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CASE REPORT/CLINICAL TECHNIQUES

Reparative Endodontic Treatment of a Perforating Internal Inflammatory Root Resorption: A Case Report

SIGNIFICANCE

Cracked teeth with radicular extensions may have higher success rates than previously thought. Using modern endodontic techniques, placing deep intraorifice barriers, and following specific postoperative protocols may improve outcomes for these cases.

ABSTRACT

The aim of this case report was to present a reparative treatment approach of an extensive internal inflammatory resorption with a lateral perforation and apical and lateral inflammatory lesions. Only the necrotic coronal part of the pulp was removed, and the vital pulp tissue within the resorption cavity and the apical part of the root canal was left uninstrumented. Bleeding was induced, and the blood clot was covered with mineral trioxide aggregate. Hard tissue repair and healing of the apical lesion could be observed in the 3-year recall. *(J Endod 2021;47:146–155.)*

KEY WORDS

Intracoronal endodontic diagnostics; internal root resorption; odontoclast; reparative endodontic treatment; sinus tract

Internal root resorption (IRR) is characterized as a pathologic process with the loss of dental hard tissue caused by chronic pulpal inflammation[1,2]. In 1830, internal resorption in a mandibular permanent molar was described for the first time[3].

The resorption of dentin is mediated by odontoclasts, which are involved in the nonspecific cellular immune response to pathologic and physiological stimuli. These cells are similar to osteoclasts in their properties, structure, and mode of action. Osteoclasts develop from tartrate-resistant acid phosphatase–positive monocytes. The progenitor cells of odontoclasts are located in the dental pulp and the periodontal ligament and are able to express cathepsin D, tartrate-resistant acid phosphatase, and matrix metalloprotease 9[4–8]. Their activity is mediated via receptor activator of nuclear factor kappa B/receptor activator of nuclear factor kappa B ligand receptors[9]. Osteopontin can inhibit osteoclast activation[10], whereas natural inhibition occurs via osteoprotegerin[11,12], estrogen[13], calcitonin[14], and androgen[9].

After stimulation of cell differentiation and fusion of the progenitor cells to odontoclasts, they attach to mineralized dentin and produce a highly acidic milieu at a pH of approximately 4.5 to initiate the resorptive process[5]. However, osteoclasts cannot bind to demineralized tissue[15]. Odontoblasts and predentin prevent them from attaching and resorbing dentin[16]. Mesenchymal pulp cells are also able to prevent dentin resorption. It was demonstrated that pulp cells in combination with monocytes inhibit the genesis of osteoclasts[17].

Internal resorption can occur as a sequelae after acute traumatic injuries[1,18,19], chronic tooth trauma[20,21], or autotransplantation[22–24]; as a result of crown preparation with insufficient cooling[25], pulp amputation[26,27], revitalization[28,29], or hyperthyreoidism[30]; or as a result of orthodontic treatment[31]. A genetic predisposition was suspected in 1 case involving monozygotic twins[32].

IRRs are relatively rarely observed in clinical practice because they are mostly asymptomatic and are usually diagnosed just by chance during routine radiographic examination. The prevalence of patients with extensive IRR is between 0.01%–1%[8]. However, this is likely to be an underestimation because IRR can be easily overlooked. Internal resorptive destruction of dentin can be detected radiographically only in the advanced stage. Gabor et al[33] investigated 30 extracted teeth for the presence of internal resorptions. No resorption could be proven histologically in 9 teeth with healthy pulp. In contrast, 4 of 8 teeth with pulpitis and 10 of 13 teeth with pulp necrosis showed signs of internal resorption.

Damage to odontoblasts and the predentin layer resulting from dental trauma or microbial infection seems to be a precondition for the occurrence of internal resorptions[34]. A primary internal resorption can perforate the root dentin and also induce external resorption[35,36]. If the internal resorption perforates the

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dentin and the root cementum, the microbial infection can expand to the periodontal tissue, and inflammation of the lateral periodontal tissue and lateral bone with formation of a sinus tract can occur[37–39]. In cases with extensive coronal resorption, there is a high risk of a horizontal root fracture[40,41]. Ebeleseder and Kqiku[42] presented a new biological method to repair resorptive dentinal defects. After removal of the infected tissue and disinfection, the healthy pulp tissue apical of the resorption was used for the induction of hard tissue repair. As a result, hard tissue filled the resorptive defect without further progression of the resorption. This therapeutic approach has also been confirmed by other case reports[39,43,44].

CASE PRESENTATION

A 28-year-old male patient in good general health presented with a painful maxillary lateral left incisor associated with a draining sinus tract. The patient reported a trauma on tooth 11 and 12 three years ago. The traumatic injury to the incisors did not cause any permanent pain, so the patient did not seek dental advice. There was no history of orthodontic treatment. He had been wearing a splint at night for bruxism for 4 years.

Intraoral Findings

Tooth 22 was asymptomatic. Clinical examination revealed slight vestibular swelling and an actively draining sinus tract. As a result of bruxism, the dentin was incisally exposed. The clinical crown was free of dental caries and had been restored using composite (Fig. 1*A* and *B*). The tooth responded negatively to thermal and electric pulp testing and percussion. Periodontal probing depths and tooth mobility were within normal limits.

