Int.J.Curr.Res.Aca.Rev.2017; 5(3): 25-30

International Journal of Current Research and Academic Review

ISSN: 2347-3215 (Online) Volume 5 Number 3 (March-2017)

Journal homepage: http://www.ijcrar.com

doi: https://doi.org/10.20546/ijcrar.2017.503.004

Endo-Perio Symbiosis

Ishita Joshi[1*], Rachita Jain[2], Siddharth Tevatia[3] and Prateek Sharma[4]

I.T.S Dental College, Ghaziabad, India

Corresponding author

Abstract

The interrelationship between endodontic and periodontal diseases has been a subject of speculation, confusion and controversy for many years. Pulpal and periodontal problems are responsible for more than 50% of tooth mortality today. An endo-perio lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. These lesions often present challenges to the clinician as far as diagnosis and prognosis of the involved teeth are concerned. It is very essential to make a correct diagnosis so that the appropriate treatment can be provided. To make a correct diagnosis the clinician should have a thorough understanding and scientific knowledge of these lesions and may need to perform restorative, endodontic or periodontal therapy, either singly or in combination to treat them. Therefore, this paper will highlight the diagnostic, clinical guidelines and decision making in the treatment of these lesions from an endodontists point of view to achieve the best outcome.

Article Info

Accepted: 28 February 2017
Available Online: 20 March 2017

Keywords

Apical foramen,
Periodontal disease
and diagnosis.

Introduction

As clinicians we come across the term Endo-periodontal lesions. *Endo-perio lesions* are localized, circumscribed areas of bacterial infection that originates from either dental pulp or periodontal tissues surrounding the involved tooth/teeth or both. Dental pulp and periodontium both the tissues are interrelated from embryonic stage itself, as we know that precursors of both the tissues (dental papilla and dental follicle) have common mesodermal origin. In 1964, Simring and Goldberg (Simring *et al*., 1964) were the first one to describe the relationship between both the tissues.

Turner and Drew (Turner *et al*., 1919) in 1919 were the first one to describe the effect of diseases of periodontium on pulpal tissue. They validated that pulpal changes like fibrosis, calcification and cystic degeneration are induced by pyorrhoea (suppurative periodontitis). Reinforcing the opinion, Seltzer *et al*., (1963) reported that 94% 0f 85 periodontally involved extracted teeth exhibited pulpal changes such as inflammation, atrophy and even complete necrosis.

Sinnai and Soltanof (1973) along with Sharp (1977) also supported this opinion. Though few others reported that periodontal diseases either minimally or do not affect the pulpal tissue at all. They supported their opinion with a study conducted on white rats (Bergenholtz *et al*., 1978; Hettler *et al*., 1977).

Endodontic along with periodontal disease accounts for more than half of the tooth mortality. The various pathways through which microorganisms communicate between pulp and periodontal tissue are enumerated as (Mjör *et al*., 1996; Zehnder *et al*., 2002)

Anatomic/Developmental origin: These include apical foramen, auxillary /furcation canals and dentinal tubules.

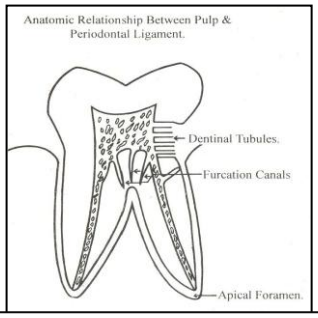
Apical foramen is believed to be the main route of communication. The microorganisms, infection transmits

from pulp to periodontal tissue and vice-versa through foramen (Rotstein *et al*., 2004).

The S- shaped microscopic channels extending from outer dentin surface to pulp is known as dentinal tubules. Periodontal disease and various procedures of periodontal therapy, developmental grooves, gap joint at cemento- enamel junction can lead to exposure of dentinal tubules (Simon *et al*., 2000).

Our teeth have huge number of accessory/auxillary canals which act as potent pathway for spread of infection. *De dues* conducted a study on 1,140 teeth and reported that 27.4% of the teeth have auxillary canals (De Deus *et al*., 1975).

Furcation area also has accessory canals which can range from 2% to 59 % (Kirkham, 1975; Shobha *et al*., 1974).



Pathological origin: These include root fracture following trauma, idiopathic root resorption, loss of cementum due to irritants.

