# Perio-endo Interrelationship

Richa Agrawal<sup>1</sup>, Sonu Peter<sup>2</sup>, Sudheer Yada<sup>3</sup>, Praveen Saxena<sup>4</sup>, Neeharika Soorgani<sup>5</sup>, Shyam Prasad<sup>6</sup>

#### ABSTRACT

Periodontal therapy deals with periodontium in disease. Endodontics deals chiefly with disease of the pulp and periapical tissues. Differentiating between periodontal and endodontic problems can at times be difficult. Both periodontal and endodontic therapy success depends on the elimination of both disease processes, whether they exist independently or as a combined lesion. This paper discusses comprehensive review of various aspects of perio-endo lesions.

Keywords: Combined lesions, Periodontium, Perio-endo lesion.

**How to cite this article:** Agrawal R, Peter S, Yada S, Saxena P, Soorgani N, Prasad S. Perio-endo Interrelationship. Int J Med Oral Res 2018;3(2):11-17.

Source of support: Nil

Conflicts of interest: None

#### INTRODUCTION

The tooth, the pulp tissue, and its supporting structures should be regarded as one biological unit. The interrelationship of these structure impacts each other during health, function, and disease. Simring and Goldberg were the first who described the relationship between periodontal and endodontic disease, in 1964.<sup>[1]</sup> The relationship between pulpal and periodontal disease can be traced back to embryological development, as the origin of both pulp and the periodontium is derived from mesoderm. The developing tooth bud extends out a portion of mesoderm that becomes pulp, while the remaining mesoderm develops into periodontium. In the sequence of root development, strands of mesodermal tissue may get trapped and in future may become lateral and accessory canals. It may also result from dentin formation around existing blood vessels or a loss of continuity of Hertwig's root sheath during the formation of dentin.<sup>[2]</sup> Thus, the pulp and the periodontium are interrelated by three main avenues, i.e., the dentinal tubules, lateral and accessory canals, and the apical foramen.<sup>[3]</sup> As time progresses, most of these communications are

sealed by cementum or secondary dentin, leaving the pulp mainly dependent on the apical foramen for metabolic exchange (and therefore, forming a low compliance system). Some of these portals of communication remain patent providing a pathway through which etiologic agents may pass between the pulp and the periodontium resulting in periodontal lesions or endodontic lesions or both occurring simultaneously.<sup>[2]</sup>

Endodontic lesion can be defined as an inflammatory process within the pulp resulting from noxious agents presents in the root canal system of the tooth, usually a root canal infection. Periodontal lesion can be defined as an inflammatory process in the periodontal tissue resulting from accumulation of dental plaque on the external tooth surface.

Pulpal infection may cause a tissue-destructive process that proceeds from the apical aspect of a tooth toward the gingival margin termed as "retrograde periodontitis." Acute manifestation of root canal infections can result in rapid and widespread destruction of the attachment apparatus.

Necrosis of the pulp tissue can lead into bone resorption at the root apex, furcation areas, or anywhere along the length of the root. Hence, endodontic lesions can also induce a loss of periodontal attachment.

On the other aspect, the pathogenic bacteria and inflammatory products of periodontal disease may affect to the dental pulp through accessory canals, apical foramina, or dentinal tubules causing pulpitis referred to as "retrograde pulpitis."<sup>[4]</sup>

The effect of periodontal inflammation on the pulp is controversial and is at debate. Some studies suggest that periodontal disease has no effect on the pulp, at least until it involves the apex while several studies propose that the effect of periodontal disease on the pulp is degenerative in nature, leading to calcification, fibrosis, and collagen resorption.<sup>[5]</sup>

The function of the tooth is severely compromised when both of these conditions are involved in the disease process. Treatment of disease conditions which involves both of these structures is challenging and frequently requires combining both endodontic and periodontal treatment procedures. Proper diagnosis, identifying the cause, and proper sequencing and performance of the treatment are critical to eliminate both the problems.<sup>[4]</sup>

Treatment and prognosis of primarily endodontic and primarily periodontal disease is very straight

