Revascularization-associated Intracanal Calcification: Assessment of Prevalence and Contributing Factors

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Abstract

Introduction: Intracanal calcifications have been reported in endodontic cases after revascularization. The purpose of the current study was to determine the incidence of intracanal calcification and potential contributing factors in retrospective revascularization cases. **Methods:** Among 37 patients who had undergone revascularization between 2010 and 2014, 29 cases were assessed with average follow-up period of 24.9 months. Clinical and radiographic examinations were performed to evaluate the treatment outcomes, eg, resolution of apical periodontitis (AP), root development, and occurrence of intracanal calcification. Radiographic assessment revealed varied calcification patterns, which were classified into calcific barrier or canal obliteration, collectively referred to as revascularization-associated intracanal calcification (RAIC). **Results:** All 29 cases demonstrated resolution of AP, whereas continued root development with apical closure occurred in 23 of 29 cases (79.3%). RAIC was noted in 18 of 29 cases (62.1%), among which 5 of 18 cases (27.8%) were classified as calcific barrier and 13 of 18 cases as canal obliteration (72.2%). Higher frequency of RAIC was noted in the cases with induced bleeding (16 of 23 cases, 69.6%), whereas the 6 cases without induced bleeding showed RAIC at 33.4%. Also, RAIC occurred more frequently in cases medicated with $Ca(OH)_2$ (10 of 13 cases, 76.9%) than in those medicated with antibiotic pastes (6 of 13 cases, 46.2%). **Conclusions:** This study indicated that RAIC is common (62.1%) among cases treated with revascularization. Multiple contributing factors may include the type of medicaments and induction of intracanal bleeding. Although RAIC does not interfere with resolution of AP, some cases may progress to complete obliteration of root canals and would impede normal function of dental pulp tissues. *(J Endod 2017;■:1–9)*

Key Words

Bleeding induction, intracanal calcification, regenerative endodontics, revascularization, root development, type of medication

Regenerative endodontic procedure (REP) is one of the treatment choices for immature permanent teeth with pulp necrosis. After its introduction in 2004 (1), numerous studies have demonstrated successful clinical outcomes of REPs in teeth with apical periodontitis (AP) and periapical abscess (2–7).

Significance

This retrospective clinical study illustrates the characteristics, prevalence, and potential contributing factors of revascularization-associated intracanal calcification (RAIC). Findings indicate high prevalence of RAIC at 62.1% of revascularization cases and the progressive nature of this condition with respect to time.

The primary objectives of REPs are the resolution of AP, continued root development, apical closure, and/or positive responses to vitality testing. On the basis of current regenerative protocol from the American Association of Endodontists, revascularization is primarily accomplished through disinfection of the root canal by using $Ca(OH)_2$ or triple antibiotic paste (TAP) and induced bleeding into the canal, followed by placement of mineral trioxide aggregate (MTA) barrier and coronal restoration (8). Considering the triad of successful pulp regeneration, ie, infection control, biomaterials, and mesenchymal stem cells (MSCs), pulpal stem cells are required for pulp-dentin regeneration (9). With safety and regulations governing cell transplantation at present, a feasible source of MSCs into the root canal space is through induced bleeding, which may recruit MSCs from bone marrow and periodontal ligament (PDL) tissues.

Jeeruphan et al (10) reported survival rate of 100% in resolving AP after revascularization. Some studies also reported successful treatment outcomes that are based on radiographic assessment, which allowed for comparison of root dimensional changes before and after revascularization (11, 12). However, continued root development in immature teeth after revascularization is not a predictable outcome as compared with

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resolution of AP in part because the responses may vary and often occur with intracanal calcifications. Chen et al (13) suggested intracanal calcification as one of the healing outcomes, and various types of intracanal calcification/obliteration have been reported in several prior studies (2,13–15).

Although root canal calcification is not considered a diseased state, excessive calcification would impede vitality/function of revascularized tissues and complicate endodontic treatment if so indicated in the future (4, 16). In addition, complete obliteration of root canal space negates restoration of pulpal function with revascularization. In regard to the nature of calcification in the root canal space, large animal studies and histologic case reports indicate ectopic bone, cementum, and fibrotic tissue formation, resembling dystrophic calcification (17–21). The purpose of the current retrospective study was to determine the frequency and pattern of intracanal calcification in cases treated by revascularization and to identify potential contributing factors.

Materials and Methods

Case Selection

We recruited a total of 37 patients who had undergone revascularization at 3 academic institutions, UCLA School of Dentistry, Seoul National University School of Dentistry, and Yonsei University School of Dentistry, between 2010 and 2014. Healthy patients with at least 1-year follow-up and documented revascularization procedures were included, whereas those without sufficient clinical exam and/or lack of documentation in regard to induced bleeding during revascularization were excluded.

