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Case Report

# Favorable response of an extensive periapical lesion to root canal treatment

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**Abstract: This article presents non-surgical resolution of an extensive periapical lesion of endodontic origin associated with the maxillary left lateral incisor. Clinical examination revealed an asymptomatic bony hard swelling confined to the palate, while radiographic analysis showed a lesion measuring 22 mm in diameter and nearly 389 mm$^2$ in area. Through apical patency, 4 ml of intracanal exudate was drained. After thorough biomechanical preparation, a calcium hydroxide/CPMC root canal dressing was applied and periodically renewed for 11 months. The exudate was eliminated at treatment onset and significant bone formation was observed at the periapical region in the following months with concomitant resolution of the cortical expansion. Complete radiographic resolution of the periapical lesion was observed two years after the root canal filling. Thus, non-surgical treatment of this supposedly cystic, extensive periapical lesion provided favorable clinical and radiographic response. (J. Oral Sci. 50, 107-111, 2008)**

Keywords: calcium hydroxide; periapical cyst; root canal treatment.

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## Introduction

From a microbiological perspective, after pulp necrosis, the root canal system becomes increasingly susceptible to colonization by the microorganisms that inhabit the oral cavity and interconnected systems (1). Due to the close physiopathological relationship between the pulp and the periapical region, bacteria, fungi, and cell components may trigger an inflammatory process in periapical tissues, progressively affecting them through the resorption process (2-4). Subsequently, immunopathological mechanisms lead to the formation of abscesses, granulomas and periapical cysts (5,6). Nair et al. (7) histologically analyzed a sample of 256 periapical lesions and found that 35.0% were abscesses, 50.0% were granulomas, while only 15.0% were cysts; nonetheless, 52.0% of the lesions had an epithelial component in their structure.

Supposedly cystic periapical lesions may undergo asymptomatic evolution and reach large dimensions, clinically leading to cortical expansion and displacement of roots with consequent crown crowding. At this stage, the cortical plates of the alveolar process have a paper-like texture upon palpation; at a later stage, they may present fluctuation, and the mucosa may exhibit bluish discoloration (8). Radiographically, these plates display a round or elliptical contour, involve the apical third, and are delimited by a continuous radioopaque hard lamina (9).

From a therapeutic standpoint, most cases can be managed with a combination of endodontic and periapical surgical treatments. This article presents the resolution of an extensive, supposedly cystic, maxillary lesion exclusively

by means of endodontic treatment.

## Case Report

The patient, 32 years of age, of black ethnicity, attended the Endodontics Clinic of UFVJM for endodontic treatment of the maxillary left lateral incisor. The patient reported recent occurrence of swelling and pus, which was resolved by emergency treatment provided at a public health center. Extraoral examination was unremarkable. Intraoral assessment revealed an extensive coronal restoration with composite resin and consistent expansion of the palatal cortical plate with no pain on palpation. Electronic (Analytic Technology Pulp Tester, Sybron Endo, Orange, CA, USA) and thermal pulp sensitivity testing (Endo-ice, The Hygienic Corporation, Cuyahoga Falls, OH, USA) were negative only for the referred tooth. Periapical radiographic examination revealed a well-defined periapical radiolucent area with a round contour, which involved the apical region of teeth 11, 12, and 13 (Fig.1), measuring 22 mm in diameter and nearly 389 $mm^2$ in area, according to the software Image Tool Utscha 3.0. The clinical history, coupled with clinical and radiographic examination, led to the presumptive diagnosis of extensive, supposedly cystic, chronic periapical pathology of endodontic origin. Thus, endodontic treatment was initially proposed, with the possible need for complementary surgical intervention at a later stage. The patient consented to the treatment plan.

At the first session, after local anaesthesia, a rubber dam was set in place and endodontic access was performed, followed by neutralization of the infected root canal contents with a Kerr file, under irrigation with 5.25% sodium hypochlorite solution (NaClO). After achieving apical patency with a Kerr file #20 (Fig. 2), there was a spontaneous discharge of nearly 4 ml of citrine yellow serous exudate. Intracanal aspiration extracted another 1 ml of bloody serous exudate. Subsequently, biomechanical preparation was performed by the crown-down technique, under copious irrigation with 5.25% NaClO. After drying, a calcium hydroxide paste was placed in the root canal (Calen/CPMC - S.S. White, Rio de Janeiro, RJ, Brazil). Calen/CPMC paste is composed of 2.5g calcium hydroxide, 0.5 g zinc oxide, 0.05 g colophony, 2 ml polyethylene glycol 400, and 0.04 g camphorated paramonochlorophenol (CPMC). The root canal dressing was renewed 6 times in 11 months; as demonstrated in Figs. 3 and 4, during which period a progressive involution of periapical radiolucency occurred. No root canal exudate was observed after the second change of the root canal dressing. Before root canal filling, microbiological samples were collected and incubated under anaerobic conditions in a culture medium (Brain Heart Infusion - BHI). The samples showed negative microbiological growth. Root canal obturation was performed with AH-Plus sealer (Dentsply, Rio de Janeiro, RJ, Brazil) and gutta-percha points (Odous, Belo Horizonte, MG, Brazil) using the thermomechanical compaction technique, followed by a coronal restoration with composite resin (Fig. 5). After a two-year follow-up period, the patient was asymptomatic. Moreover, radiographic examination revealed complete resolution of the periapical radiolucency (Fig. 6).