<sup>&</sup>lt;sup>1,3</sup>Reader, <sup>2,4-6</sup>Postgraduate Student

<sup>&</sup>lt;sup>1-6</sup>Department of Periodontics, Mansarovar Dental College, Bhopal, Madhya Pradesh, India

**Corresponding Author:** Dr. Richa Agrawal, Reader, Department of Periodontics, Mansarovar Dental College, Bhopal, Madhya Pradesh, India. e-mail: dr.richaagrawal12@gmail.com

#### Agrawal, et al.

forward, but prognosis of combined forms of perio-endo lesion is relatively more difficult to predict. The success of both periodontal and endodontic therapy depends on the elimination of both disease processes.<sup>[6]</sup>

It is found that the pulpal infection can lead to periodontal changes and periodontal lesions can involve pulp so it is very important to understand the various aspects of perio-endo interrelationship for appropriate diagnosis and treatment planning. The success rate of perio-endo lesion is endorsed to the fact that elimination of periodontal as well as the endodontic source of disease is very essential.

#### BIOLOGICAL CONCEPT OF INTERRELATIONSHIP BETWEEN ENDO-PERIO OR PERIO-ENDO LESIONS

Since perio-endo lesions are closely interrelated, its biological concept of occurrence has to be understood before leading to any conclusion. The interrelation between endo-perio or perio-endo lesions could be related to several aspects.

#### **Embryonic Interrelationship**

The epithelial enamel organ, the dental papilla, and the dental sac are the tissues that form a tooth and its supporting structures. The cells of the dental papilla will form tooth pulp and dentin [Figure 1]. The cells in the dental sac will form cementum and the periodontal ligaments of the tooth.<sup>[7]</sup> The enamel organ plays an key part in root development by forming Hertwig's epithelial root sheath, which molds the shape of the roots and initiates radicular dentin formation. As the root develops, ectomesenchymal channels get incorporated, either due to dentin formation around existing blood vessels or breaks in the continuity of the sheath of Hertwig, to develop accessory or lateral canals. Majority of accessory canals are found in the apical part of the root and lateral canals in the furcation regions of molars. Tubular communication between the pulp and periodontium may occur when dentinal tubules become exposed to the periodontium due to the lack of overlying cementum. These are the pathways that may provide the way by which pathological agents pass between the pulp and periodontium, thereby creating the perio-endo lesion.<sup>[8]</sup>

### Anatomic Interrelationship

The embryonic development gives form to anatomical connections between the pulp and the periodontium, which remains all over life. Following are the several pathways of communication in the development of periodontal endodontic lesions, namely.



Figure 1: Diagram showing the development of tooth bud into various entities

#### Physiological pathways

#### Enamel

The enamel consists mainly of inorganic materials which results in demineralization and caries due to ion exchange between saliva and enamel ions. Deep pits, fissures, and lamella may harbor bacteria and initiate the carious lesions which lead to pulpal disease. The wedging effect of ideal cusp-fossa relations in teeth frequently produces incomplete or complete crown-root fractures which may produce a pulpal-periodontal lesion. Enamel projections into furcation occur frequently and may be directly involved in pocket formation in an area where accessory canals into the pulp may lead to pulpal-periodontal disease. In such cases, the epithelial attachment may migrate to the cementoenamel junction located at some point or points in the furcation area. When separation of epithelium from enamel occurs, a pocket or deep crevice remains in an area which is difficult to clean due to its complex configuration. Inflammatory disease may follow with true pocket formation.<sup>[9]</sup>

#### Cementum

As periodontal disease progresses along the root, the ligament between cementum and bone is destroyed making the surface cementum exposed to the oral environment leaving it vulnerable to bacterial invasion into the dentin. Fissures in the cementum may, if exposed by disease or over instrumentation, serve to channel inflammation into the deeper periodontal tissues. Areas of incomplete calcification may incorporate bacteria and initiate tiny areas of demineralization between the cementum and the dentin. Lateral and accessory canals plus multiple apical foramina transverse the cementum and if exposed it permits bacteria to move from periodontal to pulpal or pulpal to periodontal tissues.<sup>[9]</sup>

