Received: 3 March 2024 | Accepted: 5 May 2024

DOI: 10.1002/cap.10297

CASE STUDY

A clinical dilemma: Performing or avoiding root instrumentation in the treatment of the acute phase of endodontic-periodontal lesions? A case report

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Abstract

Background: This study presents the diagnois, management, and tissue response to an acute periodontal lesion with deep pocketing affecting a maxillary central incisor in a young patient devoid of caries or a history of periodontitis.

Methods: Clinical and radiographic examinations facilitated the diagnosis of the pathology as an endoperiodontal lesion (EPL) with root damage, exhibiting supracrestal invasive root resorption. Orthograde endodontic therapy was employed to decontaminate and seal the endodontic space. The resorptive site was treated through the endodontic access, debrided, and sealed. No periodontal therapy (surgical or nonsurgical) was performed. No mechanical instrumentation was performed within the pocketed root surface.

Results: At 6-month and 1-year follow-ups after endodontic therapy the periodontium displayed a physiologically healthy condition without pus or inflammation, exhibiting a circumferential probing depth of 2 mm, and absence of tooth mobility. These favorable outcomes persisted throughout a 4-year follow-up period.

Conclusions: The spontaneous healing of pocketing and abscess occurred without mechanical root instrumentation following endodontic therapy and treatment of external invasive root resorption in an EPL.

KEYWORDS
case reports, endodontics, periodontal ligament, periodontics, root planing, root resorption, wound healing

Key Points

- Accurate diagnosis and identification of relevant etiologic factors are pivotal for effectively managing endodontic-periodontal lesions. Once a diagnosis is established, the therapy focuses on eliminating the primary cause, followed by a subsequent diagnostic phase after healing. The definitive understanding of the diagnosis and etiology of endodontic-periodontal lesions often becomes clear in retrospect, based on the outcomes of the therapy.
- When probing acute periodontal lesions, deep probing depths may occur without permanent loss of periodontal attachment. If the acute lesion was

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not induced by a periodontal cause and if no periodontal etiology arises secondarily, resolving the primary cause of the endoperiodontal lesion can lead to the spontaneous resolution of the pocketing. This results in spontaneous healing of periodontium without the need for intentional periodontal therapy.

- A clinical dilemma arises when considering periodontal treatment during the acute inflammatory phase of endo-periodontal pathology. It is advisable to refrain from mechanical root instrumentation particularly if a clear periodontal cause is not apparent, to prevent from iatrogenic damage to periodontal fibers and the potential risk of gingival recessions. However, this does not imply avoiding periodontal therapy entirely for every case. Rather, it is recommended to delay the decision on root instrumentation until a new diagnostic phase is conducted following the healing of the endodontic etiology.

INTRODUCTION

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions formally defined endo-periodontal lesions (EPL) as "pathological communications between the pulpal and periodontal tissues at a given tooth, that occur in either an acute or a chronic form".[1,2] Depending on the presence of root damage in the etiology, EPL were categorized into two groups: EPL without root damage and EPL with root damage, the latter associated with root fracture, root or pulp chamber perforation, or root resorption.[1,2] Prognosis worsens when root damage is present, depending on the type of root alteration and the opportunity for repair. Some root damages, such as a vertical root fracture, cannot be repaired, while others, such as root resorption, offer opportunities for treatment.

Root resorption involves progressive loss of root structure. A classification of tooth/root resorption was recently developed, combining anatomical, physiological, and pathological approaches, identifying two broad categories of internal and external tooth resorption.[3,4] The external invasive resorption, a specific type of external resorption, has gained special consideration due to its insidious process. The invading tissue is a highly vascular fibrous connective tissue that bleeds profusely if probed.[5] As resorption progresses, ectopic calcific bone-like tissue develops. Histologically, small "finger-like" channels or projections can be observed extending further into the dentin and connecting with the periodontal ligament (PDL), providing multiple entry points for blood vessels and invading clastic cells.[3,4] The resorptive pathology can initiate at various heights of the dental root,[6] and is classified based on the extent of the invasion with four classes by Heithersay.[7,8] This concept aligns with the recently issued position statement from the European Society of Endodontology[9] and the joint statement by the American Association of Endodontists & American Academy of Oral & Maxillofacial Radiology.[10]

When the resorptive lesion reaches the periodontal space, an inflammatory process diffuses between the endodontic and periodontal tissues, generating an EPL that might be symptomatic with an abscess. At the physiological and pathological levels, little is known about the vitality of the periodontal ligament during the acute phase of an EPL with primary endodontic etiology.[11,12] Treatment of EPL associated with root resorption poses a challenge, particularly in esthetic areas of the mouth. The first phase of therapy involves treating the endodontic etiology and root damage, followed by staged treatment of the residual periodontal component. Despite this pragmatic approach described in books[13,14] and widely accepted in clinical practice, publications with clinical data are lacking. Clinical science is encouraged to clarify the timing of periodontal therapy, such as scaling and root planing, and of endodontic therapy. In the presence of an acute lesion, scaling and root planing could cause serious irreversible damage by eliminating the still-viable periodontal fibers on the infected root surface and healthy periodontal tissues adjacent to the lesion.[13,15] If a cautious approach is crucial for treating an acute periodontal abscess in a periodontally compromised patient, it is reasonable to avoid treating the dental root mechanically, especially when an acute periodontal abscess has recently manifested in a young patient with healthy periodontal status and in an esthetic area of the mouth. Eliminating potentially still-viable periodontal fibers would compromise tissue healing, eliminating the possibility of reattachment of PDL fibers and risking gingival recession after treatment.

