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From Diagnosis to Treatment: A Review of Endo-Perio Lesions

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Abstract

Endodontic and periodontal infections often present clinical similarities, making it challenging to distinguish between them. Both types of infections share common features, complicating the diagnostic process. Approximately 50% of tooth loss is attributed to either endodontic infection, periodontal disease, or a combination of the two, known as endodontic-periodontal lesions (EPL). These combined EPLs arise due to the close anatomical and functional relationship between endodontic and periodontal tissues, which share the same embryologic and anatomic origins.

When endodontic and periodontal infections coexist or exhibit similar signs and symptoms, pinpointing the primary source of infection is crucial for proper treatment planning. This diagnostic challenge often

necessitates an interdisciplinary treatment approach to ensure comprehensive care and optimal outcomes. This review aims to explore the complexities of endodontic and periodontal infections, particularly when they occur simultaneously or when one influences the other. The review analyzes contemporary literature on endodontic-periodontal interactions, providing insights into current knowledge and advancements in this field. It also discusses strategies to facilitate accurate diagnosis and effective treatment planning for these challenging conditions.

Keywords: Endodontic, Periodontic, Classification, Diagnosis, Endo-perio, Infection.

Introduction

Recognizing the complex interactions between endodontic and periodontal tissues is crucial for accurate diagnosis and effective treatment planning. This understanding hinges on the role of bacteria and the interrelationship between these tissues, which directly influences treatment decisions and ultimately leads to better patient outcomes.

The connection between periodontal and pulpal diseases has been a topic of interest for nearly a century. In 1924, Coyler hypothesized a relationship between the two, suggesting a cause-and-effect link[1]. This was further expanded in 1927 by Cahn, who proposed that 'side canals' (now known as lateral or accessory canals) could serve as pathways for infections to spread from periodontal tissues to the pulp.

In the 1960, researchers began to investigate this relationship more thoroughly. Simring and Goldberg, in 1964, coined the term 'retrograde periodontitis' to describe periodontal disease that starts in the dental pulp and progresses from the apical region towards the gingival margin. Their work highlighted the bidirectional nature of these infections and underscored the importance of considering both endodontic and periodontal factors in diagnosis and treatment.

Microbiology

The microbial flora associated with endodontic and periodontal diseases varies, though some bacteria are common in both environments.[2] In periodontal pockets, the dominant flora includes rods and motile organisms, while root canals primarily harbor rods and cocci.[3] Specific bacteria such as Actinobacillus actinomycetemcomitans, Bacteroides forsythus, Eikenella corrodens, Fusobacterium nucleatum, Porphyromonas gingivalis, Prevotella intermedia, and Treponema denticola are commonly found in both chronic apical periodontitis and chronic adult periodontitis.

Additionally, fungal species like Candida albicans are present in both endodontic infections and subgingivally in patients with adult periodontitis. Viral pathogens, including cytomegalovirus, Epstein-Barr virus, and herpes virus, may also contribute to periodontal and endodontic diseases. These viruses can exacerbate periodontal conditions by increasing the number of periodontal pathogens in periodontal pockets and potentially affecting pulpal and periapical tissues.[4]

Pathways of the Pulp and Periodontal Tissues

The dental pulp and periodontal structures share a close embryonic, anatomical, and functional connection, both originating from ectomesenchymal tissue. Anatomically, these tissues are interconnected through the apical foramina, dentinal tubules, and lateral or accessory canals. Inflammatory changes in the periodontal ligament can occur due to pulpal inflammation even before the pulp becomes completely necrotic.

Several factors can predispose individuals to endodontic-periodontal lesions, including:

1. Tooth Malalignment: Misaligned teeth can complicate cleaning and maintenance, increasing the risk of infections.
2. Anatomical Variations in Roots: The presence of a multirooted tooth in a position usually occupied by a single-rooted tooth, or additional roots (separate or fused) in multirooted teeth, can create niches for bacterial colonization.
3. Additional Canals and Root Structure Changes: Extra canals and variations in root structure can harbor bacteria, making infections more likely and treatment more challenging.
4. Cervical Enamel Projections: These projections into the furcation area of multirooted teeth can facilitate the spread of infection from periodontal to endodontic tissues.
5. Large Lateral (Accessory) Canals: Accessory canals, especially in the coronal and middle sections of roots, can serve as pathways for bacterial spread between the pulp and periodontal tissues.

