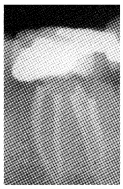

CASE REPORT

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Nonsurgical treatment of extensive cyst-like periapical lesion of endodontic origin

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Abstract

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Aim To report the repair of an extensive periapical lesion of endodontic origin, following nonsurgical treatment.

Summary Clinical and radiographic examination revealed an extensive periapical lesion related to tooth 22, extending from the distal surface of tooth 21 to the mesial surface of 26. The patient reported a previous history of dental trauma involving this quadrant and had been under orthodontic treatment for a year. Intraoral examination revealed an asymptomatic bony hard swelling, mainly confined to the palate. During root canal exploration irregular walls associated with 3 mm of apical calcification were noted. After apical patency was obtained 1 mL of bloody serous exudate was drained. Intracanal aspiration provided a further 2 mL of yellow serous exudate. Following biomechanical preparation, a dressing of calcium hydroxide with anaesthetic solution was applied and replaced four times over a period of 12 months. The clinical–pathological picture demonstrated resolution of the lesion during this period of time. The 14-month clinical and radiographic examinations revealed normal bony contour and a significant resolution of the maxillary radiolucency.

Key learning points

- Periapical lesions of endodontic origin may develop asymptomatically and become large.
- Proper biomechanical preparation followed by calcium hydroxide medication renewed periodically represents a nonsurgical approach to resolve extensive inflammatory periapical lesions.

Keywords: apical cyst, calcium hydroxide, periapical healing, root canal treatment.

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Introduction

Dental trauma is often associated with the disruption of pulp blood supply, leading to necrosis. Infection may be immediate or delayed. Less severe trauma may result in hyperaemia, oedema, haemorrhage or ischaemia. Partial ischaemic infarction may persist for months or years (Stanley *et al.* 1978) and during these circulatory changes, the pulp may exhibit various responses, including dystrophic calcification, internal resorption and pulpitis that may evolve into partial or total pulp necrosis. Circulatory breakdown causes tissue necrosis and anaerobic conditions for the growth of opportunistic microorganisms. Abscesses, granulomas and apical cysts develop in response to the intracanal antigenic content, mediated by immunopathologic mechanisms (Soares & Queiroz 2001). Efficient neutralization of microorganisms and the removal of byproducts of cells and microorganisms as well as preventing reinfection are prerequisites in treating apical pathosis. The greatest impact may therefore be achieved by effective biomechanical preparation and calcium hydroxide dressing, which promotes anti-sepsis of the root canal system and mineralized tissue formation in the apical region (Sjögren *et al.* 1997, Soares *et al.* 2005).

The presence of a cystic periapical lesion, granuloma, cholesterol crystal accumulation and a positive microbiological culture before root filling have been cited as factors which may compromise the outcome of root canal treatment (Nair *et al.* 1996, 1998, Sjögren *et al.* 1997, Nair 1998). Extensive periapical lesions may not always heal (Sjögren *et al.* 1997) and periapical surgery or even extraction may be necessary to allow the lesion to heal. Amongst surgical therapies for extensive osteolytic lesions, marsupialization may be the least invasive, although mild discomfort may be associated with long-term use of drains, maintaining hygiene may be difficult, healing may be slow and further surgery may be needed. In the case of enucleation, major vessels and nerves must be protected and reconstructive grafts may be needed, requiring specialist hospital care (Callestini 1996).

This report describes the substantial resolution of a large periapical lesion after conventional root canal treatment.

Case report

A 23-year-old female patient attended the endodontic clinic at the Federal University of Jequitinhonha Wales and Mucuri, Diamantina, MG, Brazil, requesting an oral health evaluation. One month previously, she had presented with severe pain in the area of teeth 22 and 23 and had been prescribed analgesic and antibiotic medication. Her past medical history included allergic rhinitis 6 months previously for which she had been prescribed antibiotics and anti-allergic medication. A year before, the patient had begun fixed orthodontic treatment and had her second maxillary premolars extracted in order to correct the labial displacement of tooth 22. She also reported a bicycle accident 7 years previously which resulted in trauma to tooth 22. Intraoral clinical examination revealed painless swelling of the labial mucosa adjacent to teeth 22–24. Swelling was more extensive in the palate from tooth 22 to 26 (Fig. 1) with a firm consistency. Tooth decay and periodontal pockets were absent. Electronic (Vitality Scanner; Analytic Technology, Glendora, CA, USA) and thermal (Endo-Ice; The Hygenic Corporation, OH, USA) pulp sensitivity testing were negative only for tooth 22, whilst adjacent teeth presented normal responses. All maxillary teeth were painless on vertical percussion. A diagnostic periapical radiograph showed an extensive radiolucent area extending from the distal aspect of tooth 21 including the periapical region of teeth 22 to 24 and reaching the mesial surface of tooth 26. The extent of periapical radiolucency was 32×25 mm, established by