The present case report shows the management of an acute lesion of a maxillary central incisor in a periodontally healthy patient, ultimately diagnosed with EPL with root damage associated with external root resorption. As the periodontium fully heals with treatment of the endodontic etiology alone, this case serves as proof of principle that periodontal fibers can be separated by an acute event while still maintaining viability, with the potential for reattachment when the cause of the separation is removed.

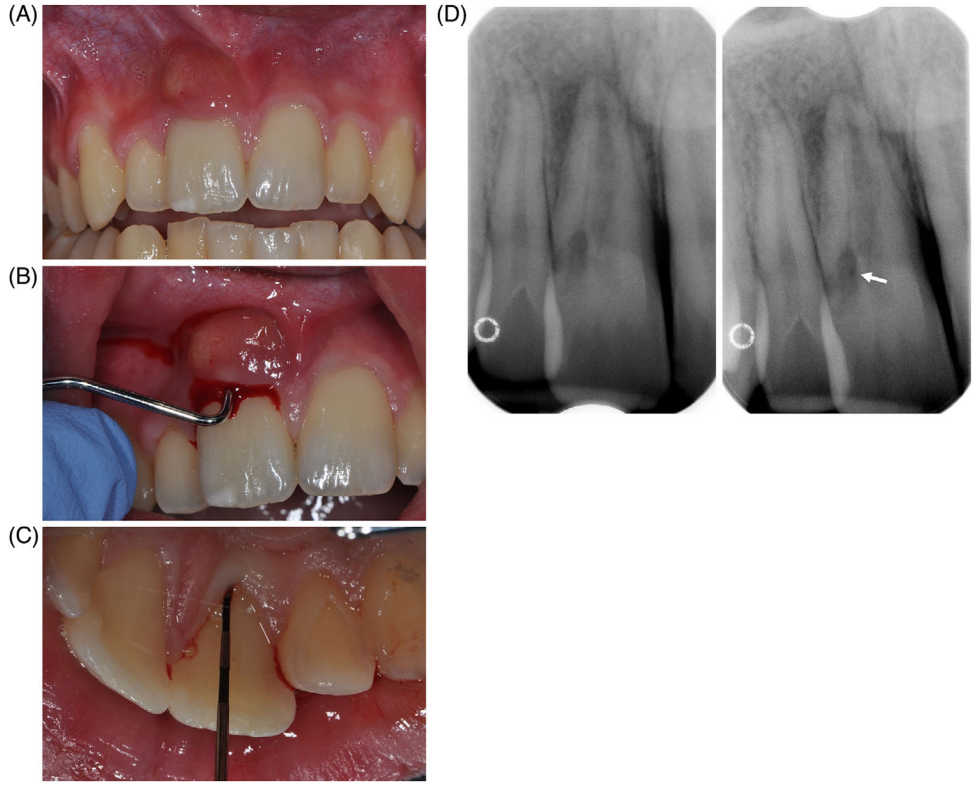


FIGURE 1 Clinical and radiographic presentation of the right maxillary central incisor. The patient complained of a severe acute abscess on the buccal side presenting with edema, swelling, and moderate tooth mobility (A). Deep pocketing (>15 mm) and profuse bleeding were present on the buccal aspect of the tooth (B), while physiological depth was noted on the palatal side (C). Two periapical radiographs taken from different angles identify the area of root resorption (D). They revealed a narrow area of root resorption starting in the most coronal part of the root (white arrow) in communication with a larger resorptive area close to the pulp tissue. Periapical pathology was also noted at the radiographic examination.

MATERIALS AND METHODS

Case report

1st visit: Diagnosis and emergency treatment

In early 2018, a 19-year-old male patient, systemically and periodontally healthy, sought care at the Department of Endodontics, University of Florence, Italy. He presented with complaints of an acute localized periodontal abscess on the maxillary right central incisor. The symptoms included bleeding, suppuration, edema, mobility, and pain. During the anamnestic interview, the patient reported a rapid onset of the abscess within the last 24 hours, accompanied by increased swelling, bleeding, edema, and pain during chewing. The patient denied a history of previous periodontal surgery, orthodontic therapy, or bleaching but recalled a trauma to the anterior mouth region at around 13/14 years of age while playing with friends.

Upon the initial intraoral examination, a severe acute abscess was evident on the buccal side of the maxillary right central incisor, presenting with pus, edema, swelling, grade 2 mobility, and pain (Figure 1A). No tooth discoloration was observed, and no external caries or lesions were visible on the dental crown. Furthermore, there was no presence of a fistula on the periodontal tissues. No recession was noted on the maxillary central incisor, while a deep pocket exhibited a probing depth of >15 mm on the buccal aspect of the affected tooth (Figure 1B), with physiological probing depths of 2 mm on the palatal side (Figure 1C). The adjacent mesial and distal teeth exhibited a healthy condition with a thick periodontal phenotype, displaying physiological sulcus depths without signs of gingivitis.