Radiographic Findings

The periapical radiograph (Kodak 6000; Carestream Dental LLC, Atlanta, GA) demonstrated an extensive internal resorption and associated periapical and lateral radiolucencies on tooth #22. A gutta-percha cone inserted into the sinus tract pointed at the lateral inflammatory lesion, suggesting resorptive perforation of the root. A second resorptive defect is visible in the middle of the root (Fig. 2*A*). Cone-beam computed tomographic (CBCT) imaging (Veraview 3D F 80; Morita, Kyoto, Japan) was performed to obtain more information about the dimensions and locations of the resorptive lesions in relation to the periodontal tissue using standard settings (exposure time of 9.4 seconds, 70 kV, and 8 mA). CBCT imaging revealed 2 large radiolucencies within the root, and a mesial perforation of the root was suspected in the coronal third of the root. The root canal and the apical foramen were wide, which was atypical given the age of the patient (Fig. 2*B* and *C*).

Based on these findings, the diagnosis was made of pulp necrosis with chronic apical abscess, a draining sinus tract, and a perforating internal inflammatory resorption. Given the advanced stage of the resorption and the reduced amount of root dentin, the root appeared to be at risk of fracture, and tooth retention seemed questionable.

Because the available findings did not allow a final assessment of the condition of the pulp, intracoronal and intracanal diagnoses were performed to assess the vitality of the pulp and to evaluate the chances of tooth retention[45].

Intracoronal Findings and Diagnosis

Using a dental microscope (ProErgo; Zeiss, Oberkochen, Germany) at 16× magnification, the pulp chamber was accessed after rubber dam isolation, and the necrotic pulp was removed using a sterile microprobe (MicroOpener 10.04; Dentsply Maillefer, Ballaigues, Switzerland). The tissue located within the resorption lacuna appeared homogeneous with its blood supply still intact (Fig. 3*A*). Careful probing provoked only slight light-red bleeding; therefore, reversible pulpitis with reparative capacity was assumed. The goals of further treatment were to save as much of immunocompetent tissue as possible and to disinfect the coronal portion of the root canal in order to stop the inflammatory process and induce reparative hard tissue repair. The patient was informed on the unknown and unclear chances of tooth retention.

Root Canal Treatment

The infected coronal part of the root canal was disinfected up to the perfused vital tissue using 3% sodium hypochlorite and an ultrasonic device for activation of the irrigant. No mechanical enlargement of the root canal was performed. Calcium hydroxide freshly mixed with sterile saline was applied coronally to the resorptive tissue. The tooth

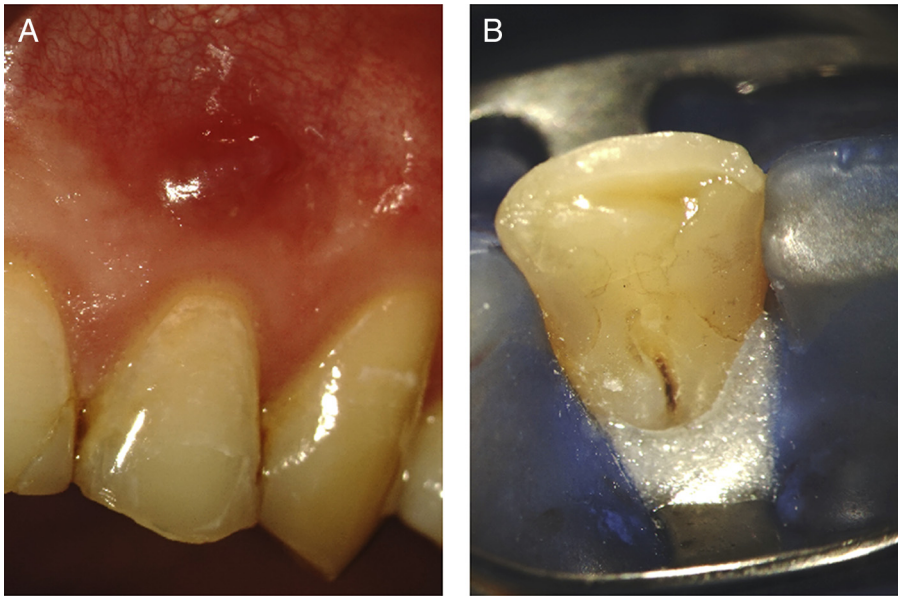

FIGURE 1 – Clinical findings of the maxillary left lateral incisor. (*A*) Swelling and a fistula are present. (*B*) Incisally dentin is exposed as a sequelae of bruxism.

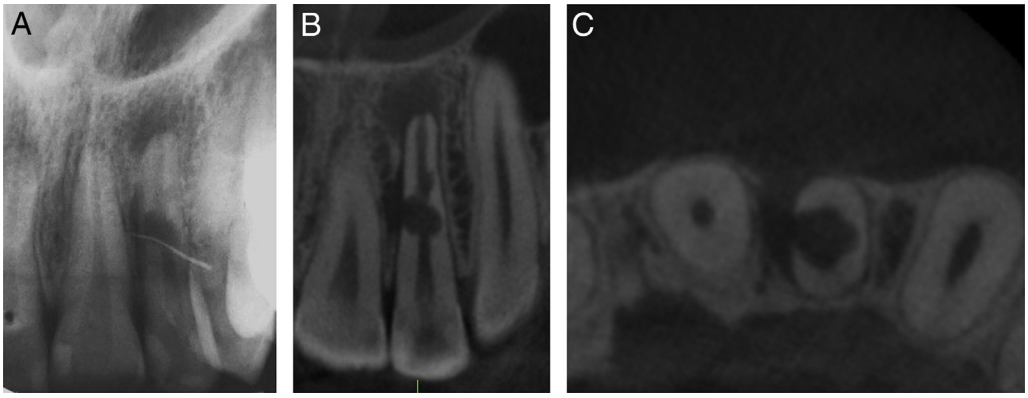

FIGURE 2 – (*A*) The gutta-percha point tracing the sinus tract points at the lateral perforation of the internal resorption. (*B*) The CBCT image in the frontal projection demonstrates the dimensions of the internal resorption and the apical and lateral radiolucencies. (*C*) In the axial reconstruction of CBCT images, the perforation is clearly visible.