There are multiple possible routes of communication between the dental pulp and the periodontal tissues. Predominantly, these are anatomical in nature, though some may be of pathological origin or occasionally result from iatrogenic factors.[5]

Dentinal Tubules

Dentinal tubules can potentially create a connection between the pulp and periodontal tissues;[6] however, they are typically safeguarded externally by enamel in the crown of the tooth and by cementum in the root area. One of the functions of cementum is to block communication between the root canal system and the periodontium through the otherwise exposed dentinal tubules[7]. The cementum layer may be compromised due to developmental defects where it is absent in certain areas, or it can be damaged or removed during dental procedures like scaling and root planing.

The cemento-enamel junction (CEJ), where neither cementum nor enamel shields the dentine, can naturally exist in certain teeth. According to reports, up to 30% of human teeth have this gap, although it usually only happens in patches rather than all the way around the CEJ.[8]

Additionally, dental trauma or dysfunctions such as bruxism may damage the cementum. Dentinal tubules may be exposed if the cementum layer is compromised, opening a channel for bacteria to move between the pulp and the periodontium.

Vital pulps are capable of fighting off potential invading pathogens from the periodontal tissues despite the physical barrier created by the cementum. This is because the pulp has an innate immune response to bacteria and their by-products, and because fluid from the pulp constantly flows outward through the dentinal tubules and into the periodontal tissues.

A continuous, dynamic physical and chemical barrier against possible invaders from the periodontal tissues is provided by this fluid flow. This is only the case with vital teeth, and no such protection is present in necrotic or root-filled teeth where this fluid flow no longer exists, as it is initiated from the pulp. [9]

Lateral And accessory canals

The pulp and periodontal tissues are connected by tiny branches called lateral and auxiliary canals. They arise during odontogenesis when Hertwig's Epithelial Root Sheath (HERS) either surrounds or invades tiny blood vessels.[10] Compared to the middle and coronal thirds of the root, they are more prevalent in the apical third. [11]

De Deus and Horizonte discovered that 17%, 9%, and around 2% of the teeth had lateral canals in the apical, middle, and coronal thirds, respectively.[11]

If periodontal disease causes tissue destruction and infection to the level of a lateral canal a potential pathway for bacterial ingress to the pulp is created. This may lead to pulp inflammation and structural alterations[12], which may eventually end in pulp necrosis.[13]

Apical foramen

Since it is larger than the other anatomical channels, the apical foramen may serve as the primary pathway for communication between the periodontal and pulp tissues. In certain cases, it is possible for bacteria and their by-products to spread from an infected root canal to the tissues around the tooth, leading to the development of apical periodontitis. On the other hand, in cases of advanced periodontal disease, bacteria can pass via the apical foramen from the periodontium to the pulp. If the pulp's defenses are weakened by the invasive bacteria and their by-products, this can lead to pulp necrosis and infection of the pulp.[12]

Developmental grooves and malformations

Radicular grooves, sometimes called palato-gingival grooves, are thought to form during tooth root formation when the enamel organ and HERS fail to fully infold.[14] They are more prevalent in lateral incisors than in central incisors and are more frequently observed on the disto-palatal surface of maxillary incisor teeth.[15] They could also appear on the mandibular second molars' lingual surface.[16]

Radicular grooves can experience localized buildup of plaque and calculus because they are hard for patients to maintain clean and are frequently associated with a deep pocket. Cementum can occasionally be scant or non-existent at the groove's more apical extension. It's also possible to thin the dentine because of the groove's infolding. This could lead to direct or very close communication between the pulp and the bacterial biofilm that invariably develops inside the groove.[14] Even with careful oral hygiene practices, patients often struggle to maintain the cleanliness of these grooves.

Non anatomical pathways

There may also be non-anatomical channels of communication between the periodontal and pulp tissues. These may result from iatrogenic or pathogenic reasons. Cracks in the root or perforating, inflammatory root resorption (internal or external) are examples of pathogenic reasons. A root perforation or furcation that occurs during endodontic or restorative dental operations are examples of iatrogenic causes. Additionally, this will result in direct communication between the periodontium and the contents of the root canal system.[17]

