

Review

The Interface of Endodontics and Periodontics: Managing Periapical and Periodontal Lesions

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Abstract

Combined endodontic-periodontal lesions present a complex interplay between pulpal and periodontal diseases, requiring a multidisciplinary approach for effective management. These lesions often arise due to anatomical pathways, such as accessory canals and dentinal tubules, which facilitate microbial spread between the two tissues. Pathogenic bacteria and inflammatory mediators contribute to the progression of these conditions, complicating diagnosis and treatment. Advances in diagnostic tools, including cone-beam computed tomography and microbiological analyses, have improved the ability to differentiate between endodontic and periodontal origins, although challenges remain in cases with overlapping clinical presentations. Treatment planning for these lesions requires addressing the primary etiology while simultaneously managing secondary complications. Endodontic therapies such as root canal treatment, coupled with periodontal interventions like scaling, root planing, and regenerative procedures, are integral to restoring both tissues. Emerging approaches, including the use of biomaterials, stem cell therapy, and antimicrobial agents, enhance the ability to manage combined lesions more effectively. However, systemic factors such as diabetes and smoking significantly influence treatment outcomes, necessitating a holistic approach that incorporates patient education and management of underlying health conditions. Prognosis is determined by factors such as the extent of bone loss, microbial control, and patient adherence to oral hygiene and follow-up care. While regenerative techniques offer promising outcomes in restoring both pulp and periodontal tissues, long-term success depends on accurate diagnosis, coordinated care, and addressing systemic health influences. The integration of advanced technologies and interdisciplinary strategies underscores the importance of collaboration between endodontics and periodontics in managing these challenging lesions, with a focus on improving patient outcomes and preventing recurrence.

Keywords: *Endodontics, Periodontics, Combined lesions, Diagnostic challenges, Regenerative therapy*

Introduction

The interface between endodontics and periodontics represents a critical aspect of dental care, as both disciplines converge in managing lesions affecting the periapical and periodontal regions. The anatomical and functional proximity of the pulp and periodontal tissues often leads to interdependence in disease progression, complicating diagnosis and treatment planning. Periapical lesions, primarily of endodontic origin, and periodontal lesions, primarily related to bacterial plaque accumulation, may overlap, resulting in what are referred to as combined endodontic-periodontal lesions (1). These lesions pose significant diagnostic challenges, as clinical and radiographic findings alone may be insufficient to determine their origin or the extent of their involvement.

The pathophysiology of combined lesions is multifactorial, involving the interaction between microbial infections, host immune responses, and structural communication pathways such as accessory canals and dentinal tubules. Studies suggest that untreated or inadequately treated endodontic infections can exacerbate periodontal disease, while severe periodontitis may compromise pulp vitality through retrograde pathways (2). Consequently, understanding the bidirectional relationship between these two dental conditions is essential for successful management.

Advances in diagnostic tools, including cone-beam computed tomography (CBCT) and molecular microbiological analyses, have improved the ability to differentiate between endodontic and periodontal etiologies (3). However, treatment success often depends on a multidisciplinary approach that integrates both endodontic and periodontal therapies. Contemporary treatment strategies emphasize the use of regenerative procedures and biomaterials to restore both tissues, highlighting the importance of innovation in achieving optimal outcomes (4). This review aims to explore the interface of endodontics and periodontics with a focus on managing periapical and periodontal lesions.

Review

The management of periapical and periodontal lesions at the interface of endodontics and periodontics requires a nuanced understanding of their shared etiological factors and complex interactions. These lesions can originate independently or as a result of pathological communication between the pulp and periodontal tissues, necessitating a multidisciplinary approach to treatment. Accessory canals and dentinal tubules serve as anatomical pathways through which microbial infections can spread, contributing to the overlap of these conditions (5).

Diagnostic differentiation is often challenging due to overlapping clinical presentations. Advanced imaging techniques, such as cone-beam computed tomography (CBCT), have emerged as indispensable tools for identifying the extent and origin of these lesions, improving diagnostic accuracy and informing treatment strategies. Additionally, microbial analyses have revealed distinct bacterial profiles in endodontic and periodontal infections, enabling targeted antimicrobial therapies (6). Treatment planning must address the primary etiology while concurrently managing any secondary complications. Endodontic therapy, when combined with periodontal interventions such as scaling and regenerative techniques, has shown improved clinical outcomes. Emerging approaches, including the use of biomaterials and growth factors, offer promise for simultaneous repair of pulp and periodontal tissues. Continued research into these integrated strategies is essential for optimizing patient care and advancing clinical outcomes.