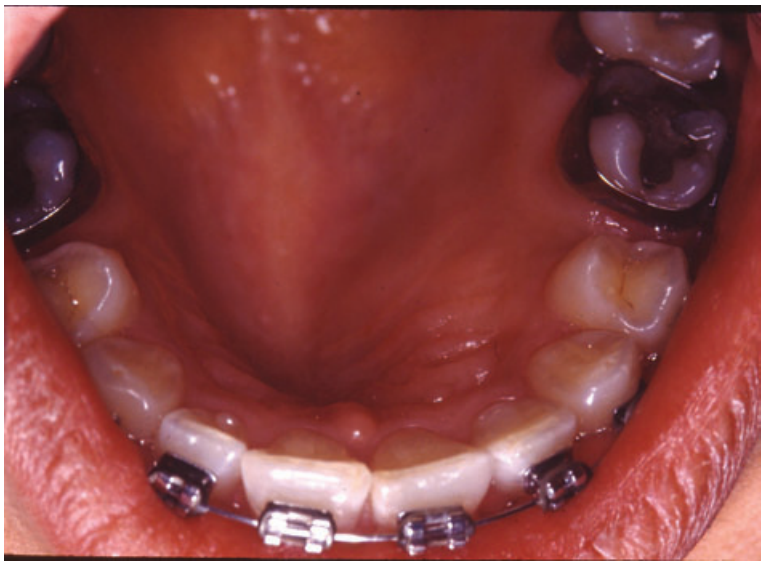

Figure 1 Asymptomatic palatal swelling that was resistant to digital compression.

Figure 2 Occlusal radiograph showing bony radiolucency involving two-thirds of the left maxilla.

occlusal radiography (Fig. 2). Taking into account her medical and dental history a presumptive diagnosis of pulp necrosis of traumatic origin with extensive inflammatory apical periodontitis was established. It was decided to perform root canal treatment on tooth 22.

Root canal treatment

After coronal access, examination of the root canal revealed wall irregularities, suggesting dystrophic calcification or resorption, as well as calcification of the apical 3 mm. This calcification presented resistance to the passage of a small K-file. In order to obtain apical patency 3 mm was cut from the tip of a 25 mm size 10 K-file. With a fine sandpaper disc normally used for finishing composite resin, the file tip was formed into a sharp shape that we describe as a 'file-probe'. After precurving the file-probe according to the long axis of the tooth as seen in the periapical radiograph, the root canal was filled with 15% trisodium ethylenediamine tetraacetic acid (EDTA), pH 7.0–7.5 (Biodinamica Quimica e Farmaceutica Ltda, Ibiporã, Paraná, Brazil). With short controlled movements and monitoring by periapical radiography, the apical calcification was trephinated and root canal patency obtained (Fig. 3). Following this procedure, 1 mL of bloody serous exudate drained through the canal and gentle aspiration with a luer lock syringe and 30/06 needle blocking the access opening with a wet cotton pellet provided a further 2 mL of yellow serous exudate (Fig. 4). The step-back technique of canal preparation was performed, accompanied by irrigation with 5.25% sodium hypochlorite. After removal of the smear layer by irrigation with 3 mL of 15% trisodium EDTA and manipulation of the master apical file for 3 min, followed by irrigation with sodium hypochlorite, the root canal was filled with calcium hydroxide mixed with anaesthetic solution (Prilocaine chlorhydrate 3% with felypressin 3 μ mL^{-1}; Astra Chemistry, São Paulo, Brazil) to a toothpaste consistency and introduced with a lentulo spiral. The intracanal dressing was programmed monthly, but

