Healing of large periapical lesions following nonsurgical endodontic therapy: Case reports

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The cases of two patients with large cystlike periapical lesions are presented. The lesions formed as a result of trauma to the associated teeth. Following conservative nonsurgical endodontic treatment, there was complete resolution of the lesions. These results suggest that the largeness of a periapical lesion does not mandate its surgical removal and that even cystlike periapical lesions heal following conservative endodontic therapy.

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Introduction

Trauma to a tooth can damage its pulp even if the crown and root are not fractured. The pulp may survive or undergo necrosis, depending on the severity of the trauma and the type of inflammatory reaction that follows. This reaction may lead to extensive destruction of the periapical tissue and an ensuing periapical lesion.

On the basis of histologic findings, chronic periapical lesions of pulpal origin are diagnosed as either periapical granulomas or cysts. In the past, large, chronic periapical lesions were generally managed by root canal treatment of the involved teeth and surgical excision of the periapical lesions. This was particularly true if the periapical lesion was suspected to be a cyst. Now, because of improvements in conventional endodontic therapy and a better understanding of the healing potential of periapical tissues, fewer patients need periapical surgery.

Presented here are two cases of periapical lesions of the maxilla that were successfully treated by nonsurgical endodontic therapy. This article suggests that surgical removal of periapical lesions of pulpal origin is not mandatory, and that, irrespective of the size of the lesion, every effort should be made to treat such lesions by conservative means.

Case reports

Case 1

A 15-year-old boy fractured his maxillary central incisors in a playground accident 5 years previously. At that time he did not seek any treatment for the fractured teeth. Three days before he reported for treatment, he felt a dull pain in the fractured teeth, and on the following day the upper lip and right cheek became swollen and painful.

Extraoral examination revealed a diffuse swelling on the right cheek and upper lip. Intraoral examination showed discoloration, fractured, and tender maxillary right central and lateral incisors. The maxillary right canine and premolars were also tender. Periapical radiographs showed a large radiolucent lesion with a well-defined margin around the apices of all the tender teeth with the exception of the maxillary left central incisor (Figs 1a and 1b). All these teeth were insensitive to thermal and electrical pulp testing. When the root canals of the nonvital teeth were opened, suppurative

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fluid flowed out. This fluid, which was examined under the microscope, was found to contain cholesterol crystals.

The canals of these teeth were dried and debrided and the access openings were closed with provisional restorations. Five days after the root canals were opened, the facial swelling completely subsided. During the next 3 weeks the patient was examined weekly, and in each visit the canals were debrided and dried. The root canals were obturated with gutta-percha and the patient was reviewed every 3 months. Twelve months after the completion of the root canal therapy, there was complete resolution of the lesion (Figs 1c and 1d).

Case 2

A 25-year-old woman was referred for treatment of a gradually increasing swelling in the palate of 4 weeks' duration. Three years previously, in an automobile accident, the patient's upper lip and maxillary incisors were injured.

Examination revealed a soft, slightly tender swelling in the palate just behind the central incisors (Fig 2a). The maxillary incisors were nonvital and periapical radiographs showed two separate, well-defined radiolucent lesions around their apices (Fig 2b). When the root canals of the nonvital teeth were opened, straw-colored fluid flowed out. On compression of the palatal
swelling, more fluid was expressed through the canals. The fluid contained cholesterol crystals.

The canals were debrided and dried and the access openings were closed with provisional restorations. A week later the canals were once again debrided, dried, and obturated with gutta-percha. At this time the palatal swelling had completely subsided; however, the area was still soft as if there were no bone under the palatal mucosa. The patient was reviewed every 3 months, and, 15 months after completion of endodontic therapy, there was complete resolution of the lesions (Figs 2c and 2d).