Diagnostic tests, including electric pulp test (Sybron Endo, Vitality Scanner model 2006; Kerr Italia), were conducted yielding negative results, indicating the loss of vitality in the central incisor. A periapical X-ray revealed a narrow area of root resorption starting in the most coronal part of the root apical to the cementoenamel junction (CEJ), communicating with a larger resorptive area close to the pulp and root canal (Figure 1D). A periapical radiolucent area was also evident. Subsequent X-rays were taken from different angles to precisely identify the area of root resorption (Figure 1D).

Based on patient's history, clinical, and radiographic information, the acute pathology on the maxillary central incisor was diagnosed as an endo-periodontal lesion with root damage, associated with root resorption at the CEJ level or coronal to the bone crest.[2] The clinician (R.P.)

considered the presence of the severe acute abscess, with pus, edema, swelling, >15 mm probing depth, and pain, as a crucial factor requiring immediate emergency treatment.

The patient was fully informed about the treatment plan and provided written consent in accordance with the Helsinki Declaration of 1975, as revised in 2013.

Endodontic treatment

Following the administration of 1.8 mL of local anesthetic solution, 2% articaine with 1:100,000 epinephrine, the tooth was meticulously isolated using a rubber dam. Microscopic visualization was employed to identify the resorption area through the palatal access cavity. The opening allowed for the identification of the canal and, distally, the resorption lacuna. To disinfect the area, a desiccant cleanser (Hybenx, EPIEN Medical Inc.) was injected into the root defect and left in place for 30 s before rinsing. Ultrasonic tips (Start-X, Dentsply Sirona) were utilized under constant sterile saline irrigation to delicately remove granulation tissue that resided in the resorption cavity, closely interacting with the pulp chamber. A minute portal of entry on the distal aspect of the cervical part of the root was identified under magnification. This small portal, obscured externally by tissue edema, established communication between the root surface and the resorption lacuna.

The root canal underwent chemomechanical debridement using mechanical instruments (ProTaper Gold, Dentsply Sirona) and a 5% sodium hypochlorite irrigation solution. The working length was accurately determined using an apex locator (Root ZX II; Morita). After a final rinse with 10% EDTA and 5% sodium hypochlorite, the root canal was meticulously dried using sterile paper points and filled with gutta-percha points (Protaper Gold Conform Fit, Dentsply Sirona) and resin sealer (AH-Plus, Dentsply Sirona) employing a vertical warm technique.

The resorption area was further cleansed with a surface cleaner (AH Plus Cleaner, Dentsply Sirona) to eliminate amines and epoxy resins from dentin and enamel surfaces, preparing the dentin surface for dentin bonding. The bonding process was achieved using a two-step self-etching light-cure bonding agent (Clearfil Se Bond 2, Kuraray Noritake). To mitigate polymerization shrinkage, a bulk-fill composite resin (SDR flow+, Dentsply Sirona) was applied to seal the coronal resorption cavity. This was crucial as the residual cavity walls were extremely thin, minimizing the risk of cracking. Finally, the palatal access cavity was sealed with composite (3M Filtek Supreme XTE Universal Composite) with A3 color (Figure 2A–F). A concluding periapical X-ray was performed to assess the effectiveness of the treatment (Figure 2G). At the conclusion of the treatment, the patient was prescribed anti-inflammatory medications (ibuprofen 600 mg, 1 tab/q 6 h as needed for pain control) and provided with detailed instructions for proper oral hygiene.

In consideration of the sudden onset of the abscess within the last few hours, the decision was made by the operator (R.P.) to refrain from mechanical instrumentation of the root in the site of the pocket. This precaution aimed to avoid the risk of damaging any potentially viable periodontal ligament fibers still attached to the radicular cementum.

RESULTS

2nd visit: 2 weeks after treatment

Fourteen days later the patient returned to the department, reporting the complete disappearance of signs and symptoms associated with the previous abscess (pain, mobility, edema) within a few days after the treatment. During the clinical examination, the periodontal tissues appeared healthy and pink, devoid of any signs of inflammation. There was no recession of the gingival margin, and the abscess had completely regressed with only the endodontic therapy. The periodontal tissues showed no signs of inflammation, and the tooth displayed no mobility (Figure 3). No probing was attempted in the healing site at this stage.

Additional follow-up examinations

Clinical examinations were conducted at 3 months, 6 months, 1 and 4 years after treatment.

At the 1-year follow-up, the periodontal tissues around the central incisor and adjacent mesial and distal teeth exhibited optimal health. Probing depth at buccal sites of the central incisor revealed a physiological gingival sulcus. No recession of the gingival margin occurred after the resolution of the pathology. The tooth was not mobile, and periapical X-rays confirmed the enduring stability of the obtained outcomes (Figure 4A–C).

Four years after treatment both clinical and radiological outcomes remained stable. Assessment of buccal, palatal, and interdental sites showed physiologic sulcus depth (1–3 mm). The tissue was light pink without signs of inflammation. Periapical X-rays confirmed the long-lasting stability of periodontal health (Figure 5A–E).

DISCUSSION

The described case has sparked considerable interest as it allows reflection specifically concerning the patient characteristics, the diagnosis of the lesion based on the existing classification framework for endoperiodontal lesions (EPL), and the decision not to perform any periodontal treatment until complete resolution of the endodontic etiology.

