

Endoperio Lesion: An Overview

Sumit Dash¹, Debkant Jena¹, Siba Jena², Swapna Kumari³, Narottam Praharaj³, Manali Nath⁴

¹Professor, ²Senior Lecturer, ³Tutor, ⁴Post Graduate Trainee, Department of Conservative Dentistry, Institute of Dental Sciences, Siksha 'O' Anusandhan (Deemed to be University), Bhubaneswar, Odisha, India

Abstract

The most common problems associated with tooth pain were endoperio lesions. It was very difficult to diagnose endoperio lesions by clinical or simply intra oral peri-apical radiograph. 50 % of tooth mortality rates are responsible for the endoperio lesion, the correct diagnosis was needed for appropriate treatment planning. This paper review the current diagnostic tools and treatment planning of endoperio lesion to achieve the best outcome.

Keywords: Endoperio lesion; current Diagnostic Tools; Treatment Planning.

Introduction

In daily dental practices, there are many patients we encounter with severe pain due to the endoperio lesion. As per definition these lesions, are localized in the particular area originate either from pulp or tissue of periodontium surrounding the involving tooth. Both of these issues have a common mesodermal origin. The first case was described by *Simmering and Goldberg*¹ in 1964. *Turner and Drew*² described the effect of periodontal and pulp tissue like fibrosis, calcification, and cystic degeneration. In the study done by *Seltzer et al*³, he found that 94% of extracted teeth which were periodontally involved had pulpal changes such as atrophy & inflammation with necrosis.

Endodontic & periodontium illness represents the greater part of the tooth mortalities¹. The different pathway through which microorganism communicate with periodontal tissue is

- Anatomic/Developmental origin:¹⁰
- One of the most common routes of infection is apical foramen which makes the communication in between pulp and Periodontal ligament.¹¹
- The second factor was anatomical deformities like developmental grooves. The gap between cemento-enamel junction can cause exposure of dentinal tubules.^{12,13}
- The other potent factor was accessory canals present in the apical area of tooth.^{14,15}
- Type of Furcation area may affect the degree of infection in the root 2% to 59 %.¹⁷⁻¹⁹

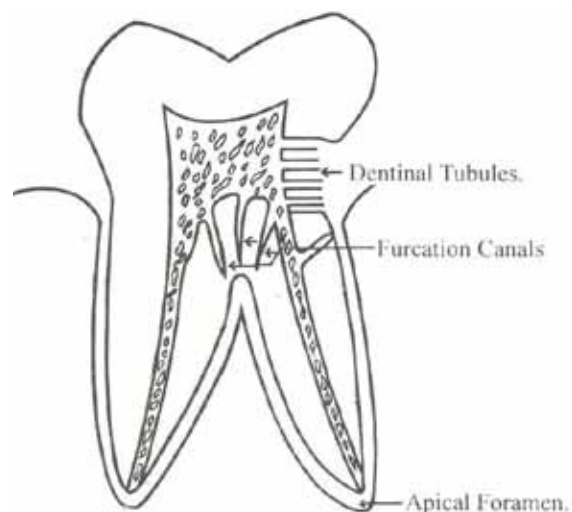


Figure 1. Anatomic relationship between pulp & periodontal ligament

Corresponding Author:

Dr. Sumit Dash

Professor, Department of Conservative Dentistry,
Institute of Dental Sciences, Siksha 'O' Anusandhan
(Deemed to be University), Bhubaneswar, Odisha,
India

e-mail: sumitdash@soa.ac.in

Pathological Origin: The factor pathologically derived and irritate the pulp due to various conditions like dental trauma, resorption, and loss of cementum due to irritants. The iatrogenic factors are those due to unintentional errors during the procedure by the dentist like root fracture, canal perforation during biomechanical preparation due to over instrumentation in root canal treatment. Root perforation opens the gateway between pulp and periodontal tissues which can lead to the poor

prognosis of the tooth. The second most important factor was vertical root fracture during the obturation compaction leads to fracture line in root leads to a fracture in the root which further leads communication between the pulp and periodontal lesion.