Etiological Factors Linking Periapical and Periodontal Lesions

Understanding the etiological factors linking periapical and periodontal lesions is fundamental to managing these complex dental conditions. The anatomical proximity of the pulp and periodontal tissues allows for pathological communication, creating a bidirectional pathway for disease progression. This interrelationship is influenced by structural features such as lateral canals, dentinal

tubules, and apical foramina, which facilitate the spread of infections and inflammatory mediators between the two tissues (5, 7). Studies indicate that accessory canals, which are present in a significant proportion of teeth, are major conduits for microbial migration, underscoring their role in combined lesions.

Microbial factors are central to the development of both periapical and periodontal diseases. The polymicrobial nature of these infections often involves distinct but overlapping bacterial populations. In periapical lesions, anaerobic bacteria such as *Porphyromonas endodontalis* dominate, whereas periodontal lesions typically harbor pathogens such as *Aggregatibacter actinomycetemcomitans* (8). These bacterial species not only drive localized inflammation but also promote systemic immune responses that exacerbate tissue destruction. The shared presence of bacterial biofilms and their metabolic byproducts further complicates the management of combined lesions.

Host immune response plays a critical role in the pathogenesis of these conditions. Inflammatory mediators such as cytokines, chemokines, and prostaglandins contribute to the breakdown of alveolar bone and soft tissues. Both periapical and periodontal infections are associated with elevated levels of interleukin-1 β and tumor necrosis factor-alpha, which amplify the inflammatory cascade (9). These mediators also affect adjacent tissues, fostering the progression of disease from one compartment to another. Moreover, systemic factors, including diabetes mellitus and smoking, have been shown to exacerbate the host immune response, linking periapical and periodontal disease more closely in affected individuals.

The mechanical impact of trauma and occlusal stress further complicates the etiological landscape. Trauma may lead to pulpal necrosis and subsequent periapical inflammation, while also disrupting the periodontal ligament and alveolar bone. Additionally, iatrogenic factors, such as over-instrumentation during endodontic procedures, may inadvertently introduce irritants into the periodontal

space, creating secondary lesions (10). These mechanical and procedural factors highlight the need for precise clinical management to prevent cross-contamination between the endodontic and periodontal systems. The interconnection between periapical and periodontal lesions is influenced by a multifactorial interplay of anatomical structures, microbial activity, host immune response, and mechanical factors. Understanding these complex relationships is essential for developing comprehensive treatment strategies that address both the primary and secondary etiological factors of these challenging dental conditions.

Diagnostic Challenges in Combined Endodontic-Periodontic Lesions

Accurate diagnosis of combined endodontic-periodontic lesions is often complicated due to their overlapping clinical presentations and multifactorial etiology. These lesions can arise independently or as a result of pathological communication between the pulp and periodontal tissues, making it difficult to determine their primary origin. Clinically, symptoms such as pain, swelling, and increased tooth mobility may appear similar in both endodontic and periodontal diseases, further complicating the diagnostic process (11). Moreover, radiographic evaluations often reveal overlapping patterns of bone loss that are insufficient for differentiating between these two conditions without additional investigations.

A major diagnostic challenge lies in the limitations of traditional radiography. Two-dimensional radiographs, such as periapical and panoramic films, can only provide limited information on the spatial extent and nature of bone destruction. In combined lesions, this limitation is particularly problematic as the lesions may involve both the apex and lateral aspects of the root. Cone-beam computed tomography (CBCT) has emerged as a critical tool for overcoming these challenges by providing three-dimensional imaging that allows for a more detailed evaluation of bone defects, root morphology, and potential communication pathways (12). Despite its advantages, CBCT has its own limitations, including cost and the potential for

radiation exposure, which may limit its widespread use.

In addition to imaging, clinical diagnostic tests are essential for distinguishing between endodontic and periodontal origins. Pulp vitality testing is frequently employed to assess the functional status of the pulp. A non-vital pulp is a strong indicator of an endodontic origin, whereas a vital pulp suggests a periodontal etiology. However, false positives and negatives can occur due to factors such as calcified canals or inadequate conduction of thermal or electrical stimuli (13). Similarly, periodontal probing can help identify periodontal pockets indicative of periodontal disease, but isolated deep pockets may also be caused by endodontic lesions, particularly in cases of vertical root fractures or accessory canals.

Microbiological analysis offers another diagnostic avenue but comes with its own set of challenges. The distinct microbial profiles of endodontic and periodontal infections can theoretically help differentiate between these conditions. For instance, obligate anaerobes such as *Porphyromonas endodontalis* are commonly associated with endodontic infections, while periodontal lesions often involve pathogens like *Porphyromonas gingivalis* and *Treponema denticola* (14). However, obtaining accurate microbial samples without contamination from adjacent tissues is difficult, and the clinical utility of such analyses remains limited.