was assessed radiographically after 2 weeks. At this time, the sinus tract had closed, and the patient presented asymptomatic. Calcium hydroxide was replaced under a rubber dam, and the cavity was temporarily sealed again using composite. Six weeks after the initial treatment, the medication was removed using ultrasonically activated 3% sodium hypochorite. It was noticed that under direct contact with calcium hydroxide, the tissue inside the resorption cavity was reduced by half (Fig. 3*B*). Before further treatment, the root canal was rinsed for 1 minute with 17% EDTA and dried using sterile paper points. Bleeding was provoked inside the resorption cavity using the MicroOpener (Fig. 4*A*). After 10 minutes, bleeding had stopped, and ProRoot mineral trioxide aggregate (MTA) (Dentsply Maillefer) was placed on top of the blood clot, dried using sterile paper points, and carefully compacted. After insertion of a cotton pellet soaked in sterile saline, the cavity was adhesively sealed with composite (Fig. 4*B*). In order to avoid discoloration of the tooth because of the infiltration of the MTA with blood, the MTA was reduced after 1 week to 1 mm below the cementoenamel junction using a long shaft bur, and the residual area was filled with composite in a layering technique.

RECALLS

At the first recall after 3 months, the apical and lateral radiolucencies appeared to have reduced in size, and the patient was free of symptoms. The sinus tract had healed completely (Fig. 5*A* and *B*). Further examinations were performed 1, 2, and 3 years later. At all recalls, electric and cold sensitivity tests were negative. The continuous formation of a hard tissue structure within the resorptive cavities and within the root canal could be followed over time on intraoral radiographs. Even after 3 years, the contour of the root surface was not entirely reestablished in the area of the perforation (Fig. 6*A*–*C*). Three-dimensional CBCT control confirmed that the apical and lateral lesions had completely healed. The root canal continuously had reduced in diameter but still was completely visible. In the 3-year control, there was a change in the defect boundaries in the area of the perforation; they appeared rounded compared with the initial condition. Probably, bone had partially grown into the defect zone

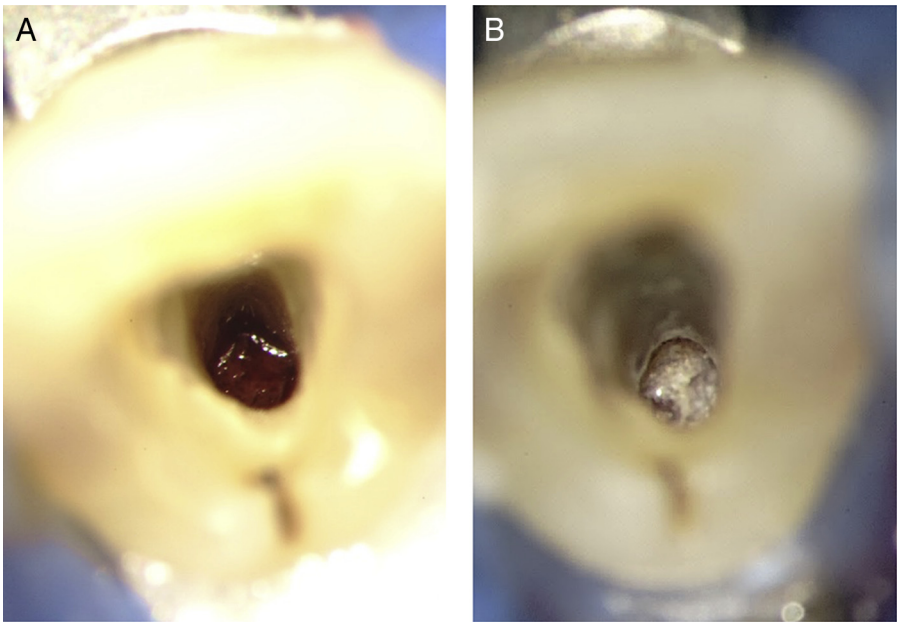

FIGURE 3 – (*A*) The perfused tissue visible in the resorption cavity. (*B*) The perfused tissue in contact with calcium hydroxide has been reduced by half.

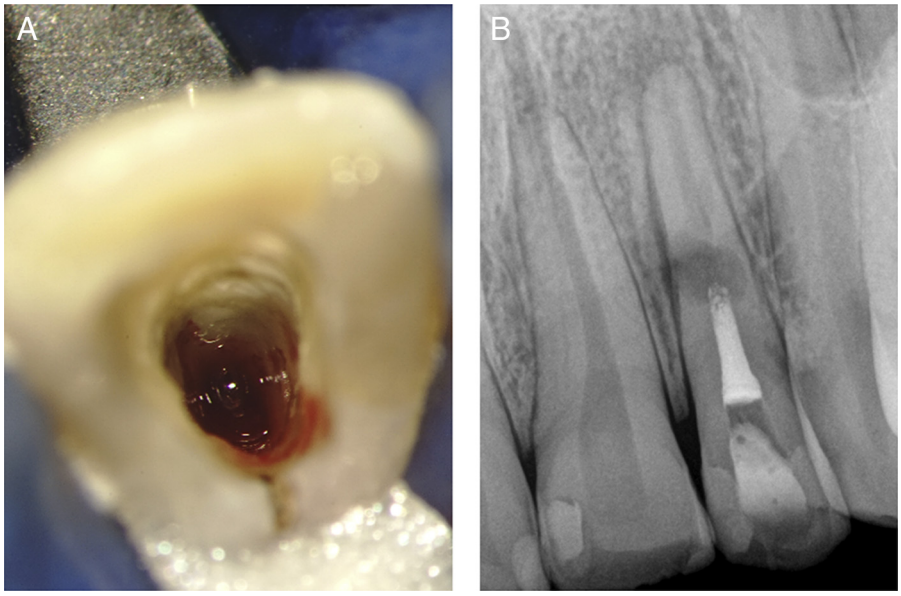

FIGURE 4 – (*A*) Induced bleeding fills the resorptive cavity with a blood clot. (*B*) Eight weeks after disinfection with calcium hydroxide and application of the MTA plug, the apical and lateral lesions already had decreased in size.

without direct contact to the root dentin; therefore, ankylosis can be ruled out (Fig. 7*A* and *B*).

