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Apexification of an Infected Untreated Immature Tooth

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A case of a traumatized permanent young tooth in which apexification occurred without the benefit of endodontic treatment is reported. It illustrates how a immature incisor that underwent pulp necrosis and periapical inflammation could progress to complete apical formation.

From the effect of dental trauma a permanent young tooth can lose pulpal vitality, halting radicular growth and causing tooth formation to be incomplete. The necrotic pulp acts as an irritant to the periapical tissues. The apical foramen remains open in a tubular or blunted form. Stimulating radicular development may close the apex and provide for suitable radicular canal obturation.

Various techniques induce the apexification process (1). Nygaard-Ostby (2) observed that after purposely lacerating the apical tissues in order to produce a blood clot a cementum-like tissue is deposited. To achieve apical closure, Bouchon (3) utilized Walkoff's paste. Kaiser (4), in 1956 and 1964, obtained apexification with calcium hydroxide paste. Frank (5) and Heithersay (6) attained successful results when calcium hydroxide in various forms was introduced in canals of immature teeth.

There are two views of the biological phenomena of apexification. The first suggests that is not necessary to introduce any type of chemical activator into the canal to stimulate the production of cementum and the genetic memory of the tooth. The elimination of residues and bacteria should be sufficient stimulus to react the cells responsible for completing radicular formation. Torneck and Smith (7) in recently erupted monkey's teeth have shown the histology of the radicular development process that occurs after pulpotomies and pulpectomies without medication or root filling. They concluded that root formation may continue without medicinal therapy, even though the growth may be retarded and irregular. Calcified bridge formation at the apex appeared to be related more to the ingrowth of trabeculae bone than to the deposition of dental hard tissues.

The second hypothesis advances the idea that apexification is a natural process, but should be stimulated by a biological activator, commonly calcium hydroxide. Steiner and Van Hassel (8) in 1971 used a mixture of calcium hydroxide and camphorated monochlorophenol to induce apical closure in devitalized immature teeth and described the radiopaque material bridging the foramen as cementum. Dylewski (9) stated that the calcified material that forms at the apex was a proliferation of connective tissues whose differentiation resembles osteodentin, which becomes continuous with the predentin at the apex.

It seems interesting in view of the foregoing to report an unusual case of apexification in which the apical portion of an immature incisor completed apical closure without any root treatment or benefit of calcium hydroxide (10).

CASE REPORT

An 8-yr-old boy suffered painful symptoms in his left central incisor and required evaluation and treatment. He had a history of having suffered a traumatic incident the previous year, with dislocation and nonpenetrating enamel dentin fracture in the tooth. A radiograph taken immediately after the incident showed the tooth

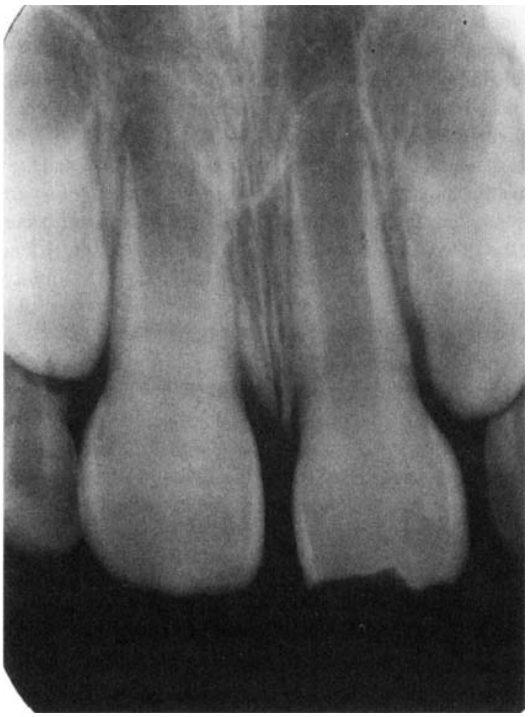

FIG 1. Radiograph of the left central incisor obtained immediately after the incident. Note the incisal fracture involving dentin and its wide root canals with incomplete apices.

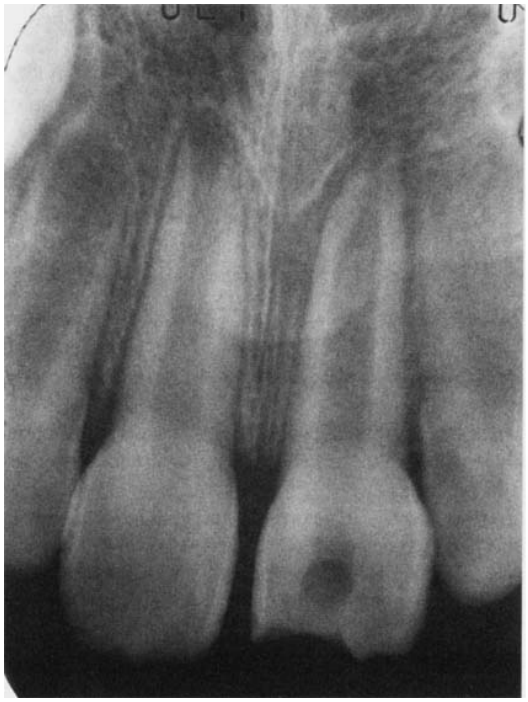

FIG 2. Radiograph obtained approximately 10 months after the incident. An apexification process had occurred despite pulp necrosis.