#### Dentin

Dentin has a higher proportion of inorganic material than cementum; it is susceptible to bacterial penetration in disease due to its tubular constriction toward pulp from surface. Tubules may be penetrated by bacteria and their toxins from either the periodontal or the pulpal side [Figure 2]. Tubules exposed from the periodontal side by caries, root instrumentation, or fracture may cause an initial pulp lesion which is manifested by sensitivity on the root surface or by pulpitis and finally death of the pulp. From the pulpal side, bacteria from caries may breach to the cemental border if present or to the periodontal tissues. Inflammation through accessory canals in the dentin and cementum can usually be more readily acknowledged than through the dentinal tubules.<sup>[9]</sup>

### Pulp

Pulp horns are more likely to assume clinical significance in the management of a carious lesion, while multiple apical foramina and accessory canals may serve to carry pulpal disease to the periodontal tissues. Spread from periodontal disease to the pulpal tissues particularly is found in association with furcations of molars.

### Periodontal ligament

Inflammation, either periapical or periodontal, may cause some elongation of the tooth due to swelling in the tissues and may create secondary occlusal traumatism. Hence, the ligament loses some of its protective-defensive ability and breaks down as disease advances. Thus, an area of function which is protective against many of the insults from inflammation and trauma may become a channel for the progress of disease.<sup>[9]</sup>



Figure 2: Diagram showing exposure of dentinal tubules

#### Vascular pathway

#### Apical foramen

The apical foramen is the major and most direct route of communication between pulp and periodontium. Following necrosis of the pulp, numerous bacterial products reach the periodontium through the apical foramen, introducing and propagating an inflammatory response there. This leads to the destruction of periodontal tissue fibers and resorption of the adjacent alveolar bone. External resorption of the cementum can also occur simultaneously.<sup>[5]</sup>

#### Lateral canals

In addition to the apical foramen, other branches connecting the main root canal system with the periodontal ligament termed as accessory canal are now used to describe any ramification that connects the root canal system to the periodontal ligament. These canals act as a path of infection.<sup>[10]</sup>

### Tubular pathway: Dentinal tubules

These consist of dentinal tubules which contain the odontoblastic process that extends from the odontoblast at the pulpal dentin border to the dentinoenamel junction or the cement-dentinal junction. Passage of microorganisms between the pulp and periodontal tissues is possible through these tubules when the dentinal tubules are exposed in areas of denuded cementum.<sup>[5]</sup>

# Congenital absence of cementum exposing the dentinal tubules

Dentinal tubules can get exposed due to developmental defects in cementum and can serve as communicating pathway between pulp and periodontal ligament. The congenital absent of cementum in the cementodentinal junction renders about 15,000 dentinal tubules exposed per mm<sup>[3]</sup> of external root surface and creates a channel of communication.<sup>[4]</sup>

### Developmental grooves

Groove begins in the central fossa of maxillary central and lateral incisors crossing over the cingulum and might continue apically down the root and is probably due to the failure of the tooth germ to form another root. When it gets contaminated by bacteria, a self-sustaining infrabony pocket can be formed. This acts as a nidus for accumulation of bacterial biofilm and an avenue for the progression of periodontal disease.<sup>[11]</sup>

International Journal of Medical and Oral Research, July-December 2018;3(2):11-17

#### Agrawal, et al.

### Developmental anomalies such as enamel projections and enamel pearls and palatogingival grooves

Enamel projections are ectopic deposits of enamel that extends into root furcation area formed by infolding of the enamel organ and the Hertwig's epithelial root sheath. It acts as locus of plaque and calculus accumulation, leading to the development of periodontitis with or without pulp pathosis, depending on the depth, extend, and complexity of the groove.<sup>[12]</sup>

### Non-physiological pathways

### Poor endodontic treatment

Poor endodontic treatments are often associated with periradicular inflammation, leading to reinfection and further to treatment failure.

### Poor restoration

Coronal leakage, defective restorations, and inadequate root fillings had a higher incidence of failures.

### Trauma

Trauma to teeth involves the pulp and the periodontal ligament. Both tissues can be affected either directly or indirectly.

