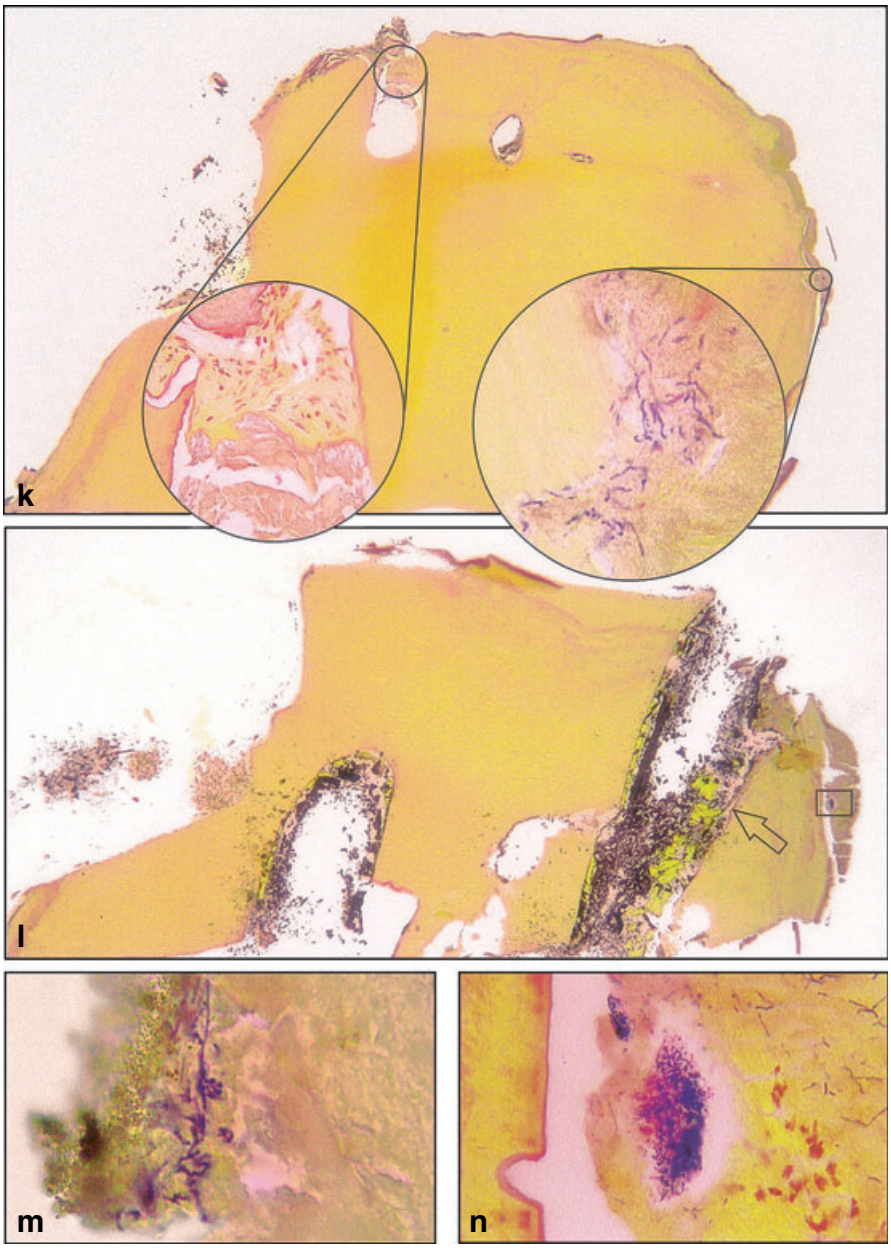

Figure 2 (Continued).

bucco-lingual plane were cut with the microtome set at 4–5 μm until the whole specimen was cut. Every fourth slide was stained with haematoxylin and eosin. Selected slides were stained for the presence of bacteria with a modified Brown–Brenn stain (Taylor 1966).

Histological sections of the pathological periapical tissue demonstrated accumulation of chronic inflammatory cells (lymphocytes, plasma cells and macrophages) (Fig. 2i) and absence of bacterial colonization (Fig. 2j).

Sections of the root apex (Fig. 2k), passing through the buccal canal, which had been filled short of the apex, exhibited debris packed at the apical constriction and bordered apically by a vital tissue with scattered chronic inflammatory cells (left inset). On the palatal aspect, the root surface was covered by an amorphous layer, which, at higher

magnification (right inset), disclosed numerous bacterial profiles in the deepest part. Bacterial colonization of this calculus-like material was more evident in the sections passing through the palatal canal (Fig. 2l). The amorphous material (demarcated by the rectangle in Fig. 2l) showed bacterial aggregates in the deeper part, and was lined by inflammatory cells (Fig. 2n). Between the root canal wall and the obturating material (arrow in Fig. 2l) there were bacterial colonies (Fig. 2m).