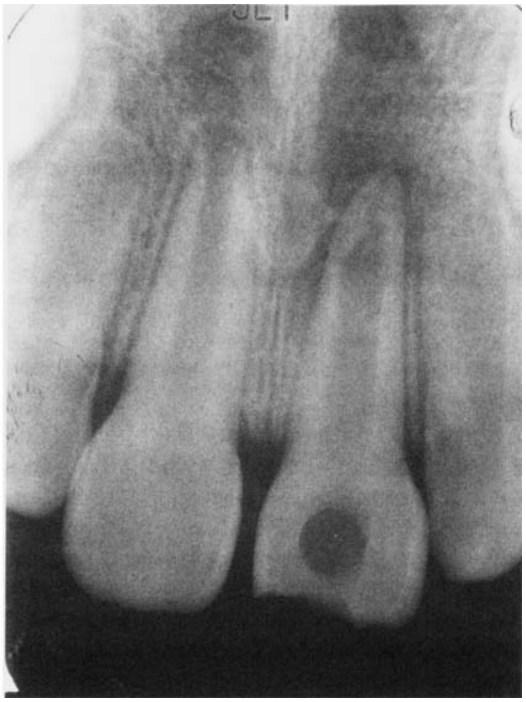

FIG 3. Radiograph showing wide canal in tooth 9; apexification and a periapical lesion present.

was in position; the root canals of this tooth and the adjacent teeth were wide and the apices incompletely formed (Fig. 1). No treatment was performed at that time. Approximately 10 months later a fluctuant region developed over the tooth. It was acutely tender to percussion. Drainage was established by lingual access in the pulp chamber. A radiograph taken at that time revealed diffuse periapical radiolucency, apical closure, and the persistence of a wide canal in the coronal two-thirds (Fig. 2).

The patient did not return for endodontic treatment and the canal remained open and exposed to the oral environment for a period of 6 months, during which time another flare-up episode occurred.

At the time of our examination, the tooth was slightly tender to palpation in the buccal vestibule and exhibited (+1) mobility. The area was found to be reddened. The access was not sealed and there was debris in the interior of the chamber.

A radiograph taken at the time of our examination revealed the apex to be closed and occupied by hard tissue (Fig. 3). Root length was approximately that of its contralateral counterpart. The neighboring incisor showed radicular development appropriate for the developmental stage. Confirming the radiograph evidence of closure, a #40 H-type file encountered resistance at the level of the root apex and failed to penetrate the periapical tissues (Fig. 4). Thin canal walls with scarce and irregular dentin deposits could be seen. Conventional endodontic treatment was begun on the affected tooth.

DISCUSSION

This case is an example of immature root apex subsequent to trauma and oral contamination without endodontic induction. It seems that the magnitude of the injury caused a severe pulp disease that progressed to the periapical tissues. Despite this complication, apical closure finished completely, probably due to residual odontogenic cells of the apical portion of the pulp that remained vital and active. A similar situation could have occurred with Hertwig's radicular sheath. The good blood supply that characterizes young permanent teeth could be responsible for the permanence of these odontogenic structures that could have been involved in the apexification of the incisor.

Torneck et al. (11, 12) reported that in monkeys incompletely formed incisors exposed to oral contamination after removing dental pulp the type and pattern of the formation of hard dental tissues—specifically dentin—vary according to the degree of the injury undergone by the residual cells. In general, the greater the injury, the more irregular and disorganized the calcified tissue (13). In our case, parietal dentin formation was quite meager and irregular, leaving two-thirds of the canal wide open, perhaps due to the severe destruction of pulp at this level. However, in the apical region, there were normal deposits of dentin, which indicates that apical pulp vitality remained for a long period of time after the injury. These facts support what Torneck et al. (12) observed during this time period. The cementum deposits have been attributed to the histodifferentiation into cementoblasts from undifferentiated cells in the immature clot (2) or to a metaplasia of mesenchymal and fibroblastic cells of residual pulp that remain viable despite the injury and the ensuing inflammation.

The pattern and type of the apical closure were normal, even though it occurred earlier than in its homologous tooth. The stimulus that accelerated the apical closure could have been due to the injury itself, or to the humoral components of the inflammation after trauma. The closure in such case could be due to inward bending of the Hertwig's radicular sheath, and the "bridge effect" to the deposits of hard tissue. However, other authors have proposed that this effect could be more a growth of osseous tissue into the apical portion of the canal than a deposit of hard tissues. It can

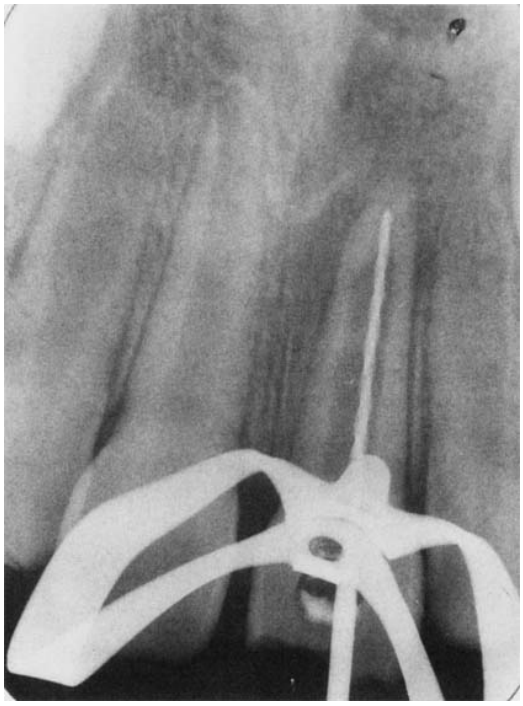

FIG 4. A #40 H-type file verifying hard tissues deposits.

be concluded from this clinical case that despite the presence of pronounced inflammatory changes in both residual and periapical tissue root growth and foraminal closure may occur. It is possible that the mechanisms of apexification could be related to a genetically determined pattern and not to a specific treatment.

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