Discussion

The precise mechanism involved in the formation of periapical lesions is not fully understood. Nevertheless it is generally agreed that if the pulp becomes necrotic, its environment becomes suitable to allow microorganisms to multiply and release various toxins into the periapical tissues, initiating an inflammatory reaction and leading to the formation of a periapical lesion. Several studies have been carried out to examine the role of bacteria in the formation of periapical lesions, and it has been observed that microorganisms are present in root canals of all teeth associated with periapical lesions. The endodontic flora consisted of
a mixture of rods, cocci, spirochetes, and filamentous forms. However, only a few periapical lesions have shown the presence of bacteria within the body of the lesion. One study even demonstrated an absence of bacterial colonies in all 18 periapical lesions examined except for the occasional isolated intracellular bacteria, which the investigators believed to be bacteria phagocytosed by macrophagelike cells.

In another experiment, periapical lesions were produced by injecting sonic extracts of cariogenic bacteria into pulps. Consequently, the inflammatory reaction in the periapical lesion may resist the spread of bacteria by confining them within the root canal, and periapical lesions may be caused not necessarily by microorganisms alone, but by other factors such as bacterial products and decomposition products of necrotic pulp. Immunopathologic mechanisms may also play a role in the initiation of periapical lesions. This may be assumed because of the presence of substantial quantities of immunologically competent cells and various immunoglobulins within the lesion.

The histogenesis of periapical cysts is uncertain, but remnants of the epithelial cell rests could possibly be the source of the epithelium in cyst lining, and various factors may contribute to the growth of periapical cysts. The inflammation in the periapical region may stimulate the proliferation of epithelium, which, by acting as a semipermeable membrane, could draw fluid into the cyst cavity by osmotic pressure. Bone-resorbing factors such as prostoglandins could contribute to further expansion of the cyst.

A comprehensive radiographic and histopathologic evaluation of periapical lesions showed that cysts are the most common periapical lesion. Depending on the radiographic area, diameter or length, from 53% to 100% of periapical lesions were cysts. Among periapical lesions with an area greater than 200 mm², 92% or more were cysts. Thus, the larger the periapical lesion, the greater the chance for it to be a cyst. In case 1, the radiographic area of the periapical lesion was more than 200 mm². On the basis of its size, the presence of cholesterol within the lesion contents, and the well-defined radiographic margin, the lesion was probably a periapical cyst. Because the treatment consisted of only conservative endodontic therapy, histopathologic examination of the lesion was not possible.

One concept for the treatment of periapical cysts was to treat the involved teeth endodontically and surgically excise the cyst lining. Other reports suggest that periapical cysts heal spontaneously after conservative endodontic therapy. The exact mechanism by which periapical cysts heal is not clearly understood. One theory is that as healing takes place collagen depositions compress the capillaries supplying the cyst, thereby cutting the blood supply to the epithelial lining. The lining degenerates and is then removed by the macrophages. Bhasker suggested that, if root canal therapy is extended beyond the apical foramen, the inflammatory reaction that develops destroys the cyst lining and converts the lesion into a granuloma. Once the causative factors are eliminated, the granuloma heals spontaneously.

Healing of periapical lesions following endodontic therapy has been compared to the healing of extraction sockets. But, unlike periapical cysts, extraction sockets do not have an epithelial lining that has to be eliminated during healing. Therefore these two lesions may not be comparable. Once the causative factors of a periapical lesion are removed, tissue-forming cells replace lost bone with new bone, while resorbed cementum and dentin are replaced by only cementum. Healing is completed when apical periodontal ligament restores its normal architecture. A periapical scar, which appears as a residual radiolucent periapical lesion, may sometimes form. The scar may be ignored if a continuous apical periodontal ligament of normal thickness is present around the involved teeth.

The periapical lesions in both patients were large and after nonsurgical endodontic therapy the lesions completely resolved without any scar. Mandatory surgical excision of cysts greater than 20 mm in diameter, as recommended previously, may not be necessary. The periapical tissues have a rich blood supply, lymphatic drainage, and abundant undifferentiated cells. All these structures are involved in the process of inflammation and repair. Therefore, because the periapical tissues have the potential to heal, treatment of periapical lesions should be directed toward only removal of the causative factors.

References