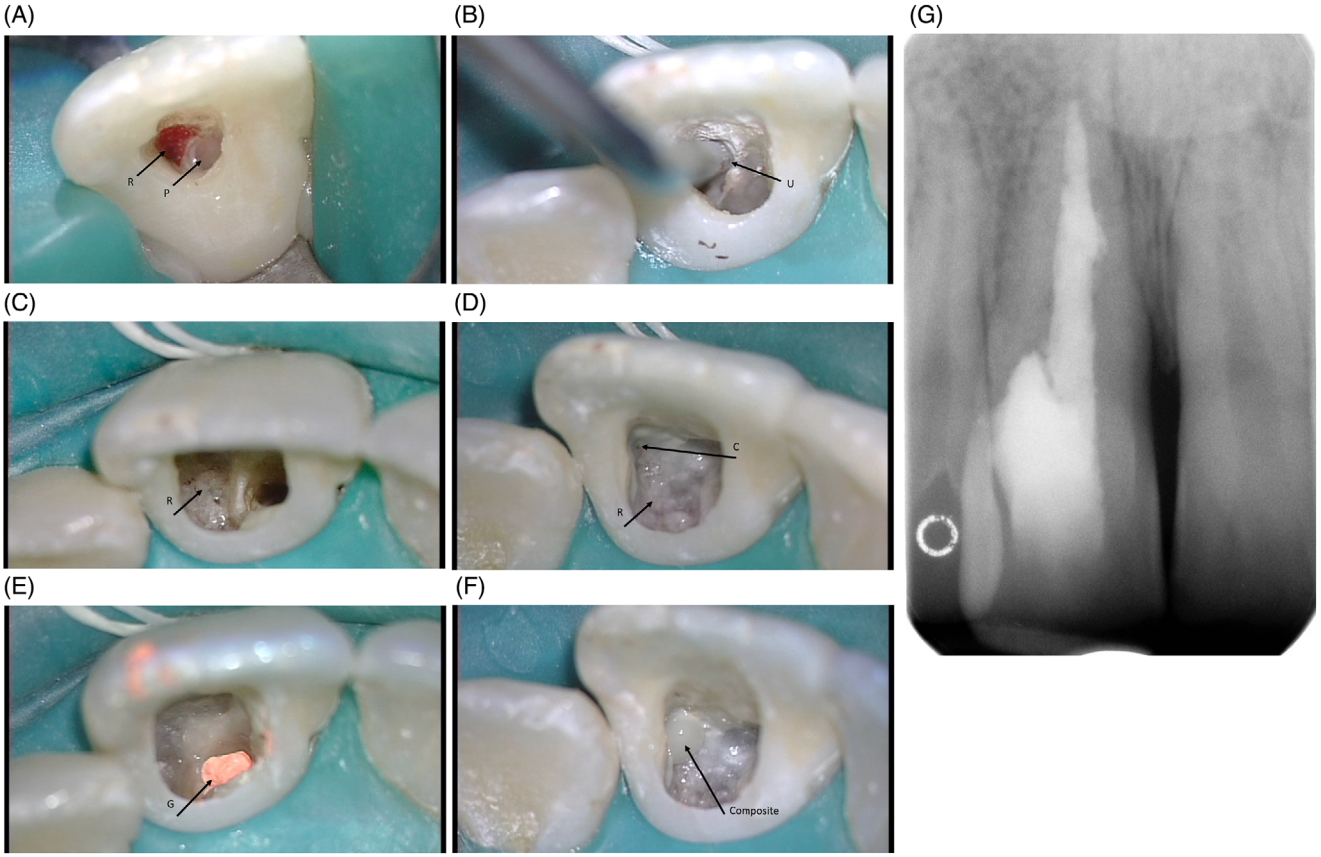


FIGURE 2 Microscope imaging was obtained during endodontic therapy. After the pulp chamber opening and initial debridement, a red granulation tissue with a tendency toward bleeding was noted inside a resorptive site (R) next to the pulp chamber (P) (A). Ultrasonic instruments (U) were used to debride and degranulate the resorptive area (B). The granulation tissue was completely removed from the resorptive site (R) (C); it allowed the exposure of a narrow channel (C) which created a communication between periodontal and pulp tissues (D). From that small channel, microbes of the oral cavity contaminated the pulp. Guttapercha (G) was used to seal the endodontic radicular space (E), while composite was used to seal the resorptive site (F). Periapical X-ray documented the successful obturation of the endodontic space as well as the repair of the tooth structure (G).



FIGURE 3 Two weeks after therapy, the periodontal tissues appeared healthy and pink, devoid of any signs of inflammation. The abscess had completely regressed without recession of the gingival margin.

Initially striking was the case of a systemically healthy 19-year-old patient, presenting at the Department of Endodontics with an acute periodontal abscess, associated with a >15 mm pocket, which manifested suddenly a few hours before the visit. The lesion presented in the absence of caries or visible coronal fracture lines and without signs of periodontal disease elsewhere in the oral cavity. The incisor was nonvital, and only a vague memory of trauma many years prior could be associated with the loss of vitality. Intraoral X-rays displayed a narrow region of supracrestal root resorption connecting externally to the oral environment via the crevicular sulcus. Internally within the tooth, this area converged into a more extensive resorptive zone close to the pulp, accompanied by a visible periapical radiolucent area.