## Discussion

Epithelial islands at the apical region of teeth with pulp necrosis may be stimulated by the inflammatory process and progress from epithelial rests of Malassez (7) to periapical cyst formation. Cystic development is common and accounts for 7 to 54% of periapical radiolucencies (8).

Radiographically, the mean diameter of periapical lesions ranges from 5 to 8 mm (4,6,10). Radiolucent areas larger than 10 mm are interpreted as possible apical cysts (8,11,12), the progressive growth of which are associated with the high osmotic pressure in their structure due to epithelial proliferation and degeneration (13).

Several decades ago, many endodontists, pathologists, and maxillofacial surgeons considered that apical cysts did not respond favorably to isolated endodontic treatment, thus requiring periapical curettage (12). However, surgical treatment of all periapical pathologies and/or large periapical lesions is not often necessary, since they may respond satisfactorily to adequate endodontic treatment (5,14).

Teeth with pulp necrosis associated with periapical radiolucent areas are similarly treated, regardless of the histological diagnosis of abscess, granuloma, or apical cyst. Based on their histological structure and relationship with the tooth apex, Simon (15) and Nair (13) stated that there are ‘apical true cysts’ and ‘apical pocket cysts’. The latter, also called ‘bay cysts’, have epithelial cavities directly communicating with the root canal system via the apical foramen; thus, they would completely heal after adequate root canal treatment. However, the ‘true apical cysts’ (13,15) would represent an inflammatory apical lesion whose cavity is completely lined by a stratified squamous epithelium and contains a liquid or semi-liquid, with no opening or connection with the apical foramen or root canal. Therefore, these cysts would not respond favorably to endodontic treatment, explaining why some periapical lesions are refractory to endodontic treatment even though such an assumption has been scientifically questioned (8).

The success of the conservative treatment of supposedly cystic periapical lesions could be explained based on the

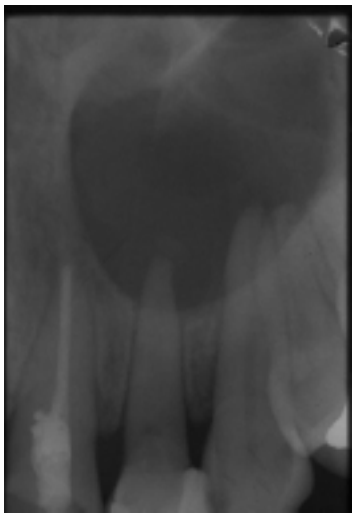

Fig. 1 Extensive periapical lesion with elliptical contour, associated with the maxillary left lateral incisor.

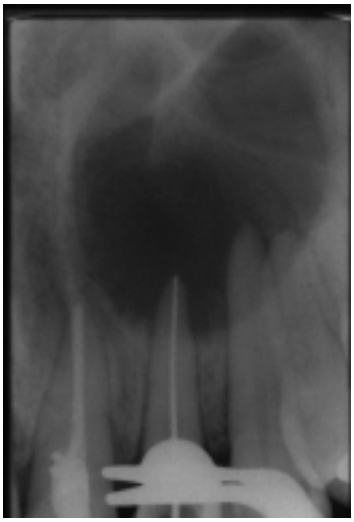

Fig. 2 Achievement of apical patency with file K #20.

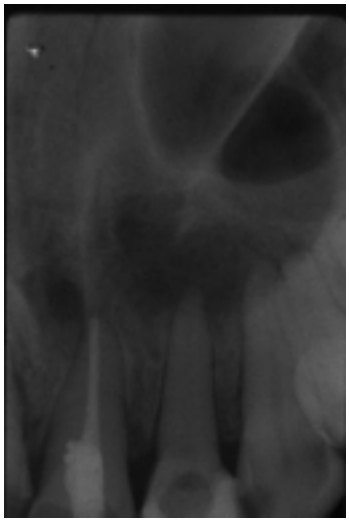

Fig. 3 Root canal dressing at five months; note the diffuse bone formation in the periapical region.

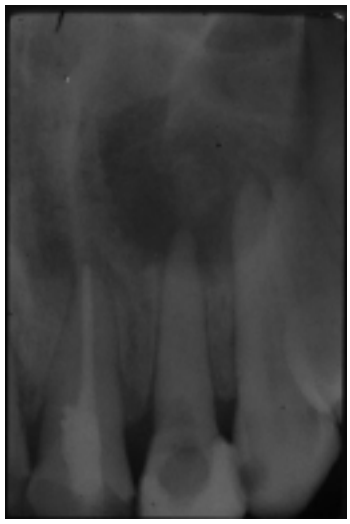

Fig. 4 Progressive involution of the periapical lesion at 8 months.