### Resorptions

Root resorption is a state associated with either a physiologic or a pathologic process resulting in a loss of dentin, cementum, and/or bone. It may be external resorption or internal resorptions.<sup>[3]</sup>

# Exposure of dentinal tubules following root planing

Due to over instrumentation in procedures of scaling and root planing, dentinal tubules can be exposed and left unprotected to the environment. Subsequent microbial colonization of the exposed root dentin may result in bacterial invasions of the dentinal tubules. As a consequence, inflammatory lesions may develop in the pulp.

### Perforations

Root perforation leads to communications between the root canal system and either periradicular tissues or the oral cavity and may often reduce the prognosis of treatment.<sup>[11]</sup>

### **CLASSIFICATION OF PERIO-ENDO LESIONS**

Classification of perio-endo lesions by Simon, Glick, and Frank, in 1972 [Figure 3].<sup>[12]</sup>

A close by relationship exists between disease of the dental pulp and periodontal disease, and it states itself in



Figure 3: Diagrammatic representation of possible endo-perio problems based on the classification of Simon JH, Glick DH, and Frank JL 26, 27

various ways. The utmost used classification was given by Simon, Glick, and Frank, in 1972. Rendering to this classification, perio-endo lesions can be classified into:

- 1. Primary endodontic lesion
- 2. Primary periodontal lesion
- 3. Primary endodontic lesion with secondary periodontal involvement
- 4. Primary periodontal lesion with secondary endodontic involvement
- 5. True combined lesion.

# Primary Endodontic Lesion

An acute exacerbation of a chronic apical lesion on a tooth with a necrotic pulp may pass coronally through the periodontal ligament into the gingival sulcus. It is a sinus tract originating from pulp that opens into the periodontal ligament.

Diagnosis: To locate the origin of the lesion insert a gutta-percha cone into the sinus tract and to take one or more radiographs to conclude the origin.<sup>[13]</sup>

### **Primary Periodontal Lesion**

These lesions are caused primarily by periodontal pathogens. Chronic periodontitis progresses apically along the root surface and in the furcation area.<sup>[13]</sup>

- Diagnosis: Pulp tests indicate a clinically normal pulpal reaction.
- Clinical examination: Accumulation of plaque and calculus and the presence of deep pockets may be noticed.<sup>[5]</sup>

# Primary Endodontic Disease with Secondary Periodontal Involvement

Primary endodontic lesion when remains untreated, it becomes secondarily involved with periodontal

breakdown. Plaque accumulation at the gingival margin of the sinus tract leads to plaque-induced periodontitis in this area. Root fractures may also present as primary endodontic lesions with secondary periodontal involvement.

• Diagnosis: Patient response may occur without pain and involves the appearance of a pocket with bleeding on probing or exudation of pus.<sup>[5]</sup>

### Primary Periodontal Disease with Secondary Endodontic Involvement

Periodontal disease may have an effect on the pulp through dentinal tubules, lateral canals, or retrograde from the apex.<sup>[5]</sup>

# **True Combined Disease**

True combined endodontic-periodontal disease occurs less frequently, formed when an endodontic disease progressing coronally joins with an infected periodontal pocket progressing apically.<sup>[5]</sup>

#### ETIOPATHOGENESIS OF ENDO-PERIO LESIONS CAN BE UNDERSTOOD UNDER THE FOLLOWING CONTEXTS

- A. Effect of pulpal disease on the periodontium
- B. Effect of periodontal disease on the dental pulp a. Atrophic changes.
  - b. Inflammatory changes.
  - c. Resorption.
  - d. Toxic products.
  - e. Microorganisms.
  - f. Effect of periodontal treatment on the pulp
    - I. Scaling and root planning.
    - II. Acid etching.
    - III. Local medication.
- C. Etiological factors leading to pulp periodontal problems
  - 1. Live pathogens.
    - a. Bacteria.
    - b. Fungi (yeasts).
    - c. Viruses.
  - 2. Non-living etiologic agents.
    - a. Extrinsic agents.
    - I. Foreign bodies.
    - b. Intrinsic agents
      - II. Cholesterol.
      - III. Russell bodies.
    - IV. Rushton hyaline bodies.
    - V. Charcot–Leyden crystals.