DISCUSSION

So far, there have been only a few case reports on regenerative/reparative approaches in teeth with internal resorption[42,44,46,47], but only the latter 2 also were associated with lateral perforation. Teeth in these case reports presented at different stages of root development, and different protocols were followed for disinfection and the initiation of repair or regeneration, therefore not allowing conclusions on a most promising treatment regimen (Table 1). Also, the outcome of treatment has been termed repair[42] or regeneration[44], respectively. Differing from the present treatment protocol, Saoud et al[47] removed the tissue down to the apical border of the lesion; finally, they achieved arrest of the resorption and healing of the lateral and apical lesions, but no calcification occurred. No preparation was performed in the present case in contrast to the protocol of Kaval et al[44]; nevertheless, the treatment outcome was similar in both cases. Despite all the differences in treatment protocols and outcome, it may be concluded that vital tissue apical to a

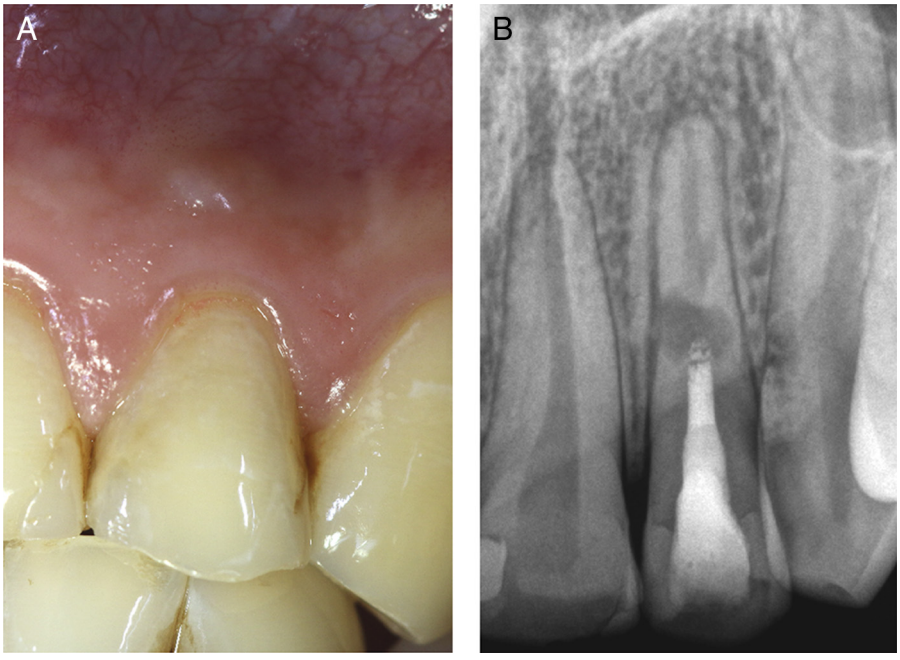

FIGURE 5 – (*A*) Three months after regenerative endodontic treatment, the gingiva looks healthy. (*B*) Radiographic examination shows further reduction of the size of the apical radiolucency.

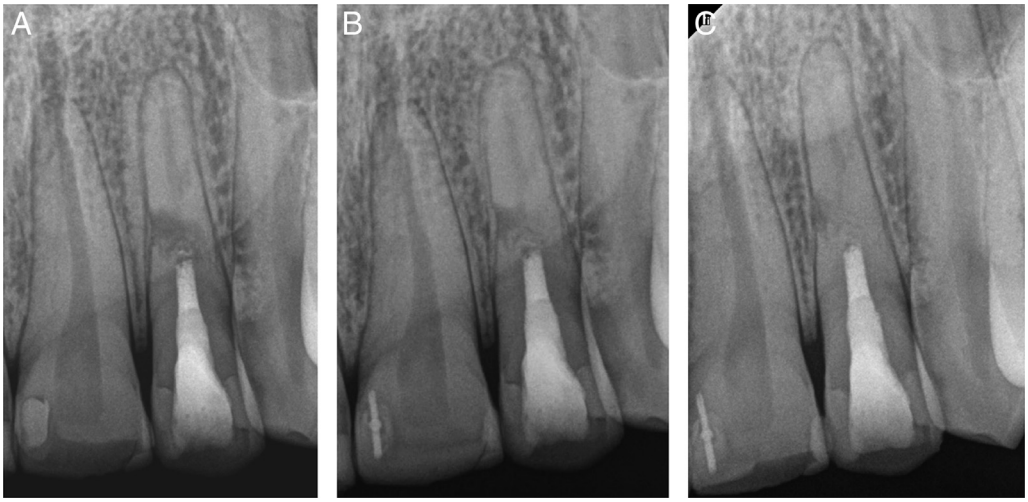

FIGURE 6 – The periapical radiographs after (*A*) 1, (*B*) 2, and (*C*) 3 years reveal that the healing process has progressed with reparative hard tissue formation inside the resorptive cavity.

resorption area can be preserved and may be helpful in apical healing.