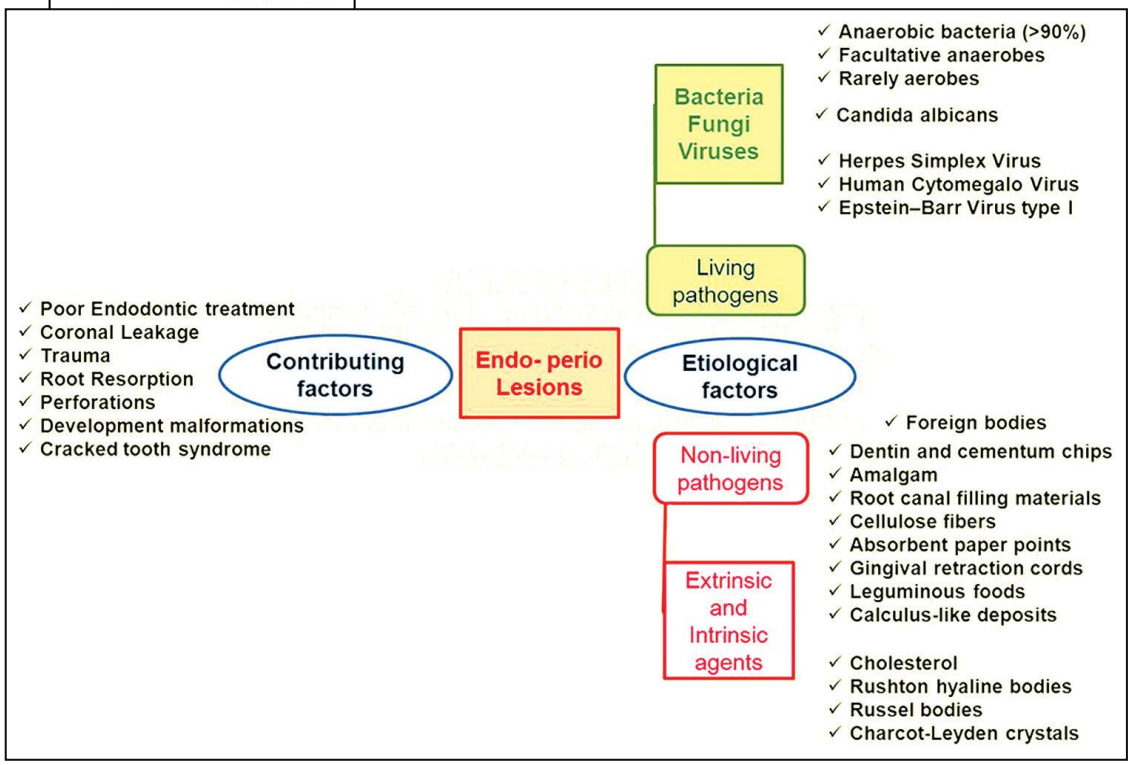
Iatrogenic origin: these include root fractures, root canal perforation.

Post preparation, over instrumentation can lead to root perforation which opens a pathway between pulp and periodontal tissue and can lead to poor prognosis of the tooth (Kerns *et al*., 2006; Kvinnsland *et al*., 1989).

Vertical root fracture is characterized by an incomplete or complete fracture line that extends through the long axis of the root toward the apex.

Etiology

The cause of endo-perio lesions can be divided into two categories, living and non living pathogens along with various contributing factors concisely tabulated below.



Microbiology of endo-perio lesions includes bacterial species like *Actinobacillus Actinomycetem comitans*, *Bacteroides forsythus*, *Ekinella corrodens*, *Fusobacterium nucleatum*, *Porphyrominas gingivalis*, *Prevotella intermedia* (Rupf *et al*., 2000). Also fungal species like *Candida albicans* (Hannula *et al*., 1997) are predominant in endodontic and periodontal lesions. Recently it has been found that Cytomegalo virus, Ebstein - barr virus, Herpes virus can also be the causative agents (Contreras *et al*., 2000).