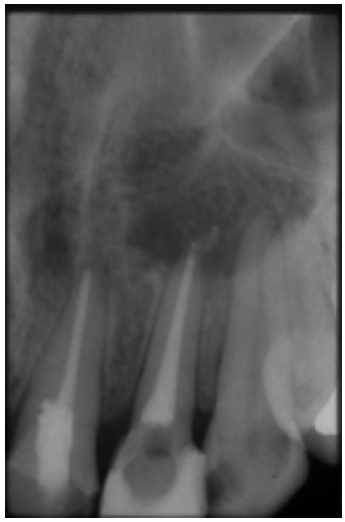

Fig. 5 Root canal filling at 11 months. Note advanced repair in the periapical region.

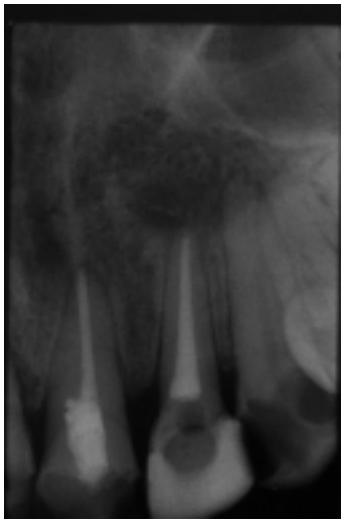

Fig. 6 Follow-up at 24 months after root canal filling demonstrating advanced repair of the periapical lesion.

following aspects: a) the effect of biomechanical preparation on intracanal microbiota; b) lesion decompression established by apical patency; c) complementary antiseptic action of calcium hydroxide due to alkalinity; d) effect of calcium hydroxide on bony repair; and e) effect of the immune system on the epithelial component of the lesion.

Therefore, effective neutralization and/or removal of infection from the root canal system would lead to non-surgical resolution of the apical cystic lesions. Adequate biomechanical preparation with the aid of 5.25% NaClO is currently recommended for the above purpose, followed by placement of a calcium hydroxide root canal dressing (3,9,20). The favorable clinical, radiographic, and histological responses obtained with calcium hydroxide are attributed to the involvement of $Ca^{2+}$ and $OH^-$ in several cellular and molecular mechanisms leading to regeneration of periapical connective tissues (9). The benefits of calcium hydroxide include anti-inflammatory action through its hygroscopic properties, such as forming calcium-proteinate bridges and inhibiting the phospholipase; the neutralization of acidic products, such as hydrolases of clasts; activation of alkaline phosphatase; and antibacterial action (16-19).

For quite some time, the Calcium hydroxide/CPMC combination has been applied in teeth with incompletely formed apices (20,21) in which CPMC acted as a vehicle. The cytotoxicity of CPMC is dose-dependent (22). In contrast with the Kaiser paste, CPMC is added to calcium hydroxide in residual quantities (0.04g), with polyethylene glycol 400 as vehicle in the Calen/CPMC paste. From a physical-chemical standpoint, such an association provides greater diffusion into the dentinal tubules and branches of the root canal. It also presents greater flow and longer action time due to the progressive release of calcium and hydroxyl ions from calcium para-chlorophenolate, which is the salt formed by the reaction between calcium hydroxide and the CPMC. In addition, the residual presence of CPMC results in a wider antimicrobial spectrum (23), an inflammatory reduction through the diminishing of free oxygen radicals (24), and proliferation of fibroblasts and osteogenic bone marrow cells (25).

The apical patency provided decompression of the periapical lesion upon the discharge of the inflammatory exudate through the root canal. Moreover, the removal of the source of antigen, associated with the regular renewal of the calcium hydroxide root canal dressing, eliminated the exudate and provided significant resolution of the periapical radiolucency. The efficacy of calcium hydroxide, owing to its antiseptic, anti-exudative, and mineralization-inducing properties, depends on the sustained release of calcium and hydroxyl ions to the root canal system and periapical region (26). As they are progressively resorbed by the periapical fluids, regular renewal of the root canal dressing is fundamental in reducing the intensity of the periapical inflammatory process; transforming the inflammatory granulation tissue into reparative granulation tissue; and simultaneously inducing the differentiation of undifferentiated mesenchymal cells into reparative cells, e.g., fibroblasts, cementoblasts and osteoblasts (9,26). These are combined with the effect of the immunological system on the epithelial component of the supposedly cystic lesion (5,27,28).

Current evidence strongly suggests that the immunological system is able to cause collapse of the epithelial wall of apical cysts, provided the source of antigen is removed from the root canal system. Rocha (5) observed different lineages of T lymphocytes and Langerhans cells in the epithelial lining. Melo et al. (14), Kettering and Torabinejad (27), and Callestini (28) all suggested the participation of Langerhans cells, natural killer cells (NK Cells), and macrophages in the rupture of the cystic structure of periapical lesions. From a biological standpoint, the ordered occurrence of these events would explain the clinic and radiographic conservative resolution of extensive, supposedly cystic, periapical lesions of endodontic origin.

We were able to non-surgically treat the extensive periapical lesion exhibiting clinical and radiographic characteristics compatible with apical cysts, exclusively with endodontic treatment consisting of proper cleaning, shaping, apical patency, and antisepsis of the root canal system, and emphasis on the extended utilization of calcium hydroxide/CPMC root canal dressing.

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