Etiology: There are two main categories of endoperio lesions are living and non-living pathogens are tabulated below.^{11, 23}

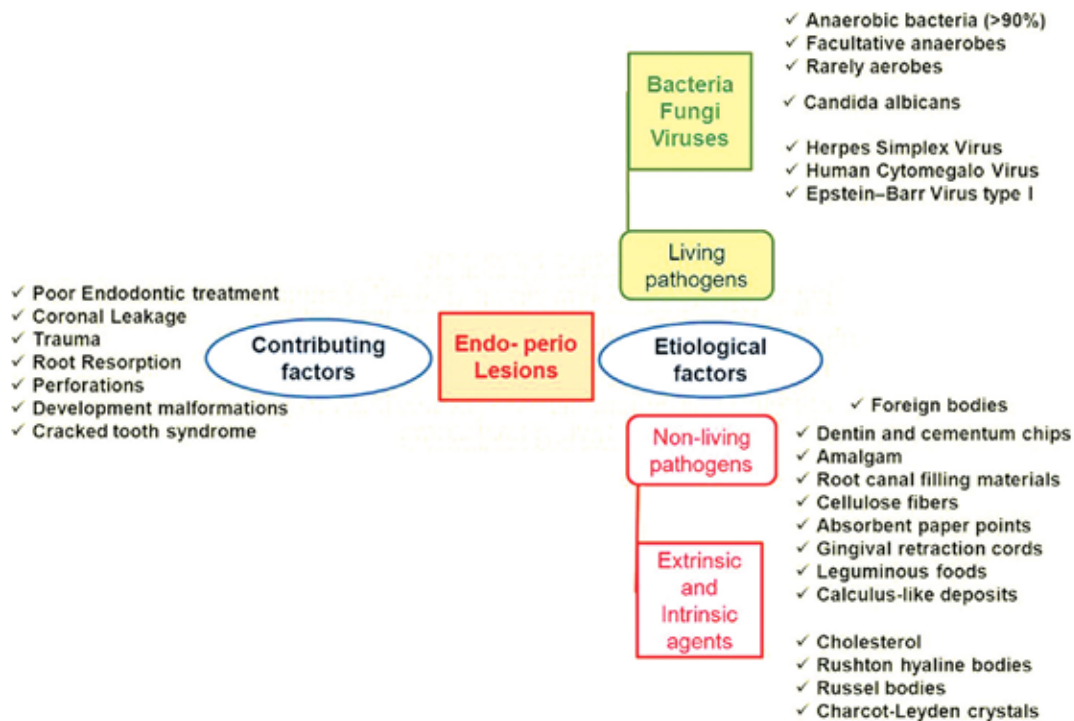


Figure 2. Main categories of endoperio lesions

The most common bacteria found in the endoperio lesions are Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Bacteroides forsythus, Eikenella corrodens, Fusobacterium nucleatum, Prevotella intermedia.²⁴ In many studies, we found fungal species Candida albicans are predominant in endodontic and periodontal lesions and virus found that EBV and herpes virus can also causative agents.^{27,28} It has been found that the causative agents like cytomegalovirus, Epstein Barr, the herpes virus can be responsible.^{27,28}

I. Classification:

- In 1972 Simon et al were the first to give

classification on endodontic periodontic lesion²⁹ based on diagnosis, prognosis and treatment.

- Primary endodontic lesion
- Primary periodontal lesion with secondary endodontic involvement
- Primary periodontal lesion
- Primary endodontic lesions with secondary periodontal involvement
- True combined lesion
- Guldener & Langeland classified according to pathologic relationship³⁰

- Periodontal–endodontic lesion
- Endodontic–periodontal lesion
- Combined lesions
- **In 1996, Torabinejad & Trope gave the classification based on a treatment plan**
- Periodontal origin
- Endodontic origin
- Combined endodontic and periodontic lesion
- Lesions with communication
- Separate endodontic and periodontal lesions
- Lesions with no communication
- **Grossman’s classification was based on therapy:**
- The condition of the tooth which requires just endodontic therapy
- The condition of the tooth which requires just periodontal therapy
- The condition of the tooth which requires both endodontic as well as periodontal therapy

Table 1. Classification on endodontic periodontic

<u>Endodontic Origin</u>	<u>Periodontic Origin</u>
1. Pulp non-vital.	1. Pulp vital except in advanced lesion.
2. Sharp throbbing pain	2. Dull, chronic pain.
3. Swelling in and extending beyond the	3. Swelling generally confined to attached mucosa
4. Tracing the fistulous tract leads to apical region or in the region or in the region of a lateral canal.	4. Tracing leads to mid root.
5. The fistulous tract is narrow and tortuous	5. Due to extensive loss of periodontal structures the fistula is wide in cervical area and can easily be probed.
6. Mobility in an acute stage involving multiple teeth limited to an isolated tooth.	6. Generalized mobility
7. Bone loss involving crestal and furcal bone limited to an	7. Generalised crestal bone loss either horizontal or verticle.