Treatment Procedure

All procedures were performed by endodontic faculty members and residents in the respective institutions. Revascularization was performed according to the methods prescribed by the American Association of Endodontists (8) with minor protocol variations, eg, type of medicaments, duration of antimicrobial treatment, and induced bleeding, which were based on the initial presentation of the cases and how the cases responded to the initial therapy. Briefly, at the first appointment, an access cavity was made under rubber dam isolation, and canals were debrided by using intracanal irrigation with 2.5% NaOCl. In some patients presenting with noted apical vital tissues, root canal instrumentation was limited to the level at which the vital tissue was detected. In these patients, intracanal bleeding was not performed during the second appointment. In these cases without induced bleeding (cases 1–6 in Tables 1 and 2), the cases are referred herein as revascularization as opposed to pulpotomy due to presentation of pulpal necrosis and chronic apical abscess. Intracanal medicament was then placed with ciprofloxacin, metronidazole, and minocycline (TAP), double antibiotics paste (DAP) (ciprofloxacin and metronidazole), or $Ca(OH)_2$ to the level of the cementoenamel junction (CEJ). Access cavity was sealed with temporary restoration, eg, Caviton (GC, Aichi, Japan) or IRM (Caulk Dentsply, Milford, DE).

During the second appointment, clinical signs/symptoms were evaluated; with continued clinical signs of infection, disinfection protocol and the medicament were repeated. For those without signs of infection, intracanal bleeding was induced after local anesthesia with 3% mepivacaine without vasoconstrictor by insertion of #10 endodontic K-file beyond the apical foramen. Blood clot was then formed to the level of the CEJ and verified under operating microscope. An MTA barrier was placed over the blood clot, and tooth was restored with temporary restoration. For those patients presenting with apical vital tissues at the first appointment, the canals were irrigated with 2.5% NaOCl, dried, and restored with MTA barrier below CEJ and a temporary restoration without inducing intracanal bleeding. All patients were brought in for the third appointment, during which time temporary restoration was replaced with permanent bonded resin after confirming MTA setting.

Clinical and Radiographic Assessment of the Treatment Outcomes

Patients were generally followed up once per 6–12 months after completion of revascularization. During every follow-up visit, clinical and radiographic examinations were performed; the clinical examination included signs and/or symptoms, mobility, tenderness to percussion/palpation, and vitality testing. Radiographic assessment was performed with periapical radiographs and/or cone-beam computed tomography (CBCT) as needed to evaluate for changes in the osseous healing pattern, root development pattern, and presence or absence of intracanal calcification. Because of concerns with exposure, CBCT was not routinely used as outcome measure.

We assessed resolution of AP through clinical and radiographic evaluations and classified each case into success and failure according to the criteria of Strindberg (22). Criteria for failure included persistence of clinical signs/symptoms and/or periapical radiolucency showing persistent AP. The criteria for success included absence of clinical signs/symptoms and radiographic evidence of resolution of AP. We also assessed the effects of revascularization on root development by noting thickness and length of the root and/or apical closure, as previously reported (10).

Radiographic assessment of intracanal calcification was performed independently by 2 endodontic faculty members (M.S., C.Y.) to determine the presence or absence of calcification per each case and to describe the pattern of calcification. Cases with resolution of AP without radiographically visible calcific bodies in the lumen of the root canals were observed, as exemplified in Figure 1; these cases were assigned "none" for intracanal calcification. In those cases that demonstrated calcific body formation, the pattern of intracanal calcification varied and was largely categorized into either calcific barrier (CB) or canal obliteration (CO). Collectively, we denoted these calcific bodies as revascularization-associated intracanal calcification (RAIC). CB generally formed along the mid-root area of the root canal space, leaving the remaining canal lumen still visible in the radiographs, as typified by the case shown in Figure 2*A*. Cases with CO generally show complete obliteration of the root canal space from the root apex to the orifice level, as shown in Figure 2*B*.

Results

Of the 37 revascularization cases performed at the 3 academic institutions from 2010 to 2014, 29 cases that had more than 12-month follow-up were included in this study. Table 1 summarizes the patient demographic data and the relevant clinical parameters, eg, preoperative symptoms, diagnosis, and radiographic findings. Etiology of pulpal disease was stratified into dens evaginatus (14 cases, 48.3%), trauma (6 cases, 20.7%), and caries (2 cases, 6.9%), with the remaining 7 cases (24.1%) with undocumented cause (Table 1). Age of the patients who received revascularization ranged from 8 to 18 years, with mean value of 12.4 years; 27 of 29 cases were diagnosed with pulp necrosis and varying degrees of periradicular presentation, which ranged from symptomatic AP and acute apical abscess to established chronic apical abscess with sinus tract. The vast majority of the cases presented with periapical abscess and distinct periapical radiolucency.

Among the 29 cases, $Ca(OH)_2$ was used as intracanal medicament in 13 cases, TAP/DAP was used in 13 cases, and the remaining 3 cases were treated with combination of $Ca(OH)_2$/TAP/DAP because