The exact etiology of the internal resorption in the present case could not be determined retrospectively. Possible causes are the traumatic injury or a previous deep carious lesion. Because an internal resorption usually remains asymptomatic, these are often only noticed coincidentally during radiologic examination[8,48]. The symptoms in this patient were caused by a partially microbially infected pulp, with apical and lateral inflammatory

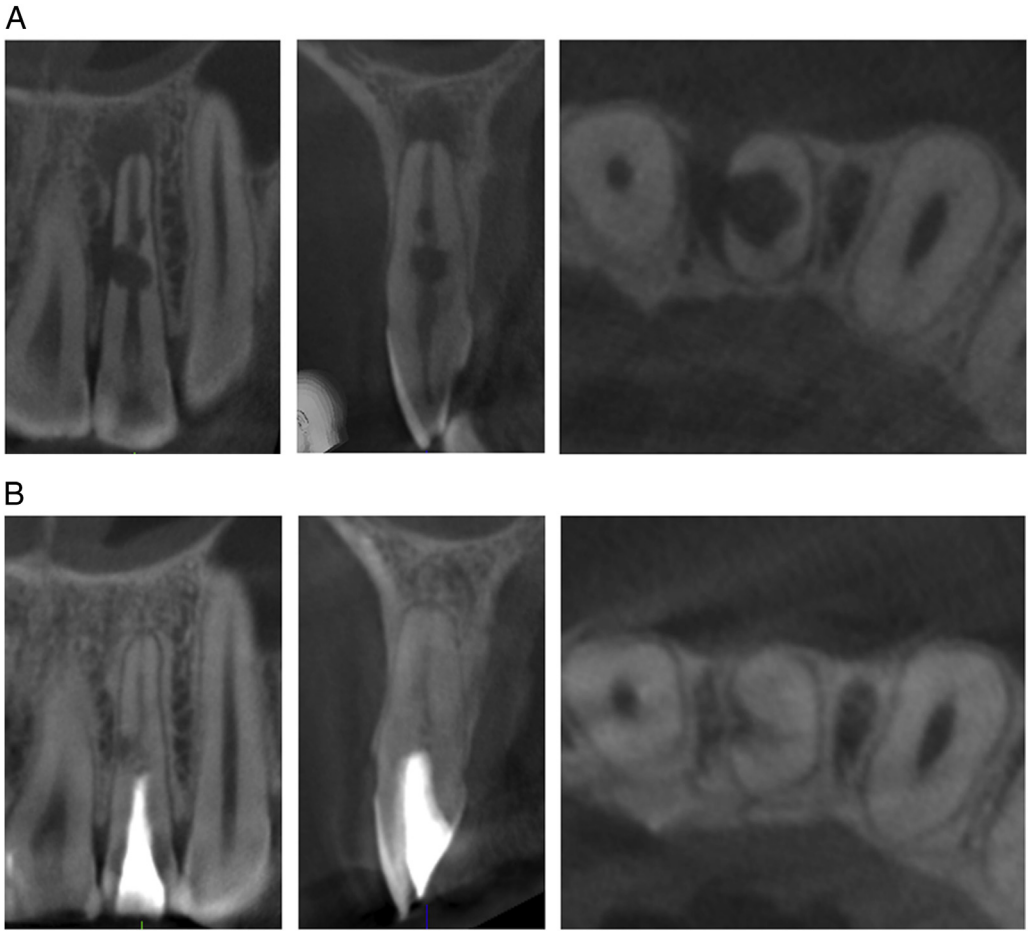

FIGURE 7 – CBCT images (*A*) before and (*B*) 3 years after treatment in the frontal, sagittal, and axial reconstruction planes. The radiolucencies are healed radiographically, and the resorption cavity is filled with mineralized tissue without progression of the resorption.

TABLE 1 - A Comparative Survey of Publications on Regenerative/Reparative Treatment of Internal Resorptions

Criteria	Ebeleseder & Kqiku (2015)[42]		Saoud et al. 2016[47]	Priya et al. 2016[46]	Kaval et al. 2018[44]	Present case
Tooth	22	21	11	21	22	22
Age of patient	38 y	28 y	16 y	11 y	14 y	28 y
Root development	Completed	Completed	Completed	Incomplete	Completed	Wide apex, wide rc
Clinical symptoms	None	None	Swelling tender to percussion	Traumatic avulsion	Moderate pain, tender to percussion	Swelling, no pain
Sensibility testing	Negative	Negative	Negative	Positive	Negative	Negative
Apical lesion	No	No	Yes	Yes	No	Yes
Perforation	Yes	No	Yes	Probably	Yes	Yes
Lateral lesion	No	No	Yes	Yes		Yes
Sinus tract	No	No	No	No	No	Yes
Irrigant	NaOCl 3.5%, CHX 0.1%	NaOCl 3.5%, CHX 0.1%	2.5% NaOCl	No debridement	NaOCl 1%, EDTA	NaOCl 3%, EDTA
Medication	$Ca(OH)_2$ + 0.1% CHX, every 3–6 mo exchange over 4 y	$Ca(OH)_2$ + 0.1% CHX, every 6 mo exchange over 2 y	$Ca(OH)_2$ & triple antibiotic paste	$Ca(OH)_2$ & double antibiotic paste	$Ca(OH)_2$	$Ca(OH)_2$
Capping material	MTA	MTA	MTA	Platelet-rich plasma & GIZ	MTA	MTA
Recall	6 y	1 and 2 y	8 and 15 mo	9 mo, 1 y	6 mo, 2 y	3 y
Results	Hard tissue barrier, resorption arrested	Resorption arrested, continuing calcification, hard tissue barrier	Resorption arrested healing of lesions, no calcification	No resorption, arrested PDL intact	Hard tissue formation remodeling of root surface, increased thickness of rc walls	Closure of sinus tract, resorption arrested, healing of l esions, hard tissue formation narrowing of rc

$Ca(OH)_2$, calcium hydroxide; CHX, chlorhexidine; GIZ, glass ionomer cement; MTA, mineral trioxide aggregate; NaOCl, sodium hypochlorite; PDL, periodontal ligament; rc, root canal.

lesions as immune reactions. As a result of the infected resorptive perforation, a sinus tract had developed.