Classification

In 1972, Simon *et al*., were the first one to give classification on endo- perio lesionn based on diagnosis, prognosis and treatment

- Primary endodontic lesion
-Primary periodontal lesion
-Primary endodontic lesions with secondary periodontal involvement
-Primary periodontal lesion with secondary endodontic involvement
-True combined lesion

Guldener and Langeland gave a classification based on pathologic relationship (Guldener, 1982)

-Endodontic –periodontal lesion
-Periodontal –endodontic lesion
-Combined lesions

In 1996, Torabinejad and Trope came with new classification from the treatment point of view
-Endodontic origin
-Periodontal origin
-Combined endo-perio lesions
-Separate endodontic and periodontal lesions
-Lesions with communication
-Lesions with no communication

Grossman's classification based on therapy is:
-Teeth that require only endodontic therapy
-Teeth that require only periodontal therapy
-Teeth that require endodontic as well as periodontal therapy

Rateitschak *et al*., gave a classification based on endodontic therapy
-Type I- primarily of endodontic origin and pulp is usually dead
-Type II-primarily periodontal disease which may affect pulp, and pulp is normal or sometimes damaged by ascending pulpitis
-Type III-combined case of root canal and periodontal disease and pulp is usually dead.

Diagnosis

The correct diagnosis can be made on the basis of patient history, hard and soft tissue examination, pulp vitality test, determining periodontal pocket depth.

Endodontic and periodontal lesions commonly present with features like

-swelling of gingiva
-pus dischar
-pocket formation
-fistulous tract formation
-tenderness to percussion (horizontal and vertical)
-tooth mobility

} Clinical features

-bone resorption
-periapical resorption

} Radiographic features

However it is difficult to distinguish between both the lesions, still there are few clinical features to help us in reaching a diagnosis as tabulated below:

The detailed description of diagnostic features for various types of endo-perio lesions is

Endodontic Origin	Periodontic Origin
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 he 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.

Primary endodontic lesion

1. There is definite presence of large carious lesion/huge restoration/erosion/crack/poor endotreated tooth.
2. There is chance of presence of sinus tract opening.
3. Patient will experience sharp pain and there will be high mobility.
4. Tooth will be tender to percussion
5. Pulp vitality test will give negative or delayed response
6. Deep narrow solitary pocket
7. X-ray(IOPA) with gutta percha points to apex or furcation area in molars
8. Periapical radiolucency will be evident in radiograph
9. Pain on chewing, especially on release of biting pressure.

Primary periodontal origin

1. Gingival inflammation/recession around multiple tooth, plaque and sub gingival calculus, periodontal abscess.
2. Pain on palpation
3. Localized to generalized mobility
4. Pulp vitality test is positive
5. Deep and wide multiple pockets
6. Sinus tract at lateral aspect of root
7. Generalized vertical bone loss

Primary endodontic and secondary periodontal lesion

1. Marginal gingivitis and exudates
2. Root perforation/fracture/misplaced post
3. Sharp shooting pain usually, dull ache in chronic conditions
4. Tender on percussion
5. Localized mobility
6. Pulp vitality test gives no response
7. Sinus tract at the apex or furcation area
8. Wide solitary pocket
9. Presence of large carious lesion/huge restoration/erosion/crack/poor endotreated tooth with wide periapical radiolucency
10. Pain on chewing, especially on release of biting pressure

Primary periodontal and secondary endodontic lesion

1. Plaque, calculus and gingival inflammation around multiple teeth, localized/generalized recession exudates and pus
2. Tender on percussion
3. Generalized mobility
4. Vitality test may be positive in case of multi rooted tooth
5. Wide deep multiple periodontal pockets
6. Sinus tract at lateral aspect of root
7. Angular bone loss

True combined lesion

1. Plaque, calculus and periodontitis, swelling around single/multiple teeth
2. Dull ache but in acute conditions pain will be severe
3. Tender on percussion
4. Generalized mobility with high mobility of the involved tooth
5. Vitality test will give negative response. except in case of multi rooted tooth
6. At edge of swelling, probe suddenly drops till apex of the tooth .This swelling is characterized as "blown – out"

Results and Discussion

Endo perio lesions pose a challenge in our clinical practice. Dahlen *et al*., (2008) studied the microbiology of the lesion and they reported that diagnosis depends on vitality of the tooth. Kerekes and Olsen reported similarity in micro biota of root canal and periodontal pocket. Zehnder *et al*., study also affirmed this report.