The initial diagnosis, in compliance with the official classification of the 2018 World Workshop, was of an endoperiodontal lesion with root damage.[1,2] The resorption was identified as external invasive resorption,[3,4] Class II according to Heithersay et al.[6–8] because of the moderate involvement of the cervical dentin. A cone beam computed tomography (CBCT) could have enhanced both preoperative diagnosis and postoperative follow-up. Unfortunately, despite today's technological advances and availability, immediate access to a CBCT machine is often limited,

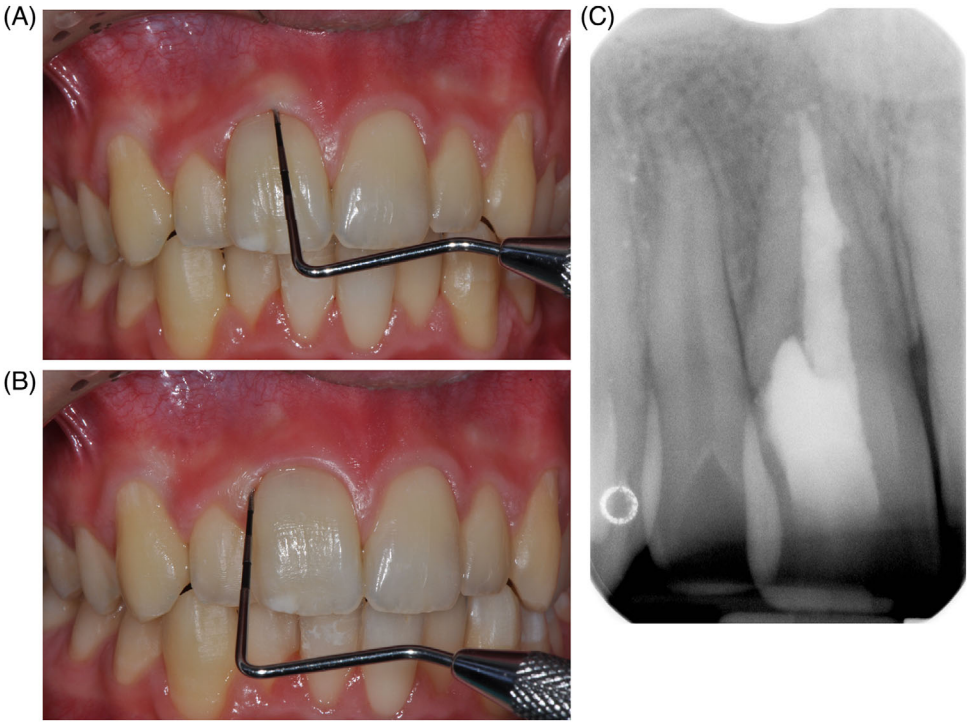


FIGURE 4 At the 1-year follow-up, the periodontal tissues surrounding the central incisor showed excellent health. Probing buccal (A), interproximal (B), and palatal sites of the central incisor indicated a normal sulcus depth. Periapical X-rays verified the sustained stability of the achieved results (C).

especially in the hospital setting where the patient was treated. Given the acute phase of the endo-periodontal pathology, the acute pain, and the substantial risk for permanent periodontal damage in case of delayed therapy, the decision was made to rely on clinical examination and intraoral radiography for diagnosis. Additionally, the use of CBCT would not have altered the treatment approach, which prioritizes endodontic treatment followed by addressing any remaining periodontal issues. Furthermore, local regulations on radiation exposure during follow-up examinations, coupled with patient reluctance to cover CBCT costs (not waivable in a hospital setting), made CBCT acquisition impractical for follow-up purposes. The decision to treat the case without CBCT imaging remained in line with the latest recommendations, advocating for CBCT use only when lower-exposure options fail to provide necessary diagnostic information.[16]

It is believed that the emergence of the invasive root resorption acted as a pathological trigger. Microbes penetrated this narrow periodontal entry point, infiltrating the resorption site, leading to infection within the pulp chamber, the root canal, and ultimately extending to the apex. However, the cause of the resorption remains unclear.[3] In this case, it was not possible to determine whether root resorption occurred first, leading to pulp necrosis, or if pulp necrosis occurred first and/or independently of the root resorption. No previous records were available to establish whether the pulp was already necrotic due to the old trauma and the root resorption started independently for other causes, or if the trauma triggered the root resorption which ultimately led to pulp infection. It is also interesting to note that the acute phase of the pathology did not create a buccal stoma, presumably because the patient had a thick periodontium, causing the pus to drain into the periodontal ligament until it exited into the gingival sulcus.

Literature has explored the epidemiology, etiopathogenesis and progression of cervical resorption revealing associations with tooth location, such as a higher prevalence for maxillary central incisors, a history of orthodontic therapy, trauma, parafunctional habits, poor oral health, malocclusion, previous aggressive bleaching techniques, and extraction of a neighboring tooth.[17,18] Once established, the resorptive process of cervical resorption follows three stages: initiation, progression/resorption, and reparative phase.[18] Hypoxia has been linked to cervical resorption because it can regulate clastic cell formation, migration, and activation. Investigations showed that there is a gradient of hypoxia within the resorptive lesion and surrounding tooth structure, furtherly confirmed by the formation of pulp stones.[19]

Because the patient presented for the first time to our observation with an acute endo-periodontal lesion, with cervical resorption and unknown periodontal etiology, diagnosis, and treatment following a staged approach. Initially, the endodontic treatment was prioritized, allowing time for the patient to heal before initiating a new diagnostic phase. With these valuable insights, immediate planning was made solely for the endodontic therapy to address the endodontic space and the resorptive lesion.