In the present case, there was active internal resorption, as indicated by the presence of vital tissue inside the resorptive area. Without treatment, resorption results in progressive loss of tooth structure as long as vital tissue is present, and complications such as perforations or horizontal root fractures can occur[49,50]. When the cause of resorption can be eliminated, the remaining pulp tissue seems to be able to produce dentinlike hard tissue[42–44,51]. Nevertheless, the prerequisites and best treatment modalities for such reparative treatment still have to be elucidated.

So far, it has not been examined whether the surviving vital pulp tissue in or below a resorption lacuna can be used for regenerative or reparative procedures, and it still is not possible to determine exactly the degree of tissue inflammation (reversible or irreversible). Rather, it was assumed that complete removal of the resorptive tissue was the only option to prevent further resorption, if such a tooth was savable at all[34,48]. On the other hand, it is known that resorption progresses only when microorganisms and their metabolites are not removed to a certain degree. In an animal experiment, it was demonstrated that internal resorption only occurs in cases with a microbially infected pulp. If damage of predentin and odontoblasts occurred alone without microbial infection, only transient surface resorption was observed, and the progression of resorption started only after additional microbial infection[16].

Conversely, if microbial infection can be eliminated, the remaining pulp tissue is able to repair dentin defects by deposition of mineralized tissue[52,53]. Whether the pulp tissue still has sufficient regenerative potential currently cannot be reliably assessed under clinical conditions; clinical and radiologic examinations are necessary for assessment of the healing process[54]. The presence of an apical inflammatory lesion alone presents no evidence that pulp necrosis is present in the entire root canal[55,56]. In case of a cariously accessed pulp with apical radiolucency, the inflammation was inhibited after pulpotomy, and the remaining pulp could be saved[57,58]. Treatment aims at stimulation of adult dental pulp stem cells to differentiate using growth factors released from the dentin. Blood clots are mostly used as scaffolds for the growth and differentiation of stem cells. As a result, further root growth, apical closure, and increased root thickness can be observed[47,54,59–61].

In this case, contact with calcium hydroxide resulted in superficial necrosis of the granulation tissue so that the resorption lacuna was no longer completely filled with the tissue. At the same time, repeated calcium hydroxide applications were used to improve disinfection and inactivate the osteoclasts[62–64]. Starting with the creation of the blood clot, the

resorption cavity continuously was filled with mineralized dental hard tissue. However, whether the final results are due to revitalization or repair is controversial. Caliskan and Türkün[40,65] suspected that regenerated pulp tissue and odontoblasts are responsible for the production of hard tissue, but no evidence of the presence of odontoblasts and dentin was found in histologic studies. Rather, fibroblasts appear to produce a nonhomogeneous dentinlike hard tissue, which is speculated to act as a defensive barrier against the entry of new microorganisms[66,67].

Revitalization procedures require frequent follow-up investigations so that pathologic findings can be diagnosed early. Clinical investigations and intraoral radiography have proven successful for monitoring the success of antimicrobial therapy. CBCT examination can provide a detailed view of the dental structures and improve periapical diagnosis[68–71]. Three years after treatment, a decrease of the apical radiolucency from a periapical index of 4 to a periapical index of 1 was observed using CBCT imaging. A small defect on the lateral surface of the root in the area of the perforation has persisted even 3 years after obturation; therefore, further follow-up investigations are indicated.

CONCLUSION

Reparative endodontic treatment can stop the IRR process and induce the production of dental hard tissue even in the presence of a perforation, inflammatory periapical and lateral lesions, and a sinus tract. The exact mechanism of the repair and the reproducibility of the method require further investigation.