Kurihara *et al*., (1995) analyzed endo- perio lesions microbiologically and immunologically and reported dissimilarity in micro flora of periodontal pocket and root canal. Also it was reported by Drucker *et al*., (1997) that *Prevotella* species found in root canal and periodontal pocket have association with pain and Bacteroides, *Fusobacterium* with pus exudates. Notwithstanding, Lin *et al*., (2007) demonstrated that combination of group of bacteria have no association with symptoms.

The management of endo-perio lesion requires both endodontic and periodontal treatment but it has to be done sequentially and it has to be taken care that cross infection does not happen. If both the treatment are indicated then endodontic treatment has to be done before periodontal therapy, as toxic material removal from canal will lead to improved soft tissue re-attachment and post periodontal therapy sensitivity and patient discomfort is also reduced.

The ideal interval between the endodontic treatment and periodontal surgery has also been challenged by controversial findings. It was reported that root canal treatment performed 2.5 months before periodontal surgery not to impair periodontal healing. Miranda *et al*., (2013) suggest that endodontic treatment performed 6 months before the surgical debridement of the furcation of mandibular molars did not impair the clinical parameters of periodontal healing (Perlmutter *et al*., 1987).

There has been lot of studies, case reports and publications done on endo- perio lesions and this review article was an attempt to give an insight of this topic.

References

Bergenholtz, G. and Lindhe, J. 1978. Effect of experimentally induced marginal periodontitis and periodontal scaling on the the dental pulp. *Clin Periodontol.*, 5: 59.

Contreras, A., Nowzari, H., Slots, J. 2000. Herpes viruses in periodontal pocket and gingival tissue specimens. *Oral Microbiol. Immunol.*, 15: 15-18.

Czaruecki, R. and Schilder, H. 1979. A histological evaluation of the human pulp in teeth with varying degrees of periodontal diseases. *J. Endod.*, 4: 242.

Dáhlen, G. 2000. Microbiology and treatment of dental abscesses and periodontal-endodontic lesions. *Periodontol.*, 28: 206-39.

De Deus, Q.D. 1975. Frequency location and direction of lateral, secondary and accessory canals, *J. Endod.*, 1: 361-66.

de Miranda, J.L., Santana, C.M., Santana, R.B. 2013. Influence of endodontic treatment in the post-surgical healing of human Class II furcation defects. *J. Periodontol.*, 84: 51–7.

Drucker, D.B., Gomes, B.P.F.A., Lilley, J.D. 1997. Role of anaerobic species in endodontic infection. *Clin. Infect. Dis.*, 25(Suppl 2): 220-1.

Guldener, P.H.A. 1982. Beziehung zwischen Pulpa-und Parodontaler krankungen. In Guldener, P.H.A., Langeland, K., Endodontologie, Thieme, Stuttgart. 368-378.

Hannula, J., Saarela, M., Alaluusua, S., Slots, J., Asikaainen, S. 1997. Phenotypic and genotypic characterization of oral yeasts from Finland and the United States. *Oral Microbiol. Immunol.*, 12: 358-365.

Hettler, A.B. *et al*. 1977. *Oral Surg*, 44: 939.

Kerekes, K., Olsen, I. 1990. Similarities in the microfloras of root canals and deep periodontal pockets. *Endod. Dent. Traumatol.*, 6: 1-5.

Kerns, D.G., Glickman, G.N. 2006. Endodontic and periodontal interrelationships. In: Cohen S and Hargreaves KM, Eds. Pathways of the pulp, 9th Ed. St. Louis: Mosby Inc, 650-67.

Kirkham, D.B. The location and incidence of accessory canals in periodontal pockets. *J. Am. Dent. Assoc*., 91: 353-6.

Kurihara, H., Kobayashi, Y., Francisco, I.A., Isoshima, O., Nagai, A., Murayama, Y. 1995. A microbiological and immunological study of endodontic-periodontic lesions. *J. Endod.*, 21(12): 617-21.

Kvinnsland, I., Oswald, R.J., Halse, A., GrØnningsæter, A.G. 1989. A clinical and roentgenological study of 55 cases of tooth perforation. *Int. Endod. J.*, 22: 75-84.