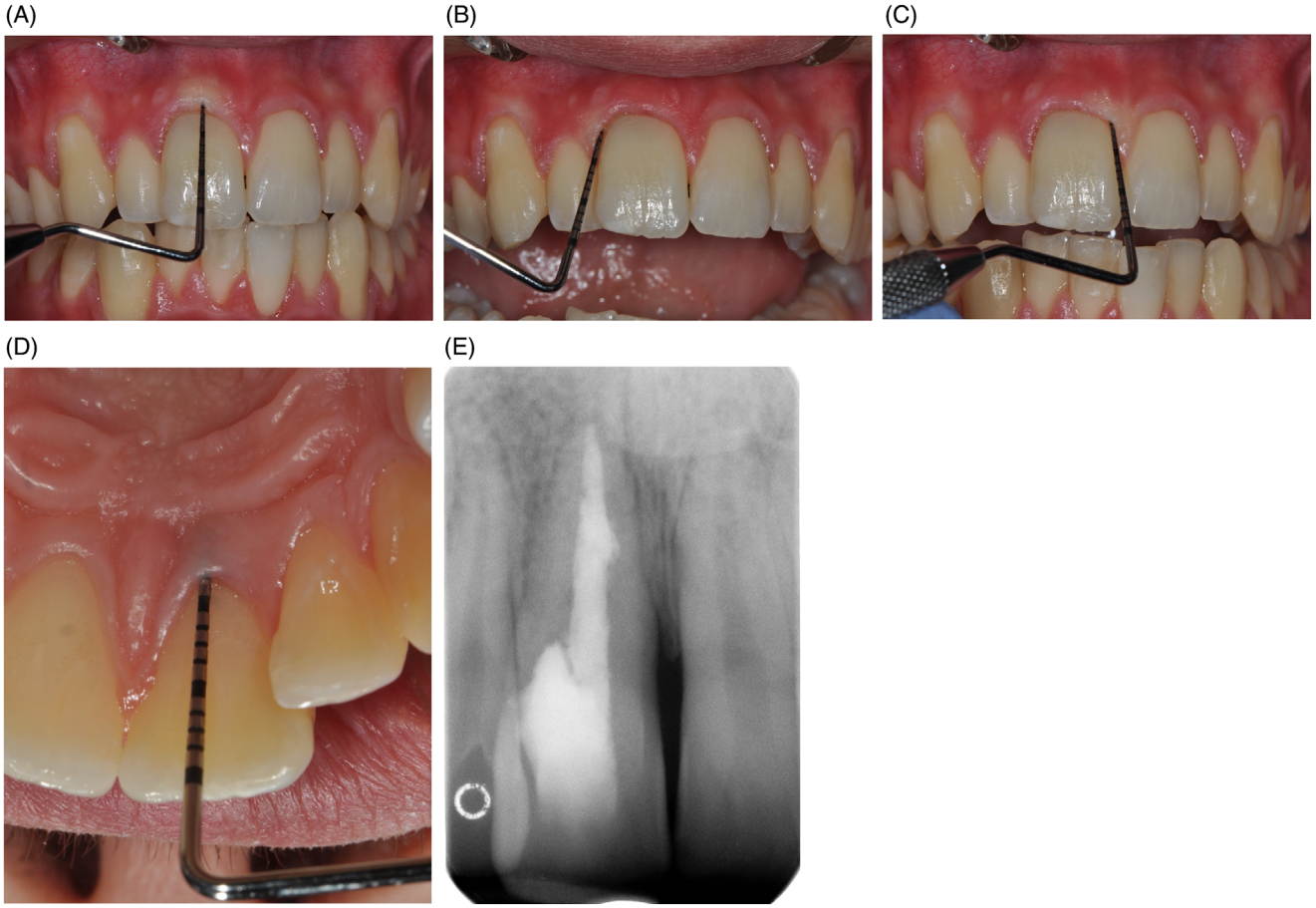


FIGURE 5 After 4 years post-treatment, both clinical and radiological assessments showed consistent outcomes. Examination of buccal, interproximal, and palatal (A–D) areas revealed normal sulcus depths. The tissue exhibited a light pink color without any signs of inflammation. Periapical X-rays confirmed the enduring stability of periodontal health (E).