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REFERENCES

1. Wedenberg C, Zetterqvist L. Internal resorption in human teeth - a histological, scanning electron microscopic, and enzyme histochemical study. J Endod 1987;13:255–9.
2. Ne RF, Witherspoon DE, Gutmann JL. Tooth resorption. Quintessence Int 1999;30:9–25.
3. Bell T. *The Anatomy, Physiology, and Disease of the Teeth*. Philadelphia: Carey and Lee Publishing; 1830. p. 171–2.
4. Furseth R. The resorption processes of human deciduous teeth studied by light microscopy, microradiography and electron microscopy. Arch Oral Biol 1968;13:417–31.
5. Hasselgren G, Strömberg T. Histochemical demonstration of acid hydrolase activity in internal dentinal resorption. Oral Surg Oral Med Oral Path 1976;42:381–5.
6. Domon T, Sugaya K, Yawaka Y, et al. Electron microscopic and histochemical studies of the mononuclear odontoclast of the human. Anat Rec 1994;240:42–51.
7. Götz W, Quondamatteo F, Ragotzki S, et al. Localization of cathepsin D in human odontoclasts. A light and electron microscopical immunocytochemical study. Connect Tissue Res 2000;41:185–94.
8. Haapasalo M, Endal U. Internal inflammatory root resorption: the unknown resorption of the tooth. Endod Topics 2006;14:60–79.
9. Dempster DW, Lambing CL, Kostenuik PJ, et al. Role of RANK ligand and denosumab, a targeted RANK ligand inhibitor, in bone health and osteoporosis: a review of preclinical and clinical data. Clin Ther 2012;34:521–36.
10. Chung CJ, Soma K, Rittling SR, et al. OPN deficiency suppresses appearance of odontoclastic cells and resorption of the tooth root induced by experimental force application. J Cell Physiol 2008;214:614–20.
11. Sahara N, Okafuji N, Toyoki A, et al. Odontoclastic resorption at the pulpal surface of coronal dentin prior to the shedding of human deciduous teeth. Arch Histol Cytol 1992;55:273–85.
12. Sahara N, Toyoki A, Ashizawa Y, et al. Cytodifferentiation of the odontoclast prior to the shedding of human deciduous teeth: an ultrastructural and cytochemical study. Anat Rec 1996;244:33–49.
13. Khosla S, Oursler MJ, Monroe DG. Estrogen and the skeleton. Trends Endocrinol Metab 2012;23:576–81.
14. Zaidi M, Adebanjo OA, Moonga BS, et al. Emerging insights into the role of calcium ions in osteoclast regulation. J Bone Miner Res 1999;14:669–74.
15. Trope M. Root resorption of dental and traumatic origin: classification based on etiology. Pract Periodontics Aesthet Dent 1998;10:515–22.
16. Wedenberg C, Lindskog S. Experimental internal resorption in monkey teeth. Endod Dent Traumatol 1985;1:221–7.

17. Zheng Y, Chen M, He L, et al. Mesenchymal dental pulp cells attenuate dentin resorption in homeostasis. J Dent Res 2015;94:821–7.

18. Andreasen JO. Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth. Scand J Dent Res 1970;78:273–6.

19. Andreasen FM, Kahler B. Pulpal response after acute dental injury in the permanent dentition: clinical implications-a review. J Endod 2015;41:299–308.

20. Tronstad L. Root resorption–etiology, terminology and clinical manifestations. Endod Dent Traumatol 1988;4:241–52.

21. Tindol JE. Internal resorption: a case presentation with bruxism as the possible etiology. Tex Dent J 1992;109:13–25.

22. Ahlberg K, Bystedt H, Eliasson S, et al. Long-term evaluation of autotransplanted maxillary canines with completed root formation. Acta Odontol Scand 1983;41:23–31.

23. Eliasson S, Låftman AC, Strindberg L. Autotransplanted teeth with early-stage endodontic treatment: a radiographic evaluation. Oral Surg Oral Med Oral Pathol 1988;65:598–603.

24. Paulsen HU, Andreasen JO, Schwartz O. Pulp and periodontal healing, root development and root resorption subsequent to transplantation and orthodontic rotation: a long-term study of autotransplanted premolars. Am J Orthod Dentofacial Orthop 1995;108:630–40.

25. Dilts WE, Luebke RG. Internal resorption and crown preparation. J Prosthet Dent 1967;18:126–30.

26. Cabrini RL, Manfrede EE. Internal resorption of dentine; histopathologic control of eight cases after pulp amputation and capping with calcium hydroxide. Oral Surg Oral Med Oral Pathol 1957;10:90–6.

27. Sönmez D, Durutürk L. $Ca(OH)_2$ pulpotomy in primary teeth. Part I: internal resorption as a complication following pulpotomy. Oral Surg Oral Med Oral Path Oral Radiol Endod 2008;106:e94–8.

28. Galler KM, D'Souza RN, Federlin M, et al. Dentin conditioning codetermines cell fate in regenerative endodontics. J Endod 2011;37:1536–41.

29. Chen MY, Chen KL, Chen CA, et al. Responses of immature permanent teeth with infected necrotic pulp tissue and apical periodontitis/abscess to revascularization procedures. Int Endod J 2012;45:294–305.

30. Nagaraj E, Kaur RP, Raghuram PH, et al. Multiple internal resorption in permanent teeth associated with hyperparathyroidism. Indian J Dent Res 2013;24:128–31.

31. Silveira FF, Nunes E, Soares JA, et al. Double 'pink tooth' associated with extensive internal root resorption after orthodontic treatment: a case report. Dent Traumatol 2009;25:e43–7.

32. Urban D, Mincik J. Monozygotic twins with idiopathic internal root resorption: a case report. Aust Endod J 2010;36:79–82.

33. Gabor C, Tam E, Shen Y, Haapasalo M. Prevalence of internal inflammatory root resorption. J Endod 2012;38:24–7.

34. Patel S, Ricucci D, Durak C, et al. Internal root resorption: a review. J Endod 2010;36:1107–21.

35. Tamse A. Internal resorption with external perforation (case report). Refuat Hapeh Vehashinayim 1976;25:15–8.

36. Abuabara A, Costa RG, Morais EC, et al. Prosthetic rehabilitation and management of an MTA-treated maxillary central incisor with root perforation and severe internal resorption. J Prosthodont 2013;22:413–8.

37. Hsien HC, Cheng YA, Lee YL, et al. Repair of perforating internal resorption with mineral trioxide aggregate: a case report. J Endod 2003;29:538–9.

38. Nunes E, Silveira FF, Soares JA, et al. Treatment of perforating internal root resorption with MTA: a case report. J Oral Sci 2012;54:127–31.

39. Abdullah D, Eziana Hussein F, Abd Ghani H. Management of perforating idiopathic internal root resorption. Iran Endod J 2017;18:257–60.

40. Calişkan MK, Türkün M. Prognosis of permanent teeth with internal resorption: a clinical review. Endod Dent Traumat 1997;13:75–81.