Lin, S., Sela, G., Sprecher, H. 2007. Periopathogenic bacteria in persistent periapical lesions: an in vivo prospective study. *J. Periodontol.*, 78(5): 905-8.

Lowman, J.V., Burke, R.S., Pelleu, G.B. 1973. Patent accessory canals: Incidence in molar furcation region. *Oral Surg. Oral Med. Oral Pathol.*, 36: 580-584.

Mjör, I.A., Nordahl, I. 1966. The density and branching of dentinal tubules in human teeth. *Arch. Oral Biol.*, 41: 401-12.

Perlmutter, S., Tagger, M., Tagger, E., Abram, M. 1987. Effect of the endodontic status of the tooth on experimental periodontal reattachment in baboons: a preliminary investigation. *Oral Surg. Oral Med. Oral Pathol.*, 63: 232–6.

Rotstein, I., James, H.S. 2006. The endo-perio lesion: a critical appraisal of the disease condition. *Endodontic Topics*, 13: 34-56.

Rotstein, I., Simon, J.H.S. 2004. Diagnosis, Prognosis and decision-making in the treatment of combined Periodontal-endodontic lesions. *Periodontol.*, 34: 165-203.

Rubach, W.C., Mitchell, D.F. 1965. Periodontal disease, accessory canals and pulp pathosis, *J. Periodontol*., 36: 34-38.

Rupf, S., Kannengiesser, S., Merte, K., Pfister, W., Sigusch, B., Eschrich, K. 2000. Comparison of profiles of key periodontal pathogens in the periodontium and endodontium. *Endo. Dent. Traumatol.*, 16: 269-275.

Sabeti, M., Simon, J.H., Nowzari, H., Slots, J. 2003. Cytomegalo virus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns. *J. Endod.*, 29: 321-323.

Seltzer, S., Bender, I.B., Ziontz, M. 1963. The interrelationship of pulp and periodontal disease. *Oral Surg. Oral Med. Oral Pathol.*, 16: 1474-90.

Sharp, R.E. 1977. The relationship of the pulp and the periodontium. *Periodont. Abstr.*, 25: 130-142.

Shobha, R., *et al*. 1974. *Oral Surg.*, 38: 294.

Simon, J.H., Glick, D.H., Frank, A.L. The relationship of endodontic- periodontic lesions. *J. Periodontol.*, 43: 202-8.

Simon, J.H.S., Dorgan, H., Ceresa, L.M., Silver, G.K. 2000. The radicular groove: its potential clinical significance. *J. Endod.*, 26: 295-298.

Simring, M., Goldberg, M. 1964. The pulpal pocket approach: Retrograde periodontitis. *J. Periodontol.*, 35: 22-48.

Sinai, I.H. and Soltanoff, W. The transmission of pathologic changes between the pulp and periodontal structures. *Oral Surg.*, 36: 558.

Slots, J., Rams, T.E., Listgarten, M.A. 1988. Yeasts, enteric rods and pseudomonas in the subgingival flora of severe adult periodontitis. *Oral Microbiol Immunol.*, 3: 47-52.

Sunitha, V.R., Emmad, P., Namasivayam, A., Thyegarajan, R., Rajaraman, V. 2008. The periodontal endodontic continuum: A review. *J. Conserv. Dent.*, 11: 54-62.

Torabinejad, M., Lemon, R.L. 1996. Procedural accidents. In Walton RE, Torabinejad M, editors. Principles and practice of endodontics, 2nd edn. Philadelphia: WB Saunders Co,: 306-323.

Turner, J.H., Drew, A.H. 1919. Experimental injury into bacteriology of pyorrhea, *Proc. R Soc. Med. (Odontol).*, 12: 104.

Vertucci, F.J., Williams, R.G. Furcation Canals in the Human Mandibular First Molar. *Oral Surg.*, 38: 308–314.

Zehnder, M., Gold, S.I., Hasselgren, G. 2002. Pathologic interactions in pulpal and periodontal tissues. *J. Clin. Periodontol.*, 29: 663-71

.

How to cite this article:

Ishita Joshi, Rachita Jain, Siddharth Tevatia and Prateek Sharma. 2017. Endo-Perio Symbiosis. *Int.J.Curr.Res.Aca.Rev*. 5(3), 25-30. **doi:** https://doi.org/10.20546/ijcrar.2017.503.004