Clinical, Radiographic, and Histological Observation of a Human Immature Permanent Tooth with Chronic Apical Abscess after Revitalization Treatment

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Abstract

Introduction: Revitalization procedures have been widely used for the treatment of immature permanent teeth with apical periodontitis. The treatment procedures appear to be capable of encouraging continued root development and thickening of the canal walls. The nature of tissues formed in the canal space and at the root apex after revitalization has been shown histologically in several animal studies; similar studies in humans were recently reported. Methods: A 9-year-old boy had a traumatic injury to his upper anterior teeth. Tooth #9 suffered a complicated crown fracture with a pulp exposure, which was restored with a composite resin. The tooth developed a chronic apical abscess. Revitalization procedures were performed on tooth #9 because it was an immature permanent tooth with an open apex and thin canal walls. Twenty-six months after revitalization, the tooth had a horizontal crown fracture at the cervical level and could not be restored. The tooth was extracted and processed for routine histological and immunohistochemical examination to identify the nature of tissues formed in the canal space. Results: Clinically and radiographically, the revitalization of the present case was successful because of the absence of signs and symptoms and the resolution of periapical lesion as well as thickening of the canal walls and continued root development. The tissue formed in the canal was well-mineralized cementum- or bone-like tissue identified by routine histology and immunohistochemistry. No pulp-like tissue characterized by the presence of polarized odontoblast-like cells aligning dentin-like hard tissue was observed. Conclusions: The tissues formed in the canal of revitalized human tooth are similar to cementum- or bone-like tissue and fibrous connective tissue. (J Endod 2013;39:1078–1083)

Key Words

Apical periodontitis, bone-like tissue, cementum-like tissue, immature permanent tooth, revitalization

Since the report of Iwaya et al (1) that a type of treatment termed “revascularization” was applied to treat human immature permanent tooth with apical periodontitis and sinus tract and resulted in not only resolution of apical periodontitis but also thickening of the root canal walls and continued root development, many human immature permanent teeth with apical periodontitis have been treated with revascularization procedures instead of traditional apexification procedures (2). It has been discussed that the term “revitalization” is more appropriate to describe such clinical treatment; therefore, revitalization will be used herein (3, 4). The nature of tissues formed in the root canals of immature teeth with apical periodontitis after revitalization was described histologically as mineralized tissue resembling cementum or bone and periodontal ligament-like tissue in several animal studies (5–8). The nature of the tissue formed in the root canals of human revitalized teeth was recently reported (9–11). We report clinical, radiographic, and histological observation of a human immature permanent tooth with chronic apical abscess after revascularization treatment.

Case Report

Diagnosis and Treatment Planning Visit

A 9-year-old boy was referred from the Postgraduate Pediatric Clinic to the Postgraduate Endodontic Clinic at New York University College of Dentistry for the evaluation of tooth #9. The patient had a history of trauma to the upper anterior teeth that occurred from a fall approximately 3 months ago. The patient’s chief complaint was “I hit my front teeth 3 months ago, and now I have an infection.” The patient did not report any symptoms. Clinical examination showed that tooth #9 had a complicated crown fracture with a pulp exposure, which was restored with a composite resin. A sinus tract was present in the apical area of tooth #9. The tooth was not sensitive to percussion and palpation. It also did not respond to Endo Ice (Coltene/Whaledent Inc, Cuyahoga, OH) or an electric pulp tester (Vitality Scanner; SybronEndo, Glendora, CA). Radiographic examination revealed a well-circumscribed large periapical radiolucent lesion measuring approximately 8 × 8 mm around the apex of tooth #9 (Fig. 1A). The immature tooth had thin canal walls and an open apex (Fig. 1A). The clinical diagnosis for tooth #9 was pulpal necrosis and chronic apical abscess. Treatment options including revitalization,
mineral trioxide aggregate apexification, and calcium hydroxide apexification were discussed with the mother and child, and after discussion the decision was made to perform a revitalization procedure.

First Treatment Visit
At the first treatment appointment, access to the pulp cavity was obtained under rubber dam isolation without administration of local anesthetic. Purulent exudate and bleeding were noted in the canal. A working length radiograph was taken and recorded at 23 mm. The canal was gently irrigated with copious amounts of 2.6% sodium hypochlorite (Sultan Healthcare, Hackensack, NJ) and dried with paper points. Calcium hydroxide (Henry Schein, Melville, NY) mixed with saline solution was used as an intracanal medication and placed into the apical portion of the canal. The access was closed with a sterile cotton pellet followed by an intermediate restorative material (Dentsply International, Milford, DE).

Second Treatment Visit
Eleven days after the initial treatment, tooth #9 was asymptomatic, and the sinus tract had closed. Local infiltration with 3% mepivacaine without vasoconstrictor was administered. The access cavity was reopened after isolation with a rubber dam. The root canal was irrigated with copious amounts of 2.6% sodium hypochlorite and dried with paper points. Bleeding was induced into the canal up to the coronal third by irritating the periapical tissues with a #30 K-file. Mineral trioxide aggregate (Dentsply Tulsa Dental, Johnson City, TN) mixed with saline solution was placed on the top of the partially coagulated blood clot. The access was sealed with a light-cured composite restoration (Amelogen Plus; Ultradent, South Jordan, UT) (Fig. 1B).