- Rateitschak et al gave a classification based on endodontic therapy
- Type I- In a condition where the pulp was dead and the disease by primarily endodontic origin
- Type II- In this condition the main cause of the disease by periodontal tissue which affects the pulp and sometimes caused pulpitis.
- Type III- In this condition pulp was dead and both origins were affected

II. Diagnosis:

The diagnosis can be made by clinical and radiography finding,

On clinical examination, swelling of the gingiva,

Pus discharge, pocket formation, sinus tract can be seen as associated teeth. On radiographic examination bone resorption can be seen there is wide radiolucency present in the apex of the tooth. It was very difficult to distinguish between the origin of the disease.

The clinical features of endoperio lesions are:

- **Primary endodontic lesion:** The large carious lesion, the presence of sinus in the periapical area, Presence of pain with mobility with a tender to percussion, periapical radiolucency can be seen with periodontal pocket and pain on biting pressure.
- **Primary periodontal origin:** Pain on palpation, grade I mobility, the vitality of pulp will positive, plaque and subgingival calculus, periodontal

abscess, vertical bone loss and periodontal pocket with sinus tract at the lateral aspect of the root which can be appreciated on a radiograph.

- **Primary endodontic with secondary periodontal lesion:** Exudates and marginal gingivitis, root perforations, fractures, ill-placed post, a dull ache, or shooting sharp pain in chronic conditions with localized mobility with deep periodontal pockets.
- **Primary periodontal/secondary endodontic lesions:** Gingival inflammation and plaque around multiple teeth, Generalized mobility, localized/generalized recession exudates, and pus. Pulp test may be positive in cases of the multi-rooted tooth and wide deep multiple periodontal pockets.
- **True combined lesion:** Chronic dull ache and swelling are present when calculus and periodontitis are around single/multiple teeth but in acute conditions, the pain will be more with generalized mobility with mobility of the involved tooth. Vitality tests will give negative responses except in the case of a multi-rooted tooth. At the edge of swelling the probe suddenly drops till apex of the tooth. This swelling is characterized as ‘blown out’.

Discussion

It is a challenge to treat endoperio lesions. In the study, Dahlen et al suggested that the vitality of tooth was an important factor for final diagnosis.³¹ Kerekes and Olsen found that the microorganisms are the same in both periodontal pocket and root canal treatment.³² Kurihara et al found that there is dissimilarity in microflora microbiologically and immunologically in pocket and root canal area.³³ Drucker et al³⁴ reported that Prevotella species associated with severe pain and commonly found in the root canal and periodontal pocket. Lin et al³⁵ demonstrated in the study there was no association of bacteria with the symptom. When we started with the treatment in combined endoperio lesion the endodontic treatment was done before periodontal therapy, the canal was completely clean with endodontic instruments with proper irrigation protocol which improve the reattachment procedure. The most common postoperative dentinal hypersensitivity. The time between to procedure is still not mentioning in any study it was still controversial. In various studies, the result found that endodontic treatment performed 2.5 months before periodontal surgery.^{32,35} Miranda et al. showed that there was no change in the clinical parameter endodontic performed before the periodontal surgery.³⁶

Ethical Permission: Not Required

Conflict of Interests: None

Funding: None

References

1. Simring M, Goldberg M. The pulpal pocket approach: Retrograde periodontitis. J Periodontol 1964;35:22-48.
2. Turner JH, Drew AH. (1919): Experimental injury into bacteriology of pyorrhea, Proc R Soc. Med(Odontol) 12:104
3. Seltzer S, Bender IB, Ziontz M. The interrelationship of pulp and periodontal disease. Oral Surg Oral Med Oral Pathol 1963;16:1474-90.
4. Sinai IH and Soltanoff W. The transmission of pathologic changes between the pulp and periodontal structures. Oral Surg. 1973;36:558.
5. Sharp RE. The relationship between the pulp and the periodontium. Periodont. Abstr. 1977;25: 130-142.
6. Bergenholtz G and Lindhe J. Effect of experimentally induced marginal periodontitis and periodontal scaling on the dental pulp. Clin Periodontol. 1978;5:59.
7. Hettler AB et al. Oral Surg. 1977; 44:939.
8. Czaruecki Rt and Schilder H. A histological evaluation of the human pulp in teeth with varying degrees of periodontal diseases. J.Endod. 1979;4:242.
9. Mjör IA, Nordahl I. The density and branching of dentinal tubules in human teeth. Arch Oral Biol 1996;41:401-12.
10. Zehnder M, Gold SI, Hasselgren G. Pathologic interactions in pulpal and periodontal tissues. J Clin Periodontol 2002;29:663-71.
11. Rotstein I, Simon JHS. Diagnosis, Prognosis and decision-making in the treatment of combined Periodontal-endodontic lesions. Periodontology 2000. 2004;34:165-203.
12. Simon JHS, Dorgan H, Ceresa LM, Silver GK. The radicular groove: its potential clinical significance. J Endod 2000;26:295-298.
13. Rotstein. I, James H.S. The endoperio lesion: a critical appraisal of the disease condition. Endodontic Topics, 2006;13:34-56.
14. Rubach WC, Mitchell DF. Periodontal disease,