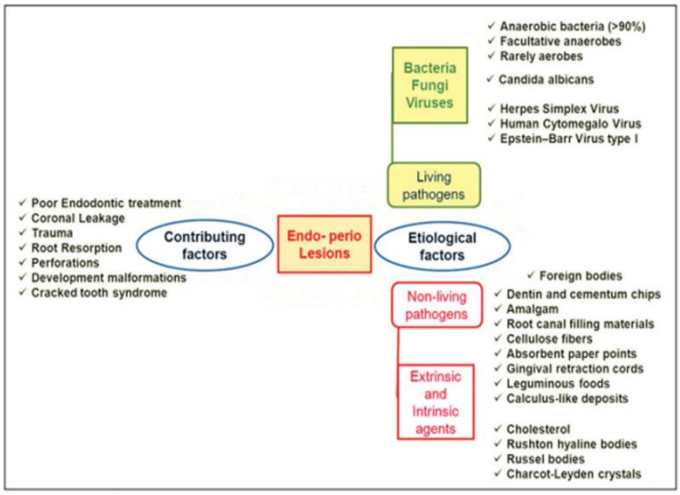


Figure 1: Contributing and etiological factors for Endo-perio lesions.

Classification

Various classification for endo perio lesions are as follows[4]

- Classification recommended by the world workshop for classification of periodontal diseases (1999), Periodontitis Associated with Endodontic Disease :-

1. Endodontic-periodontal lesion,
2. Periodontal-endodontic lesion,
3. Combined lesion.

- Classification by Simon et al :-

1. Primary endodontic lesions,
2. Primary endodontic lesions with secondary periodontal involvement,
3. Primary periodontal lesions,
4. Primary periodontal lesions with secondary endodontic involvement,
5. True combined lesions.

- Classification by Torabinejad and Trope in 1996 based

On the origin of the periodontal pocket:

1. Endodontic origin,
2. Periodontal origin,
3. Combined endo-perio lesion,
4. Separate endodontic and periodontal lesions,
5. Lesions with communication,
6. Lesions with no communication.

- In 2014, Al-Fouza suggested a new endodontic periodontal interrelationship classification, based on the primary disease and its secondary effect.

1. Retrograde periodontal disease

- Primary endodontic lesion with drainage through the periodontal ligament
- Primary endodontic lesion with secondary periodontal involvement

2. Primary periodontal lesion
3. Primary periodontal lesion with secondary endodontic involvement
4. Combined endodontic-periodontal lesion
5. Iatrogenic periodontal lesion

Diagnosis

The diagnosis of primary endodontic and periodontal diseases is uncomplicated. While the pulp in basic periodontal lesions responds to heat and electric testing, the tooth in primary endodontic disorders does not respond to these tests because it is nonvital and diseased. However, radiographically and clinically, true mixed lesions, primary endodontic lesions with secondary periodontal involvement, and primary periodontal lesions with secondary endodontic involvement are comparable. A thorough clinical examination along with procedures including radiography, pulp vitality testing, pocket probing, fistula tracking, and broken tooth testing can yield an accurate diagnosis. [18]

Table 1: Diagnostic procedures used to identify the endo-perio lesion[19]

Examination/Test	Primary Endodontic Lesion	Primary Periodontal Lesion	Primary Endodontic with Secondary Periodontal Lesion	Primary Periodontal with Secondary Endodontic Lesion	True Combined Lesion (Independent Endodontic and Periodontal Lesion
Visual (magnifying loupes and operative microscope can be effective)	Initial clear presence of sinus tract	Inflamed gingival soft tissue/margins around multiple teeth	Plaque/biofilm at the gingival margins of the involved teeth	Plaque/biofilm at the gingival margins of the involved teeth	Plaque, calculus and inflammation with periodontal pockets of varying degrees in the affected area

Pain	Sharp	Usually dull ache	Initially sharp, changing to dull ache	Initially dull ache, sharp pain if acute exacerbation is presen	Dull ache usually; sharp in acute exacerbations if it involves the periodontium
Palpation is positive	Yes	Yes	Yes	Yes	Yes
Tender on percussion	Yes	The severity of the inflammation of the periodontium often correlates with the degree of tenderness	Yes	Yes	Yes
Pulp is vital	No	Yes	No	No	No
Pocket probing	Usually present with deep narrow probing depth	Multiple wide and deep pocket	Presence of multiple wide and deep periodontal pockets	Presence of multiple wide and deep periodontal pockets	Probing reveals the typical mixed periodontal type of probing depths and profuse bleeding in the affected area, typical of periodontiti
Radiographs	Presence of deep carious lesions, or previous inadequate endodontic treatment resulting in periapical pathology with deep pocketing	Vertical bone loss and furcation involvement	Presence of deep carious lesions, or previous inadequate endodontic treatment resulting in periapical pathology with deep pocketing	Presence of deep carious lesions, or previous inadequate endodontic treatment resulting in periapical pathology with deep pocketing	Mixed radiographic findings indicating both periapical and periodontal pathology

Acute exacerbation of chronic apical lesions with persistent necrotic drainage into the gingival sulcus via the periodontal ligament is referred to as primary endodontic lesions.[20] These teeth typically have a history of trauma, caries, or restorative fillings. They exhibit radiolucency in the apical area and do not react to vitality tests. Additionally, when a sinus tract manifests, symptoms including pain, swelling, tenderness to percussion, and tooth mobility point to a pulpal infection rather than a periodontal abscess.