The interplay between systemic conditions and diagnostic complexity cannot be overlooked. Diseases like diabetes mellitus and osteoporosis may exacerbate both endodontic and periodontal lesions, altering their clinical presentation. Additionally, iatrogenic factors, including previous treatments such as overextended root fillings or inadequate scaling and root planing, can obscure the primary etiology and complicate diagnosis. These challenges underscore the need for a multidisciplinary approach that combines advanced diagnostic technologies, clinical expertise, and a thorough understanding of the patient's systemic health to navigate the complexities of combined lesions.

Interdisciplinary Approaches to Treatment Planning

Effective management of combined endodontic-periodontic lesions relies heavily on an interdisciplinary approach that integrates expertise from both endodontics and periodontics. Collaboration between these specialties ensures that both the pulpal and periodontal components of the lesion are addressed, optimizing clinical outcomes. Treatment planning begins with a thorough assessment of the lesion's primary etiology and the extent of tissue damage. Establishing whether the lesion originates from the pulp, the periodontium, or is a true combined lesion significantly influences the treatment pathway (15). Endodontic and periodontal therapies must be carefully sequenced to address the source of infection while facilitating healing in adjacent tissues.

Endodontic therapy is often the first line of treatment in lesions with a primary endodontic origin, as resolving pulpal infection can significantly improve the periodontal condition. Root canal therapy involves the thorough debridement of the pulp space, disinfection using antimicrobial agents, and obturation to prevent reinfection. Adjunctive treatments, such as calcium hydroxide dressings, are used in cases of extensive infection to reduce microbial load and promote periapical healing (16). However, isolated endodontic treatment may not be sufficient in lesions with significant periodontal involvement, necessitating concurrent periodontal interventions. Periodontal therapy aims to restore the health of the periodontium through mechanical debridement, scaling, and root planing. These procedures remove bacterial biofilms and calculus that contribute to periodontal inflammation and bone loss. In cases of advanced bone destruction, regenerative procedures such as guided tissue regeneration (GTR) are often employed. GTR utilizes barrier membranes to facilitate selective repopulation of the defect by periodontal ligament and bone-forming cells, promoting tissue regeneration (17). The success of such procedures depends on controlling the endodontic infection, as unresolved pulpal infection can hinder periodontal healing.

Emerging treatment modalities emphasize the integration of regenerative technologies that simultaneously address both pulp and periodontal tissues. The use of stem cell-based therapies and bioactive materials, such as mineral trioxide aggregate (MTA) and bioceramics, has shown promise in achieving this goal. These materials not only promote root-end healing but also support the regeneration of periodontal structures. Such advances highlight the need for a coordinated approach between specialties to leverage these technologies effectively (18). The role of systemic health in treatment planning cannot be ignored, as conditions such as diabetes and smoking adversely impact treatment outcomes. Addressing these factors requires input from other healthcare providers, including general physicians or endocrinologists, further emphasizing the interdisciplinary nature of managing combined lesions. Regular follow-up and patient education are also integral components of treatment planning, ensuring long-term success and the prevention of recurrence.

Prognostic Factors Influencing Treatment Outcomes

The prognosis of combined endodontic-periodontic lesions is shaped by a complex interplay of biological, clinical, and systemic factors. These factors not only dictate the immediate response to treatment but also influence long-term outcomes. One of the most critical determinants of success is the accurate identification and management of the lesion's primary etiology. In cases where the primary cause remains unresolved, secondary complications can compromise the overall prognosis and increase the likelihood of recurrence (19). Thus, achieving a definitive diagnosis is foundational to improving treatment outcomes.

The extent and type of bone loss significantly influence prognosis. Horizontal bone loss, commonly associated with periodontal disease, often has a better outcome when managed with conventional periodontal therapies. In contrast, vertical or combined defects present greater challenges, particularly when associated with endodontic pathology. Regenerative approaches,

including the use of growth factors and barrier membranes, have shown potential to improve outcomes in such cases, but success depends heavily on the size and morphology of the defect (20). Furthermore, the preservation of adequate root structure is vital, as excessive loss of root length or diameter can render the tooth unsalvageable.

Microbial control is another critical prognostic factor. The success of both endodontic and periodontal therapies hinges on the effective elimination of pathogenic microorganisms. Persistent infection within the root canal system, often involving resistant bacteria such as *Enterococcus faecalis*, is a common cause of endodontic treatment failure. Similarly, the presence of complex subgingival biofilms in periodontal pockets can lead to chronic inflammation and delayed healing (8, 21). Advances in antimicrobial delivery systems, such as locally applied antibiotics and irrigants, aim to enhance microbial control, but their efficacy depends on the meticulous execution of debridement procedures.

Systemic health conditions, including diabetes, cardiovascular disease, and smoking, play a pivotal role in influencing healing and treatment outcomes. Diabetes, for instance, is associated with impaired immune response and delayed wound healing, often leading to poorer outcomes in periodontal and endodontic treatments. Smoking exacerbates these issues by reducing blood flow and inhibiting the proliferation of fibroblasts and osteoblasts, which are essential for tissue regeneration (21). Patients with these systemic conditions require tailored treatment plans that address both their dental and medical needs to optimize healing potential.