- accessory canals and pulp pathosis, *J Periodontol.* 1965;36:34-38.
15. Lowman JV, Burke RS, Pelleu GB. Patent accessory canals: Incidence in molar furcation region. *Oral Surg Oral Med Oral Pathol.* 1973;36:580-584.
 16. De Deus QD. Frequency location and direction of lateral, secondary and accessory canals *J Endod* 1975;1:361-66.
 17. Kirkham DB. The location and incidence of accessory canals in periodontal pockets. *J Am Dent Assoc* 1975; 91:353-6
 18. .Shobha R et al, *Oral Surg.* 1974; 38:294.
 19. Vertucci, F.J., Williams, R.G. Furcation Canals in the Human Mandibular First Molar. *Oral Surg.* 1974;38:308–314.
 20. .Kerns DG, Glickman GN.(2006): Endodontic and periodontal interrelationships. In: Cohen S and Hargreaves KM, Eds. *Pathways of the pulp*, 9th Ed.St.Louis: Mosby Inc, 650-67.
 21. .Kvinnslund I, Oswald RJ, Halse A, Grønningstær AG. A clinical and roentgenological study of 55 cases of tooth perforation. *Int Endod J* 1989;22:75-84.
 22. Torabinejad M, Lemon RL. (1996): Procedural accidents. In Walton RE, Torabinejad M, editors. *Principles and practice of endodontics*, 2nd edn. Philadelphia:WB Saunders Co, : 306-323.
 23. Sunitha VR, Emmadi P, Namasivayam A, Thyegarajan R, Rajaraman V. The periodontal endodontic continuum: A review. *J Conserv Dent* 2008;11:54-62.
 24. Rupf S, Kannengiesser S, Merte K, Pfister W, Sigusch B, Eschrich K. Comparison of profiles of key periodontal pathogens in the periodontium and endodontium. *Endo Dent Traumatol.* 2000;16:269-275.
 25. Hannula J, Saarela M, Alaluusua S, Slots J, Asikainen S. Phenotypic and genotypic characterization of oral yeasts from Finland and the United States. *Oral Microbiol Immunol.* 1997;12:358-365
 26. Slots J, Rams TE, Listgarten MA. Yeasts, enteric rods and pseudomonas in the subgingival flora of severe adult periodontitis. *Oral Microbiol Immunol.* 1988;3:47-52.
 27. Sabeti M, Simon JH, Nowzari H, Slots J. Cytomegalo virus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns. *J. Endod.* 2003;29:321-323.
 28. Contreras A, Nowzari H, Slots J. Herpes viruses in periodontal pocket and gingival tissue specimens. *Oral Microbiol Immunol.* 2000;15:15-18.
 29. Simon JH, Glick DH, Frank AL. The relationship of endodontic-periodontic lesions. *J Periodontol* 1972;43:202-8.
 30. Guldener P.H.A. (1982): Beziehung zwischen Pulpa-und Parodontaler krankungen. In Guldener, P.H.A., Langeland, K.: *Endodontologie*. Thieme, Stuttgart. 368-378.
 31. Dählen G. Microbiology and treatment of dental abscesses and periodontal-endodontic lesions. *Periodontol* 2000. 2002;28:206-39.
 32. Kerekes K, Olsen I. Similarities in the microflora of root canals and deep periodontal pockets. *Endod Dent Traumatol.* 1990;6:1-5.
 33. Kurihara H, Kobayashi Y, Francisco IA, Isoshima O, Nagai A, Murayama Y. A microbiological and immunological study of endodontic-periodontic lesions. *J Endod.* 1995;21(12):617-21.
 34. Drucker DB, Gomes BPF, Lilley JD. Role of anaerobic species in endodontic infection. *Clin Infect Dis.* 1997;25(Suppl 2):220-1.
 35. Lin S, Sela G, Sprecher H. Periopathogenic bacteria in persistent periapical lesions: an in vivo prospective study. *J Periodontol.* 2007;78(5):905-8.
 36. de Miranda JL, Santana CM, Santana RB. Influence of endodontic treatment in the post-surgical healing of human Class II furcation defects. *J Periodontol.* 2013;84:51–7.