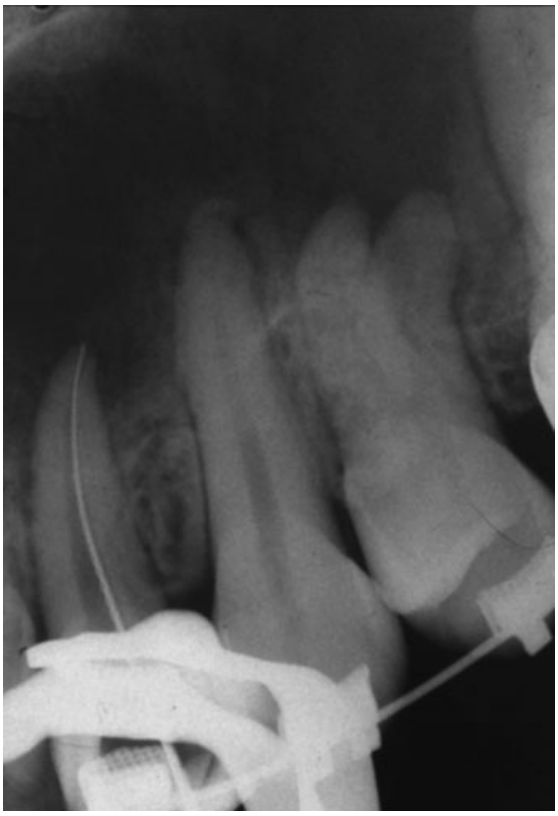

Figure 3 Apical patency.

Figure 4 Two millilitres of exudate drained from root canal.

poor patient compliance resulted in only four dressing changes over a period of 12 months. After the second change of dressing, there was no intracanal exudate and the periapical radiolucency progressively reduced without pain (Figs 5 and 6). Following these procedures, the root canal was filled with gutta-percha cones and Sealapex cement (Kerr/Sybron Dental Specialities Inc., Glendora, CA, USA; Fig. 7). The follow-up at 14 months demonstrated no intraoral swelling and radiographic evidence of bony repair (Fig. 8).

Discussion

Typically, inflammatory periapical lesions of endodontic origin range from 5 to 8 mm in diameter (Murphy *et al.* 1991, Sjögren *et al.* 1997). Traditionally, lesions up to 10 mm were considered granulomas whilst larger ones were considered as apical cysts (Lalonde 1970, Morse *et al.* 1973). The epithelial lining of radicular cysts is thought to arise from epithelial cell rests of Malassez in the periodontal ligament and is believed to proliferate as a result of inflammation, usually following pulp space infection. Such lesions may grow by a variety of mechanisms, including osmotic fluid accumulation in the lumen, epithelial proliferation and molecular mechanisms (Nair 1998). Çaliskan (2004) demonstrated extensive lesions with characteristics of apical cysts varying from 7 to 18 mm diameter radiographically. In the present case (lesion size 32 × 25 mm), a bony swelling and the presence of straw-coloured exudate strongly indicated an apical cyst. It is unclear whether the maxilla had been radiographically screened for pathosis before orthodontic treatment.

Until the 1960s, endodontists, pathologists and oral and maxillofacial surgeons considered that apical cysts would not respond to root canal treatment alone and that surgery was always required (Winstock 1980). However, this concept has changed.

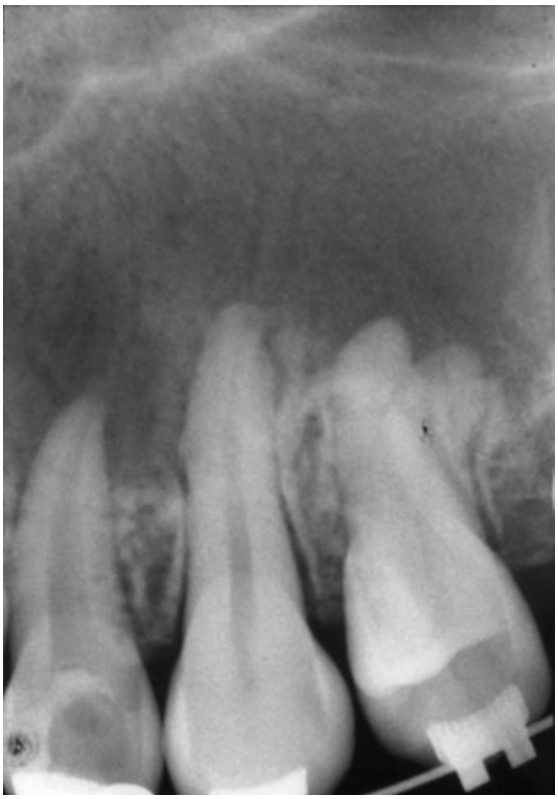

Figure 5 Signs of lesion repair at 4 months follow-up.