Patient compliance and follow-up care are equally critical in determining prognosis. Adherence to oral hygiene practices and regular dental visits are essential for preventing reinfection and maintaining the stability of treatment outcomes. Non-compliance, whether due to lack of understanding or neglect, can undermine even the most meticulously executed treatment plan. This highlights the need for thorough patient education and engagement in their own care, as well as the importance of periodic

evaluations to identify and address any emerging issues promptly.

Conclusion

In managing combined endodontic-periodontal lesions, a comprehensive approach that addresses both etiological and systemic factors is essential. Accurate diagnosis, effective microbial control, and interdisciplinary collaboration play pivotal roles in ensuring successful treatment outcomes. Emerging technologies and regenerative techniques offer promising avenues for improving prognosis. Ultimately, patient compliance and tailored care plans remain fundamental to achieving long-term stability and preventing recurrence.

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Conflict of interest

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Ethical Consideration

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Data availability

Data that supports the findings of this study are embedded within the manuscript.

Author Contribution

The authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

References

1. Simring M, Goldberg M. The pulpal pocket approach: retrograde periodontitis. *The Journal of Periodontology*. 1964;35(1):22-48.
2. Seltzer S, Bender I, Ziontz M. The interrelationship of pulp and periodontal disease. *Oral Surgery, Oral Medicine, Oral Pathology*. 1963;16(12):1474-90.
3. Patel S, Dawood A, Ford TP, Whaites E. The potential applications of cone beam computed tomography in the management of endodontic

problems. *International endodontic journal*. 2007;40(10):818-30.

4. Cortellini P, Tonetti MS. Clinical concepts for regenerative therapy in intrabony defects. *Periodontology* 2000. 2015;68(1):282-307.

5. Nair PR. Pathogenesis of apical periodontitis and the causes of endodontic failures. *Critical Reviews in Oral Biology & Medicine*. 2004;15(6):348-81.

6. Kakehashi S, Stanley H, Fitzgerald R. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. *Oral surgery, oral medicine, oral pathology*. 1965;20(3):340-9.

7. Chailertvanitkul P. Pathogenesis of apical periodontitis and the causes of endodontic failures. *Pathogenesis of apical periodontitis and the causes of endodontic failures* 2010.

8. Love R. *Enterococcus faecalis*—a mechanism for its role in endodontic failure. *International endodontic journal*. 2001;34(5):399-405.

9. Graves D, Li J, Cochran D. Inflammation and uncoupling as mechanisms of periodontal bone loss. *Journal of dental research*. 2011;90(2):143-53.

10. Fouad AF. Systemic implications of endodontic infections. *Treatment of Endodontic Infections*. 2022.

11. Moss HD, Toscano N, Holtzclaw D. Recognition and management of odontogenic referred pain. *Gen Dent*. 2009;57:388-91.

12. Scarfe WC, Levin MD, Gane D, Farman AG. Use of cone beam computed tomography in endodontics. *International journal of dentistry*. 2009;2009(1):634567.

13. Fernandes M, de Ataíde I. Nonsurgical management of periapical lesions. *Journal of Conservative Dentistry and Endodontics*. 2010;13(4):240-5.

14. SIQUEIRA JF, RÔÇAS IN. Microbiology and treatment of endodontic infections. *Cohen's Pathways of the Pulp*. 2011:559-600.

15. Bender I, Seltzer S. The effect of periodontal disease on the pulp. *Oral Surgery, Oral Medicine, Oral Pathology*. 1972;33(3):458-74.

16. Segura-Egea JJ, Martín-González J, Castellanos-Cosano L. Endodontic medicine: connections between apical periodontitis and systemic diseases. *International endodontic journal*. 2015;48(10):933-51.
17. Cortellini P, Prato GP, Tonetti MS. Periodontal regeneration of human intrabony defects with bioresorbable membranes. A controlled clinical trial. *Journal of Periodontology*. 1996;67(3):217-23.
18. Parirokh M, Torabinejad M. Mineral trioxide aggregate: a comprehensive literature review—part III: clinical applications, drawbacks, and mechanism of action. *Journal of endodontics*. 2010;36(3):400-13.
19. Ørstavik D. Apical periodontitis: microbial infection and host responses. *Essential endodontology: prevention and treatment of apical periodontitis*. 2019:1-10.
20. Polimeni G, Xiropaidis AV, Wikesjö UM. Biology and principles of periodontal wound healing/regeneration. *Periodontology* 2000. 2006;41(1).
21. Grossi SG, Zambon JJ, Ho AW, Koch G, Dunford RG, Machtei EE, et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of periodontology*. 1994;65(3):260-7.