41. Brito-Júnior M, Quintino AF, Camilo CC, et al. Nonsurgical endodontic management using MTA for perforative defect of internal root resorption: report of a long term follow-up. Oral Surg Oral Med Oral Path Oral Rad Endod 2010;110:784–8.

42. Ebeleseder KA, Kqiku L. Arrest and calcification repair of internal root resorption with a novel treatment approach: report of two cases. Dent Traumatol 2015;31:332–7.

43. Heithersay GS. Management of tooth resorption. Aust Dent J 2007;52:105–21.

44. Kaval ME, Güneri P, Çalışkan MK. Regenerative endodontic treatment of perforated internal root resorption: a case report. Int Endod J 2018;51:128–37.

45. Arnold M, Friedrichs C, Tulus G, et al. Intracoronal and intracanal endodontic diagnostics (ICD). Endod Pract Today 2018;12:9–20.

46. Priya MH, Tambakad PB, Naidu J. Pulp and periodontal regeneration of an avulsed permanent mature incisor using platelet-rich plasma after delayed replantation: a 12-month clinical case study. J Endod 2016;42:66–71.

47. Saoud TM, Mistry S, Kahler B, et al. Regenerative endodontic procedures for traumatized teeth after horizontal root fracture, avulsion, and perforating root resorption. J Endod 2016;42:1476–82.

48. Hülsmann M, Schäfer E. Resorption. In: Hülsmann M, Schäfer E, editors. *Problems in Endodontics*. 1st ed. Berlin: Quintessenz; 2009. p. 421–34.

49. Anil S, Raji MA, Beena VT, et al. Fracture of tooth by internal resorption: case report. Endod Dent Traumatol 1993;9:79–80.

50. Hariharan VS, Nandlal B, Srilatha KT. Management of recurrent fracture of central incisor with internal resorption using light transmitting (luminex) post. J Indian Soc Pedod Prev Dent 2010;28:288–92.

51. Lyroudia KM, Dourou VI, Pantelidou OC, et al. Internal root resorption studied by radiography, stereomicroscope, scanning electron microscope and computerized 3D reconstructive method. Dent Traumatol 2002;18:148–52.

52. Duncan HF, Cooper PR, Smith AJ. Dissecting dentine-pulp injury and wound healing responses: consequences for regenerative endodontics. Int Endod J 2019;52:261–6.

53. Ricucci D, Siqueira JF Jr, Li Y, et al. Vital pulp therapy: histopathology and histobacteriology-based guidelines to treat teeth with deep caries and pulp exposure. J Dent 2019;86:41–52.

54. European Society of Endodontology (ESE) developed by: Duncan HF, Galler KM, Tomson PL, et al. European Society of Endodontology position statement: management of deep caries and the exposed pulp. Int Endod J 2019;52:923–34.

55. Lin L, Shovlin F, Skribner J, et al. Pulp biopsies from the teeth associated with periapical radiolucency. J Endod 1984;10:436–48.

56. Kovacević M, Tamarut T, Jonjić N, et al. The transition from pulpitis to periapical periodontitis in dogs' teeth. Aust Endod J 2008;34:12–8.

57. Russo MC, Holland R, de Souza V. Radiographic and histological evaluation of the treatment of inflamed dental pulps. Int Endod J 1982;15:137–42.

58. Calişkan MK. Pulpotomy of carious vital teeth with periapical involvement. Int Endod J 1995;28:172–6.

59. Banchs F, Trope M. Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? J Endod 2004;30:196–200.

60. Murray PE, Garcia-Godoy F, Hargreaves KM. Regenerative endodontics: a review of current status and a call for action. J Endod 2007;33:377–90.

61. Petrino JA, Boda KK, Shambarger S, et al. Challenges in regenerative endodontics: a case series. J Endod 2010;36:536–41.

62. Massarstrom LE, Blomlof LB, Feiglin B, et al. Effect of calcium hydroxide treatment on periodontal repair and root resorption. Endod Dent Traumatol 1986;2:184–9.

63. Jiang J, Zuo J, Chen SH, et al. Calcium hydroxide reduces lipopolysaccharide-stimulated osteoclast formation. Oral Surg Oral Med Oral Path Oral Radiol Endod 2003;95:348–54.

64. Mohammadi Z, Dummer PM. Properties and applications of calcium hydroxide in endodontics and dental traumatology. Int Endod J 2011;44:697–730.

65. Calişkan MK, Türkün M. Root canal treatment of a root-fractured incisor tooth with internal resorption: a case report. Int Endod J 1996;29:393–7.

66. Lin LM, Ricucci D, Huang GT. Regeneration of the dentine-pulp complex with revitalization/revascularization therapy: challenges and hopes. Int Endod J 2014;47:713–24.

67. Ricucci D, Loghin S, Lin LM, et al. Is hard tissue formation in the dental pulp after the death of the primary odontoblasts a regenerative or a reparative process? J Dent 2014;42:1156–70.

68. Patel S, Dawood A, Wilson R, et al. The detection and management of root resorption lesions using intraoral radiography and cone beam computed tomography - an in vivo investigation. Int Endod J 2009;42:831–8.

69. Kamburoğlu K, Kursun S. A comparison of the diagnostic accuracy of CBCT images of different voxel resolutions used to detect simulated small internal resorption cavities. Int Endod J 2010;43:798–807.

70. Bhuva B, Barnes JJ, Patel S. The use of limited cone beam computed tomography in the diagnosis and management of a case of perforating internal root resorption. Int Endod J 2011;44:777–86.

71. Estrela C, Holland R, Estrela CR, et al. Characterization of successful root canal treatment. Braz Dent J 2014;25:3–11.