# DIAGNOSIS OF PERIO-ENDO LESIONS

Practitioner has to gather all relevant information through history, examination, and appropriate diagnostic aids;

to synthesize and correlate this information; to determine the conditions which might be consistent with the findings; and to differentiate between them to arrive at the correct diagnosis.<sup>[9]</sup>

Second, clinical tests are important for obtaining correct diagnosis and differentiating between endodontic and periodontal disease. Therefore, extraoral and intraoral tissues are examined for the presence of any abnormality or disease. One test is usually not sufficient to obtain a definite diagnosis.<sup>[11]</sup>

Diagnostic procedures are as follows:

- 1. History
- 2. Visual examination
- 3. Crevicular examination
- 4. Palpation
- 5. Percussion
- 6. Mobility
- 7. Radiograph
- 8. Pulp vitality testing
  - a. Cold test
  - b. Electric test
- 9. Blood flow test
  - a. Laser Doppler flowmetry
  - b. Pulse oximetry
  - c. Magnetic resonance imaging
- 10. Cavity test
- 11. Restored teeth testing
- 12. Pocket probing
- 13. Fistula tracking
- 14. Cracked tooth testing
  - a. Transillumination test
  - b. Wedging
  - c. Staining
- 15. Selective anesthesia

# **Differential Diagnosis of Perio-endo Lesions**

A: Clinical finding in endodontic and periodontal lesions <sup>[14]</sup>			
Clinical finding	Perio lesions	Endo lesions	
Cause	Periodontal infection	Pulpal infection	
Vitality	Vital	Non vital	
Plaque and calculus	Present	Absent	
Caries	Absent	Present	
Pockets	Multiple, wide coronally	Single, narrow	
Mobility	Present	Absent	
Microbial	Complex	Few	
Ph	Alkaline	Acidic	
Trauma	Contributing factors	Primary or secondary	
Restorative	Deep or extensive	Not related	
Inflammation	Acute	Chronic	

International Journal of Medical and Oral Research, July-December 2018;3(2):11-17

B: Radiological features <sup>[14]</sup>			
Clinical findings	Perio lesions	Endo lesions	
Pattern	Generalized	Localized	
Bone loss	Wider coronal	Wider apically	
Vertical bone loss	Yes	No	
Periapical	Non-related	Radiolucent	

C: Histopathological features <sup>[15]</sup>			
Clinical finding	Perio lesion	Endo lesion	
Junctional epithelium	Apical migration present	Absent	
Granulation tissue	Coronary (large)	Apical (minimal)	
Gingival	Recession	Normal	

# TREATMENT OF PERIO-ENDO LESION

The most important element in the treatment of perioendo lesion is a correct diagnosis. This is attained by careful history taking, examination, and the use of special tests. The main features to consider are pulp vitality and type and extent of the periodontal defect.

### **Treatment of Primary Endodontic Lesions**

- Conventional endodontic therapy alone can resolve the lesions.
- Primary endodontic lesions have seen good prognosis.<sup>[15]</sup>

### **Treatment of Primary Periodontal Lesions**

- They are treated by Phase I (scaling and root planing) therapy.
- Poor restorations and developmental groove are removed.
- Periodontal surgery is performed after the completion of Phase I therapy.

Pulpal pathology may be induced while carrying out periodontal therapy, especially in lesion which involved the furcation area.<sup>[15]</sup>

# Treatment of Primary Endo Lesion with Secondary Periodontal Involvement

- These lesions not fully resolved only by endodontic treatment.
- Root/reroot canal treatment done immediately and cleaned shaped canal filled with calcium hydroxide paste.
- Treatment results should be evaluated in 2–3 months after it and only then should periodontal treatment be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition.