Recall Visits
At the 12-month recall appointment, the tooth was asymptomatic. There was radiographic evidence of resolution of the periapical lesion and thickening of the canal walls (Fig. 1C). Thus, this case was both an endodontic success in that the periapical lesion had healed and a revitalization success because there was obvious increased thickening of the canal walls and continued root development. Twenty-six months after the completion of the revitalization treatment, the patient presented to the Postgraduate Endodontic Clinic with a horizontal crown fracture at the cervical level of tooth #9 (Fig. 1D), and the tooth was determined to be nonrestorable. Radiographically, the tooth showed complete resolution of the periapical lesion and marked thickening of the canal walls as well as closure of the root apex (Fig. 1E). Tooth #9 was extracted and processed for routine histological and immunohistochemical examination.

The primary mouse monoclonal antibodies were used against human bone sialoprotein (BSP), dentin sialoprotein, and neurofilament 200 N52 (LifeSpan Biosciences, Inc, Seattle, WA). The avidin-biotin

Figure 1. (A) Preoperative radiograph: tooth #9 exhibits incomplete formation of the root. Periapical radiolucency is present. (B) Postoperative radiograph: the periapical radiolucency area appears to be larger than the preoperative lesion, with sharp margins. (C) Follow-up radiograph taken 12 months after revitalization: the periapical radiolucency has resolved with only slight thickening of the periodontal ligament around the root apex. The canal space is reduced in size and the thickness of the canal walls is increased. (D) The patient presented after 25 months because the crown was mobile. A complete crown fracture was diagnosed, and the tooth could not be restored. Clinical view after removal of the fractured crown. (E) Radiograph taken before removal of the fractured crown. Thickening of the root canal walls increased further. The periapical lesion completely resolved.
complex staining kit was obtained from Santa Cruz Biotechnology, Inc (Santa Cruz, CA). The routine histological and immunohistochemical staining procedures were described in our previous study (9).

Quantification of Radiographic Changes in the Root Length and Area at the 12-month Recall

Images were analyzed using ImageJ (National Institutes of Health, Bethesda, MD). The preoperative and 3-month recall films were aligned using the TurboReg Plug-In tool. (Unfortunately, the 12-month follow-up film was too elongated to use for comparison.) After alignment, the radiographic root width was measured by outlining the total root width and canal width at 3 discreet points (1 midroot, 1 at the apical third, and 1 in the apical fifth). The root length was measured by drawing a line from the most mesial aspect of the cemento-enamel junction (CEJ) as well as the most distal aspect of the CEJ to the center of the most apical portion of the root canal space. All measurements were completed twice, and the values were averaged. The percentage change in the measurements from the preoperative to the recall film was then calculated.

A comparison of the preoperative and postoperative films showed that a quantifiable increase in the radiographic area of the root was observed subsequent to the procedure. At the 3-month recall, we observed minimal change (mean = 2.3% increase, average change measured from the mesial and distal CEJ) in root length. However, we were able to measure more significant increases in root width (mean = 10.4% increase, average change measured at 3 discreet

![Figure 2](image-url)

Figure 2. (A) Section passing approximately at the center of the root canal. A mineralized tissue fills the apical portion of the canal (hematoxylin-eosin; original magnification ×16). (B) Detail of the apical portion in A. An island of soft tissue is present in the calcified tissue apically (original magnification ×25). The inset shows magnification of the ramification indicated by the lower arrow. Its lumen contains uninflamed connective tissue (original magnification ×400). (C) A high-power view of the apical soft tissue in B. Vital connective tissue with fibroblasts and abundance of collagen fibers. Absence of inflammatory cells (original magnification ×400). (D) Magnification of the area indicated by the upper arrow in B. The calcified tissue filling the apical canal is irregular and demarcated apically by a cementum-like tissue, with some osteoblast-like lacunae. Increased root length is caused by deposition of cementum-like tissue (original magnification ×400). (E) A high-power view of the area of the root canal wall indicated by the left arrow in A. From left to right: area with high concentration of dentinal tubules (tubules are cut transversally by the microtome blade), area with less tubules, and calcified tissue with no dentin tubules (original magnification ×400). (F) A high-power view of the area of the root canal wall indicated by the right arrow in A. From right to left: area with high concentration of dentin tubules (cut obliquely by the microtome blade), area with only few tubules, and calcified tissue with no dentin tubules (original magnification ×400).
Importantly, these findings suggest that the case should be considered a successful revitalization procedure because of the quantification of increased root width.