The precise removal of all granulation tissue present in the resorption site from the endodontic access allowed for precise sealing of the resorptive portal, thereby avoiding the need for a surgical flap to reach the resorption space.[20–22] The removal of granulation tissue was performed with chemical (desiccant cleanser) and mechanical (ultrasonics) tools, which allowed the complete removal of soft tissue and a dry environment free from bleeding. The desiccant agent (Hybenx, EPIEN Medical Inc.) used for decontamination of the root surface is a hygroscopic solution of acidified phenols, consisting of 60% sulfonated phenols, 28% sulfuric acid, and 12% water, developed to eradicate dental biofilm. Its effect is associated with the electrostatic interaction between the sulfate group and water molecules, which induces dehydration and disintegration of organic material. These chemical properties also contribute to the reduction of intracanal bleeding, as they facilitate rapid desiccation of debris at the damaged vascular bed interface within the root canal. The sulfate groups of the product molecularly remove water from the detritus at this interface, resulting in instant denaturation of the tissue debris. This process can lead to coagulation in the vascular bed and subsequent hemostasis.[23]

Moreover, this chemical property causes the denaturation of biofilm, as sulfonic and sulfuric acids rapidly extract water from the matrix, leading to the detachment of biofilm materials from the root surface. This facilitates root instrumentation and disinfection.[23] The use of calcium hydroxide was not implemented in the protocol because bacteria may have persisted after calcium hydroxide intracanal treatment due to intrinsic resistance, anatomical barriers, neutralization by tissues or bacteria, insufficient exposure time, and adaptive changes in gene expression allowing survival in unfavorable conditions.[24–26] Ultimately, composite were preferred as restorative material because of improved seal and micropenetration.[26,27]

After completing the endodontic treatment, due to the lack of established literature on the damage and survival of fibers affected by an acute EPL pathology, the question persisted regarding whether to treat or leave untreated the root affected by the inflammatory process. The decision not to smooth/instrument the root was made considering two variables: (1) the onset time of the acute abscess and (2) the gingiva-root healing pattern.

The onset time of the acute abscess, occurring suddenly without prodromal symptoms 24 h before the visit, raised

hopes that some or all periodontal fibers might have maintained vitality despite the presence of inflammatory cells and their extracellular enzymes.

In this case, the hope was for the formation of a connective reattachment between the tooth and root, aligning with knowledge about histological healing of re-implanted teeth.[28,29] According to the same studies, the loss of fibers would not allow for their reattachment to the dental root but would only result in the formation of long junctional epithelium, providing protective functions in favorable cases or leading to root resorption or ankylosis in more unfavorable instances.[28,29] The decision not to instrument the root proved suitable and perhaps fortunate. However, even after healing, the consistency and color of the associated gingival tissues, coupled with a physiological probing suggested the possibility of connective reattachment. Yet, it was impossible to definitively establish the type of histological healing that occurred on the dental root.

CONCLUSION

Accurate diagnosis forms the cornerstone in addressing endo-periodontal lesions. Established classification systems facilitated an in-depth understanding of the etiologic factors that led to disease and provided guidance on the therapy.

Assessing the final outcomes of this case, the clinical dilemma of choosing to perform or avoid root instrumentation in the treatment of an acute endoperiodontal abscess in a young periodontally healthy patient seems to find a positive resolution in the decision to refrain from root instrumentation during the acute phase of the lesion. The decision regarding the necessity of root instrumentation can be deferred until a new diagnostic phase is conducted after the healing of the endodontic etiology.

AUTHOR CONTRIBUTIONS

All authors significantly contributed to the realization of the study and approved the manuscript before submission. **Riccardo Pace**: Conceptualization; formal analysis; supervision; writing—original draft; writing—review and editing. **Riccardo Di Gianfilippo**: Formal analysis; methodology; writing—original draft; writing—review and editing. **Debora Franceschi**: Formal analysis; methodology; supervision; writing—review and editing. **GiovanPaolo Pini Prato**: Conceptualization; writing—original draft; writing—review and editing.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