Histopathological studies have shown a similar prevalence of granulomas and apical cysts (Spatafore *et al.* 1990, Melo *et al.* 2004, Ribeiro *et al.* 2004). Healing of apical periodontitis in 80–95% cases after root canal treatment alone suggest that cysts may heal without surgery. By contrast, a few studies based on meticulous serial sectioning of periapical lesions retrieved *in toto* have shown that the actual incidence of radicular cysts is approximately 15% of all periapical lesions. In this context, Simon (1980) and Nair (1998) revealed that 'there are cysts and cysts'. The 'true cyst' is an inflammatory apical lesion with the cavity totally covered by stratified squamous epithelium, containing fluid or semi-solid tissue in its lumen and without an opening or connection with apical foramen or root canal. The apical true cyst is less likely to be resolved without surgical intervention. On the other hand, those containing epithelial-lined cavities that are open to the root canals and named 'bay cysts' or 'periapical pocket cysts' may heal completely after endodontic treatment. The prevalence of 'true cysts' is probably around 9.0%, explaining some apical refractory lesions (Simon 1980, Nair *et al.* 1996, Nair 1998).

Clinically and radiographically, it is impossible to differentiate granulomas and cysts as well as true apical cysts and pocket cysts, or whether the epithelial tissue is inert or proliferating. Biologically, many hypotheses have been proposed about the mechanisms of cyst healing after root canal treatment. Examples include the mechanical action of endodontic instruments upon cyst walls inducing healing (Bhaskar 1972) and enzymatic mechanisms (Catanzaro-Guimarães & Alle 1973). There is only one prospective clinical study that investigated the healing response of cystic lesions by electrophoretic analysis of fluid withdrawn after nonsurgical root canal treatment (Morse *et al.* 1973). Nowadays there is strong evidence to suggest that the immunological system contributes to the

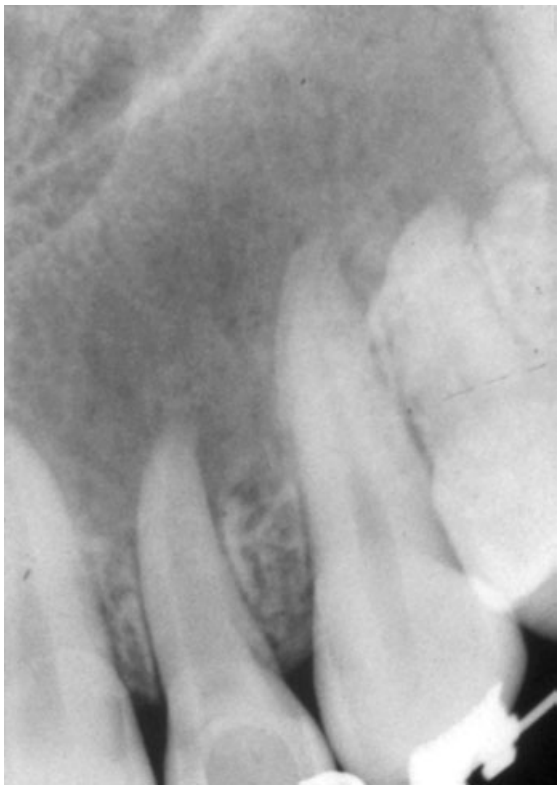

Figure 6 Periapical lesion at 7 months follow-up.

breakdown of epithelial radicular cyst linings which concomitantly removes or neutralizes the intracanal antigenic source. Rocha (1991) noted different patterns of T-lymphocytes and Langerhans cell arrangements in epithelial lining. Melo *et al.* (2004), Kettering & Torabinejad (1993) and Callestini (1996) suggested the participation of Langerhans cells, natural killer cells (NK cells) and macrophages in the cystic structure of apical lesion breakdown respectively.