Discussion

The principal cause of failure of root canal treatment is the persistence of bacteria within the endodontic system (Nair *et al.* 1990, 1999, Lin *et al.* 1991, 1992). In a small number of cases, extraradicular infection by bacteria of the genera *Actinomyces* and *Propionibacterium* can sustain post-treatment apical periodontitis (Nair & Schroeder 1984, Byström *et al.* 1987, Sjögren *et al.* 1988, Figdor *et al.* 1992). Recently, various scanning electron microscopic studies of apices of teeth with necrotic pulps with periapical lesions have demonstrated the presence of bacterial plaque on the external root surface, in lacunae of the cementum or in areas of resorption (Lomçali *et al.* 1996, Leonardo *et al.* 2002). The presence of these extraradicular bacterial biofilms has been related to refractory apical periodontitis (Noiri *et al.* 2002, Sunde *et al.* 2002).

Carranza (1990) describes calculus and its formation. Calculus is dental plaque that has undergone mineralization. The soft plaque is hardened by the precipitation of mineral salts, which usually starts between the 1st and 14th days of plaque formation. All plaque does not necessarily undergo calcification. Early plaque contains a small amount of inorganic material, which increases as the plaque develops into calculus. Plaque that does not develop into calculus reaches a plateau of maximal mineral content within 2 days. Separate foci of calcification increase in size and coalesce to form solid masses of calculus. As calcification progresses, the number of filamentous bacteria increases and foci of calcification change from basophilic to eosinophilic. Calculus is formed in layers, which are often separated by a thin cuticle that becomes embedded in the calculus as calcification progresses. There are two principal theoretical mechanisms by which plaque becomes mineralized, namely that mineral precipitation results from a local rise in the degree of saturation of calcium and phosphate ions, and that seeding agents induce small foci of calcification that enlarge and coalesce to form a calcified mass.

There are various ways in which calculus can develop on a root surface. Most commonly, the origin of its components is the gingival sulcus, but in the two cases described the surrounding marginal bone was intact and meticulous probing had confirmed that the sulcular depths were minimal and there did not exist any communication whatsoever with the lesions. A possible criticism could be that, in the first case, there may have been a very narrow periodontal pocket impossible to detect even by careful probing, and that the deposits were a result of an endo-perio lesion, which developed subsequent to coronal extension of the apical pathology distal to the tooth involved. This possibility of a primary endodontic lesion with secondary periodontal involvement was dismissed for the simple reason that there was, on scrupulous examination of the root surface, no evidence of any form of deposit on the more coronal aspect of the root surface beneath the level of gingival attachment (Fig. 1h).

The periapical calculus-like structure found in these cases could have formed from a bacterial plaque that had developed on the external surface of the root tip. Such plaque, when observed under a scanning electron microscope, appears to be similar to marginal periodontal plaque (Tronstad *et al.* 1990). The periapical plaque may undergo calcification. Saliva is the source of mineralization for supragingival calculus, whereas serum transudate

of gingival crevicular or pocket fluid furnishes the minerals for subgingival calculus (Carranza 1990).

In the case described by Harn *et al.* (1998) and in the two cases described in this article, there were long-standing sinus tracts. The sinus tract is potentially a 'corridor', a plausible route of communication, between the periapical area and the external environment. In addition to the minerals available from tissue fluid, it is possible that the passage of minerals and salts from the oral fluids into the periapical lesion, via the sinus tract, favours the calcification of plaque found on the root surface, hence calculus formation.

Periapical bacterial plaque may calcify in the absence of a sinus tract, the minerals being furnished by periapical inflammatory exudates (Sunde *et al.* 2002). Some bacterial species have also been shown to have the ability to form intracellular apatite crystals (Streckfuss *et al.* 1974).

Inter-appointment dressing of root canals of teeth with apical periodontitis, using calcium hydroxide, is an efficient method of root canal disinfection (Byström *et al.* 1985). Combined with thorough canal preparation and abundant irrigation using sodium hypochlorite, one would expect to obtain healing of a periapical lesion. However, an extraradicular bacterial plaque cannot be destroyed by conventional root canal irrigants or antimicrobial dressings. The microorganisms on the external surface of the root end remain untouched, the inflammatory periapical process continues and healing does not occur. This can be seen in both cases presented here. Although some authors suggest depositing endodontic disinfectants into periapical areas so as to eliminate the microorganisms of periapical bacterial plaque in persistent periapical infections (Lomçali *et al.* 1996), at present apical surgery is the evidence-based treatment of choice to resolve these refractory cases.

In the first case described here, the presence of calculus was not evident during the surgical procedure, due to the root anatomy, and the treatment failed. Even if it had been suspected to be present, it would have been difficult to identify calculus hidden in such a groove in the root. It seems a reasonable theory that the calculus-like material found in the two cases was aetiologically related to the persistence of periapical inflammation. In the second case, once the calculus-like deposit on the root end had been removed by apicectomy, healing occurred.

Conclusion

The presence of calculus-like deposit was observed on the root surface of two teeth with post-treatment apical periodontitis. It was suggested that the continuing ingress of oral fluids via the sinus tract, present in each case, allowed the formation and the maintenance of these deposits. The calculus-like material possibly constituted an attachment base for an extraradicular biofilm. The latter maintained the periapical inflammation, and prevented periapical healing, despite adequate orthograde root canal treatment. After removal of the root end in apical surgery, the clinician should carefully examine the remaining root surface for the presence of mineralized deposits. If calculus-like material is found, it should be removed in order to allow periapical healing to occur.

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