• Prognosis of primary endo lesion is good but worsens in the advanced stages of secondary periodontal involvement.<sup>[15]</sup>

# Treatment of Primary Periodontal Lesion with Secondary Endodontic Involvement

- Primary perio lesion with reversible pulpal hypersensitivity can be treated purely by periodontal therapy.
- Periodontal treatment removes noxious stimuli, and secondary dentinal tubules mineralization happens that resolve the hypersensitivity.
- Primary periodontic lesion with irreversible pulpitis, root/reroot canal treatment is carried out followed by periodontal treatment, and in some cases, surgical intervention is advantageous.
- The prognosis is poor in periodontal lesion than endo lesion and is depended on the apical extension of the lesion.<sup>[13]</sup>

#### **Treatment of True Combined Lesion**

True combined endodontic diseases require both endodontic and periodontal therapies. These lesions are treated initially as primary endo lesion with secondary periodontal involvement.

Root amputation, hemisection, or bicuspidization may allow the root configurations to be changed sufficiently for a part of the root structure to be saved.<sup>[15]</sup>

### **Treatment of latrogenic Lesions**

- Iatrogenic lesions are treated as primary endodontic lesion.
- First, priority is to close the iatrogenic communication - the aim is to produce a seal. Teeth with lesions caused by vertical root fractures have a hopeless prognosis and should be extraction.<sup>[15]</sup>

### DISCUSSION AND CONCLUSION

The periodontal-endodontic lesion is progresses on extension of either periodontal destruction apically combining with an existing periapical lesion or an endodontic lesion marginally combining with an existing periodontal lesion. The essential difference between the two disease entities is their particular source of infection. Therefore, treatment of combined lesions should intend to eliminate both the problems. Treatment is beginned with primarily endodontic and, it is important to understand that in perio-endo lesions, the endodontic treatment is the more predictable of the two entities. However, the success of endodontic therapy is dependent on the conclusion of periodontal therapy. Hence,

#### Perio-endo interrelationship

the complete hand in hand treatment of both aspects of perio-endo lesions is essential for effective long-term results.

#### REFERENCES

- 1. Simring M, Goldberg M. The pulpal pocket approach: Retrograde periodontitis. J Periodontol 1964;35:22-48.
- Mandel E, Machtou P, Torabinejad M. Clinical diagnosis and treatment of endodontic and periodontal lesions. Quintessence Int 1993;24:135-9.
- Anand PS, Nandakumar K. Management of periodontitis associated with endodontically involved teeth: A case series. J Contemp Dent Pract 2005;6:118-29.
- 4. Rotstein I, Simon JH. Diagnosis, prognosis and decision-making in the treatment of combined periodontal-endodontic lesions. Periodontol 2000 2004;34:165-203.
- Sunitha VR, Emmadi P, Namasivayam A, Thyegarajan R, Rajaraman V. The periodontal-endodontic continuum: A review. J Conserv Dent 2008;11:54-62.
- Solomon C, Chalfin H, Kellert M, Weseley P. The endodontic-periodontal lesion: A rational approach to treatment. J Am Dent Assoc 1995;126:473-9.

- Bhaskar SN, editor. Development and growth of teeth. In: Orban Oral Histology and Embryology. 11<sup>th</sup> ed. Missouri: Elsevier Pvt Ltd.; 2004. p. 28-48.
- 8. Singh S. Management of an endo perio lesion in a maxillary canine using platelet-rich plasma concentrate and an alloplastic bone substitute. J Indian Soc Periodontol 2009;13:97-100.
- 9. Hiatt WH. Pulpal periodontal disease. J Periodontol 1977;48:598-609.
- 10. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. J Clin Periodontol 2002;29:663-71.
- Meng HX. Periodontic-endodontic lesions. Ann Periodontol 1999;4:84-90.
- 12. Agrawal PK. Combined Periodontal-Endodontic Lesion. Jaipur: Department of Periodontia Govternment Dental College.
- 13. Simon JH, Glick DH, Frank AL. The relationship of endodontic-periodontic lesions. J Periodontol 1972;43:202-8.
- Wang HL, Glickman G. Endodontic and periodontic interrelationships. In: Cohen S, Burns R, editor. Pathways of the Pulp. 8<sup>th</sup> ed. New Delhi: Elsevier India Pvt Ltd.; 2004. p. 651-64.
- 15. Walker MR. The pathogenesis and treatment of endo-perio lesions. CPD Dent 2001;2:91-5.