**Histological and Immunohistochemical Observations**

Histologically, the most apical portion of the canal was filled with newly formed mineralized tissue (Fig. 2A and B). Some uninflamed vital connective tissue was noted enclosed in the mineralized tissue (Fig. 2B and C). The newly formed mineralized tissue appeared to resemble cementum-like tissue with incremental lines and osteocyte-like lacunae (Fig. 2D). The demarcation between the canal dentin and the newly formed mineralized tissue could be easily recognized because of the absence of dentinal tubules in the latter tissue (Fig. 2E and F). The increased root length was caused by apical deposition of cementum-like tissue (Fig. 2B and D). In the midportion of the canal, the canal space was partially filled with mineralized tissue with pockets of necrotic debris (Fig. 3A, B, and D). Necrotic tissue was also present.

**Figure 3.** (A) Same section as that in Figure 2. Middle third portion of the canal. The canal appears empty at this level (hematoxylin-eosin, original magnification ×16). (B) Detail of the coronal portion of the canal in A. The canal is partly occupied by a newly formed calcified tissue (original magnification ×50). (C) A high-power view of the area of the left root canal wall indicated by the left arrow in B. From left to right: highly tubular dentin, calciotraumatic line and transition to a less tubular dentin, and necrotic debris and calcified tissue with no tubules (original magnification ×400). (D) A high-power view of the area of the calcified tissue indicated by the right arrow in B. Dystrophic calcification. Small lacunae containing necrotic debris (original magnification ×400). (E) Section taken approximately 50 sections from that shown in A. The apical canal appears totally obliterated by calcified tissue (original magnification ×16). (F) Detail of the foramina area in E. Vital tissue on the left. Necrotic debris on the right (original magnification ×100). The inset shows high magnification of the area indicated by the arrow. Two neutrophilic leukocytes can be recognized (original magnification ×400).
between the canal dentin and the mineralized tissue (Fig. 3C). There appeared to be a change of pattern in dentin formation before pulp underwent necrosis, probably because of the history of trauma (Fig. 3C). The apical canal appears obliterated by calcified tissue (Fig. 3E and F). No pulp-like tissue characterized by the presence of polarized odontoblast-like cells aligning the dentin-like tissue was observed. Immunohistochemical staining was only positive for BSP (Fig. 4A–C).

**Discussion**

In all animal studies, the tissues formed in the canal spaces after revitalization procedures of immature teeth are described as mineralized tissue and periodontal ligament-like tissue (5–8). The mineralized tissue resembles either cementum or bone. The tissues formed in the canal space of the present human case after revitalization procedures are similar to those observed in animal studies (5–8) and the tissues formed in the canal spaces of a previous human case report (11). Continued root development was caused by apical deposition of cementum-like tissue without dentin, which inferred likely survival of Hertwig’s epithelial root sheath (HERS) before completion of root formation. It is HERS, and not apical papilla, that regulates root development (12, 13). The apical papilla in the present case probably did not survive chronic apical abscess; otherwise, HERS would induce progenitor/stem cells in the ectomesenchymal apical papilla to differentiate into root odontoblasts and produce root dentin (12, 14).

It has been shown that HERS is capable of inducing progenitor/stem cells in the ectomesenchymal dental follicle to differentiate into cementoblasts during root development (13). The cellular and molecular biology of revitalization is still not fully understood (15).

Immunohistochemical staining was positive for BSP. The mineralized tissue formed in the canal space in the present case most likely was not dentin because of the absence of a tubule-like structure and might be well-mineralized cementum- or bone-like tissue. Although nerve fibers were not detected immunohistochemically, this does not mean that nerve fibers were not present because vital connective tissue and blood vessels were observed in the canal of the revitalized tooth. Nerves play an important role in maintaining tissue vitality, such as the control of blood flow (16), the response to injury (17), and the regulation of immune system (18).

Based on the present case and a previous case report (11) as well as animal studies (3–6), in situ regeneration of the pulp tissue in the teeth with necrotic pulp and apical periodontitis after revitalization treatment did not occur. This is probably because of the complete destruction of the parenchymal cells of the pulp tissue and the apical papilla as predicted previously (4). It has not been shown that in situ regeneration of tissues or organs is possible if the parenchymal cells were completely destroyed, at least in the revitalization of immature permanent teeth with apical periodontitis. However, in 1 study, pulp-like tissue was observed in revitalized tooth (10).
Regeneration of the dentin-pulp complex in the teeth with total necrotic pulp and apical periodontitis likely requires tissue engineering using a cell-based approach that includes the use of autologous pulp stem/progenitor cells, scaffold, and morphogens (growth factors) (19–21) (ideally, in a sterile root canal system). The exogenously introduced cells may be a heterogeneous or subpopulation of stem/progenitor cells. The current non–cell-based approach without introducing exogenous cells would result in repair rather than regeneration (22, 23). Some investigators are testing the use of chemotactic factors to induce homing by recruiting stem cells from other area of tissues into the root canal space for pulp regeneration (24). So far, no convincing pulp regeneration data have been reported in revascularization/revitalization of immature permanent teeth with apical periodontitis.

**Conclusion**

Based on clinical and radiographic observations, revitalization of the present case was successful because of the absence of signs and symptoms and the resolution of apical periodontitis as well as thickening of the canal walls and continued root development. The tissues formed in the canal space are similar to cementum- or bone-like tissue and fibrous connective tissue.

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**References**