The success of this conservative treatment was based on appropriate cleaning, shaping, antisepsis and filling of the root canal. A calcium hydroxide paste dressing was appropriate as it removes remaining microorganisms from the root canal system (Soares *et al.* 2005), may promote periapical repair, by: (i) controlling the inflammatory reaction (by hygroscopic action; calcium proteinate bridge formation and phospholipase inhibition); (ii) neutralizing osteoclast acid products (acid hydrolases and lactic acid); (iii) inducing cellular differentiation; (iv) inducing mineralization (alkaline phosphatase activation and calcium-dependent ATPases) and (v) neutralization of endotoxins (Tronstad *et al.* 1981, Seux *et al.* 1991, Safavi & Nichols 1993). As intracanal calcium hydroxide is progressively removed through its solubility in circulating periapical fluid, periodic renewal of calcium hydroxide is therefore of fundamental importance. In a physical, chemical and biological context, the related events included in the recovery of this extensive periapical lesion might be: (i) effect of biomechanical preparation on intracanal microbiota; (ii) lesion decompression established by apical patency by mechanical opening of the root foramen; (iii) complementary antiseptic action of calcium hydroxide because of alkalinity; (iv) action of calcium hydroxide on bony repair; and (v) action of immune system on epithelial component of the lesion.

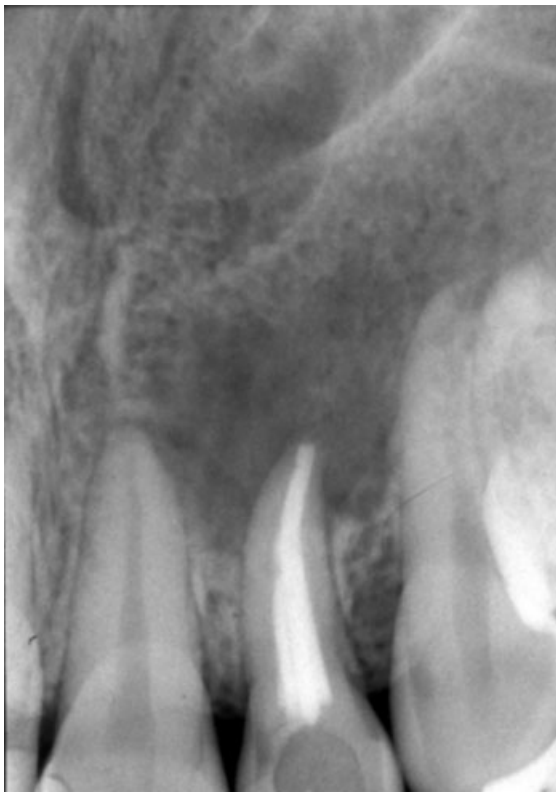

Figure 7 Periapical repair at 12 months follow-up.

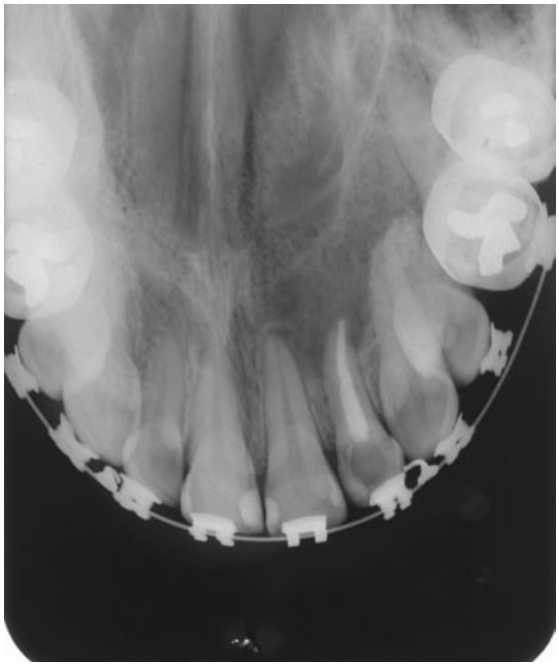

Figure 8 Advanced periapical bone repair at 14 months after root canal filling.

Conclusion

An extensive periapical lesion with the clinical and radiographic features of an apical cyst may respond to nonsurgical treatment involving biomechanical preparation, followed by lesion decompression by intracanal aspiration, associated with long-term renewal of aqueous calcium hydroxide paste and conventional obturation.

Disclaimer

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