REFERENCES

1. Herrera D, Retamal-Valdes B, Alonso B, Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *J Periodontol*. 2018;89(1):S85-S102. Suppl. doi:10.1002/JPER.16-0642
2. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol*. 2018;89(1):S173-S182. Suppl. doi:10.1002/JPER.17-0721
3. Abbott PV, Lin S. Tooth resorption-Part 2: a clinical classification. *Dent Traumatol*. 2022;38(4):267-285. doi:10.1111/edt.12762
4. Lin S, Moreinos D, Kaufman AY, Abbott PV. Tooth Resorption—Part 1: the evolvement, rationales and controversies of tooth resorption. *Dent Traumatol*. 2022;38(4):253-266. doi:10.1111/edt.12757
5. Patel S, Saberi N, Pimental T, Teng PH. Present status and future directions: root resorption. *Int Endod J*. 2022;55(4):892-921. Suppl. doi:10.1111/iej.13715
6. Heithersay GS. Clinical, radiologic, and histopathologic features of invasive cervical resorption. *Quintessence Int*. 1999;30(1):27-37.
7. Heithersay GS. Invasive cervical resorption: an analysis of potential predisposing factors. *Quintessence Int*. 1999;30(2):83-95.
8. Heithersay GS. Invasive cervical resorption. *Endodontic Topics*. 2004;7(1):73-92.
9. European Society of Endodontology, Patel S, Durack C, et al, European Society of Endodontology. European Society of Endodontology position statement: the use of CBCT in endodontics. *Int Endod J*. 2014;47(6):502-504. doi:10.1111/iej.12267
10. American Association of Endodontists, American Academy of Oral Maxillofacial Radiology. Use of cone-beam computed tomography in endodontics. Joint Position Statement of the American Association of Endodontists and the American Academy of Oral and Maxillofacial Radiology. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2011;111(2):234-237. doi:10.1016/j.tripleo.2010.11.012
11. Dahlen G. Microbiology and treatment of dental abscesses and periodontal-endodontic lesions. *Periodontol 2000*. 2002;28:206-239. doi:10.1034/j.1600-0757.2002.280109.x
12. Herrera D, Alonso B, de Arriba L, Santa Cruz I, Serrano C, Sanz M. Acute periodontal lesions. *Periodontol 2000*. 2014;65(1):149-177. doi:10.1111/prd.12022
13. Herrera D, van Winkelhoff A, Sanz M. Abscesses in the periodontium. In: Lang NP, Lindhe J, eds. *Clinical Periodontology and Implant Dentistry*. 6 ed. Wiley; 2015:463-471. chap Chapter 24.
14. Bergenholtz G, Ricucci D, Beatrice S-G, Matthias Z. Endodontics and periodontics. In: Lang NP, Lindhe J, eds. *Clinical Periodontology and Implant Dentistry*. 6 ed. Wiley; 2015:830-860. chap Chapter 41.
15. Muzzi L, Rotundo R, Cairo F, Pini Prato G, Cattabriga M, Pace R. Periodontic-endodontic lesions: diagnostic and therapeutic indications. *Minerva Stomatol*. 2002;51(1-2):41-48. [Article in Italian: Lesioni endo parodontali. Indicazioni diagnostiche e terapeutiche.].
16. Benavides E, Krecioch JR, Connolly RT, et al. Optimizing radiation safety in dentistry: clinical recommendations and regulatory considerations. *J Am Dent Assoc*. 2024;155(4):280-293. doi:10.1016/j.adaj.2023.12.002. .e4.
17. Mavridou AM, Bergmans L, Barendregt D, Lambrechts P. Descriptive analysis of factors associated with external cervical resorption. *J Endod*. 2017;43(10):1602-1610. doi:10.1016/j.joen.2017.05.026
18. Patel S, Mavridou AM, Lambrechts P, Saberi N. External cervical resorption-part 1: histopathology, distribution and presentation. *Int Endod J*. 2018;51(11):1205-1223. doi:10.1111/iej.12942
19. Mavridou AM, Hilkens P, Lambrichts I, et al. Is hypoxia related to external cervical resorption? A case report. *J Endod*. 2019;45(4):459-470. doi:10.1016/j.joen.2018.12.013
20. Pace R, Giuliani V, Nieri M, Di Nasso L, Pagavino G. Mineral trioxide aggregate as apical plug in teeth with necrotic pulp and immature apices: a 10-year case series. *J Endod*. 2014;40(8):1250-1254. doi:10.1016/j.joen.2013.12.007
21. Pace R, Giuliani V, Pagavino G. Mineral trioxide aggregate as repair material for furcal perforation: case series. *J Endod*. 2008;34(9):1130-1133. doi:10.1016/j.joen.2008.05.019
22. Pace R, Giuliani V, Pagavino G. Mineral trioxide aggregate in the treatment of external invasive resorption: a case report. *Int Endod J*. 2008;41(3):258-266. doi:10.1111/j.1365-2591.2007.01338.x

23. Pace R, Di Nasso L, Nizzardo A, Tauro L, Pagavino G, Giuliani V. The effects of a new decontaminant solution on root canal bleeding during endodontic treatment: a randomized controlled study. *J Biol Regul Homeost Agents*. 2019;33(3):1-9. Suppl. 1. DENTAL SUPPLEMENT.
24. Siqueira JF Jr, Lopes HP. Mechanisms of antimicrobial activity of calcium hydroxide: a critical review. *Int Endod J*. 1999;32(5):361-369. doi:10.1046/j.1365-2591.1999.00275.x
25. Sathorn C, Parashos P, Messer H. Antibacterial efficacy of calcium hydroxide intracanal dressing: a systematic review and meta-analysis. *Int Endod J*. 2007;40(1):2-10. doi:10.1111/j.1365-2591.2006.01197.x
26. Bedran NR, Nadelman P, Magno MB, et al. Does calcium hydroxide reduce endotoxins in infected root canals? Systematic review and meta-analysis. *J Endod*. 2020;46(11):1545-1558. doi:10.1016/j.joen.2020.08.002
27. Schwartz RS, Fransman R. Adhesive dentistry and endodontics: materials, clinical strategies and procedures for restoration of access cavities: a review. *J Endod*. 2005;31(3):151-165. doi:10.1097/01.don.0000155222.49442.a1
28. Lindhe J, Nyman S, Karring T. Connective tissue reattachment as related to presence or absence of alveolar bone. *J Clin Periodontol*. 1984;11(1):33-40.
29. Karring T, Isidor F, Nyman S, Lindhe J. New attachment formation on teeth with a reduced but healthy periodontal ligament. *J Clin Periodontol*. 1985;12(1):51-60.

How to cite this article: Pace R, Di Gianfilippo R, Franceschi D, Pini Prato GP. A clinical dilemma: Performing or avoiding root instrumentation in the treatment of the acute phase of endodontic-periodontal lesions? A case report. *Clin Adv Periodontics*. 2025;15:130–138. https://doi.org/10.1002/cap.10297