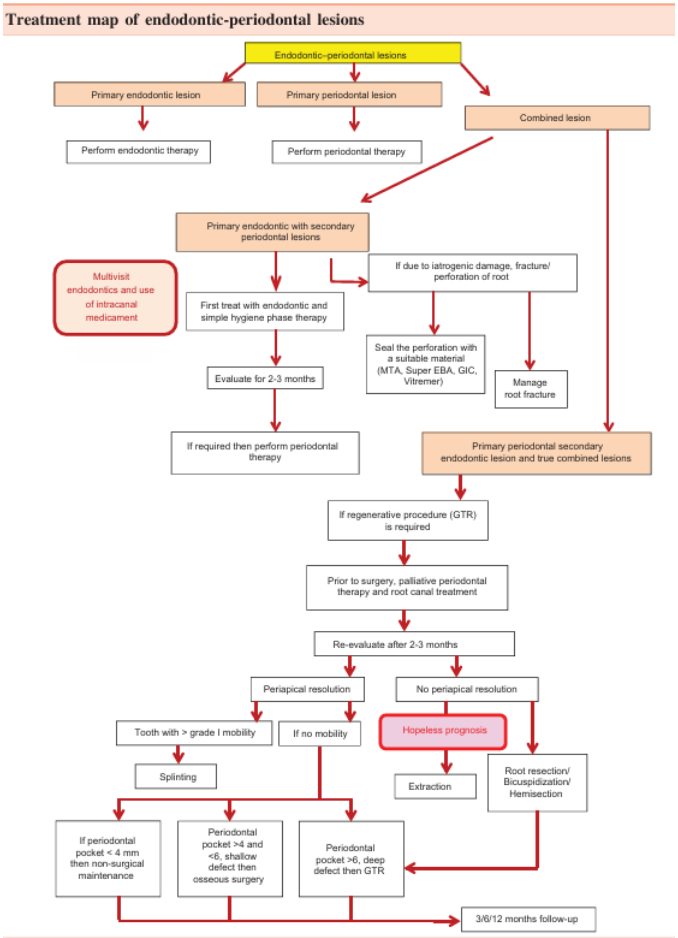
A gutta-percha point inserted into the sinus tract entrance may be useful for the differential diagnosis.[21] Moreover, unlike EPL, which necessitate both endodontic and periodontal therapy, primary endodontic lesions typically recover following a comprehensive endodontic treatment.

Progressive marginal periodontitis leads to primary periodontal lesions. When periodontal disease treatment is delayed, it can spread from the marginal gingiva to the supportive periodontal tissue. In severe situations, a periodontal abscess that resembles the signs of a pulpal illness may develop when both the soft and hard supporting tissues are damaged. As occlusal trauma increases the probing depth surrounding the broken tooth, it can exacerbate the clinical appearance. Since the tooth still responds to vitality tests in the majority of cases, primary periodontal lesions only require periodontal treatment.[22]

When periodontal disease and pulpal necrosis develop together on the same tooth, they are referred to as true combined lesions. These types of lesions are less common than other endodontic or periodontal diseases. The necrotic pulp that contributes to apical periodontitis gradually advances apically where it joins the bone resorption that is already occurring as a result of a periodontal pocket [23]. These lesions' radiographic aspect may mimic a root fracture, in which differential diagnosis is crucial to choose the appropriate course of treatment.

Treatment

Figure 2:



Diagnosis and primary considerations

Soft tissues, alveolar mucosa, and the attached gingiva are examined for signs of inflammation, ulceration, or sinus tract infection.

Vitality testing should be carried out on relevant teeth as well as radiographic examination, paying close attention to shape, location and extension of any lesion, crestal and furcation involvement and signs of fracture or perforation[24].

Radiographic examination will aid in detection of carious lesions, extensive or defective restorations, pulp caps, pulpotomies, previous root canal treatment and

possible mishaps, stages of root formation, canal obliteration, root resorption, root fractures, peri radicular radiolucencies, thickened periodontal ligament, and alveolar bone loss.

Treating primary endodontic lesions

Primary endodontic diseases usually heal following root canal treatment.

Primary endodontic lesions with subsequent periodontal involvement will not be entirely resolved with endodontic treatment. Root/re-root canal therapy is initiated immediately, and the cleansed and shaped root canal is filled with intracanal medicament.

As it is bactericidal, anti-inflammatory and proteolytic, it inhibits resorption and favors repair. It also inhibits periodontal contamination from instrumented canals via patent channels connecting the pulp and periodontium before periodontal treatment removes the contaminants.

The prognosis for primary endodontic lesions is good but worsens in the advanced stages of secondary periodontal involvement.

Treating primary periodontal lesions

Primary periodontal lesions are treated by phase I non-surgical scaling and root planing therapy in the first instance. Periodontal surgery is performed if needed after evaluation of the phase I therapy.

Treating true combined lesions

Acute symptoms may include periodontal abscess formation, discomfort, swelling, pus discharge, pocket formation, and tooth movement. A more chronic response may occur without discomfort, characterized by the formation of a pocket with bleeding on probing or pus exudation.

True combined lesions are essentially two separate conditions, management must involve both endodontic and periodontal treatment.

True-combined lesions are treated initially as primary endodontic lesions with secondary periodontal involvement.

Due to the nature of combined lesions, the amount of bone loss is generally substantial and this has a negative impact on prognosis. This reduced prognosis must be disclosed and taken into consideration when recommending treatment options to patients. In multi rooted teeth where the condition is localized to one particular root, hemi sectioning of the tooth or resection of the affected root may be considered if the tooth is amenable and the patient motivated. Extraction is often the end result in single-rooted teeth[25].Surgical treatments incorporating regenerative periodontal modalities have also been used as a form of reconstructive treatment for more severe periodontal defects, including those that are at, or even beyond the apex of the tooth (apico-marginal defects). The aim of such treatment is regeneration, rather than repair[26]

Abott[27], in a detailed analysis on treatment considerations, recommends the following protocol.

Initial management

Remove existing restorations and caries

Chemo mechanically prepare canals

Medicate canals (depends on symptoms)

Follow-up management

Change intracanal dressing after 3-4 weeks

Provide initial periodontal treatment

Review healing after 3 months

Reassess need for further periodontal treatment

If more periodontal treatment (e.g., surgery) is required,

Change intracanal medication again

If healing response is favourable,

Complete root canal filling

Longer-term management

Defer root filling until after

Need for periodontal surgery assessed

Surgery completed with satisfactory outcome

Surgical treatment of endodontic-periodontal disease: The objectives of such an approach are to remove the infected bio burden in the form of plaque and calculus, and to allow regeneration of the periodontal tissues.

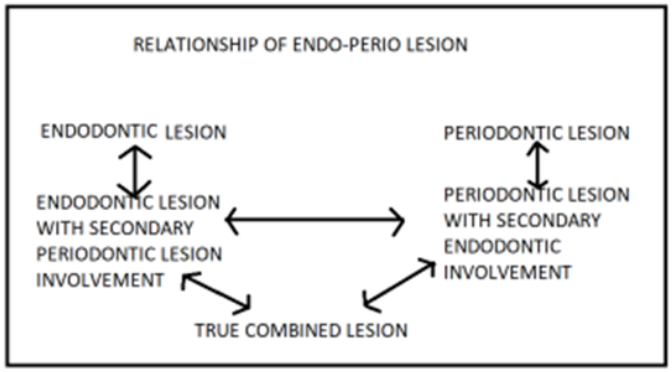


Figure 3: Relationship between endo perio lesion

Prognosis

The prognosis of periodontal lesions is poorer than endodontic lesion and is dependent on the apical extension of the lesion[28]. The prognosis of an affected tooth can also be improved by increasingly bone support which can be achieved by bone grafting and guided tissue regeneration. This is due to the most critical determinant of prognosis being a loss of periodontal support. Overall prognosis has been assessed to be adequate enough to justify further endodontic and restorative treatment and their costs.

Conclusion

A perio-endo lesion can have a variety of etiology, ranging from simple to somewhat complex. Having a thorough understanding of these disease processes is critical for making an accurate diagnosis.

Because of the complexities of these diseases, an interdisciplinary approach involving endodontists, periodontists is indicated. Only with a careful diagnosis can you choose the most effective treatment method and increase the success rate.

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