

Endo - Perio Lesions: A Diagnostic Dilemma

Abstract

Endo-perio lesions primarily occur by way of the intimate anatomic and vascular connections between the pulp and the periodontium. Diagnosis is often challenging because these diseases have been primarily studied as separate entities and may mimic clinical characteristics of each other. When a primarily endodontic lesion changes into a secondary periodontal lesion the resultant clinical features become even more bizarre. This may delay the diagnosis and hence the correct treatment.

Key Words

Endo-Perio lesions

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INTRODUCTION

The periodontium and the pulp are closely related, they have embryonic, anatomic and functional interrelationships. In 1919 Turner and Drew first described the effect of periodontal disease on the pulp. The relationship between periodontal and pulpal disease was first described by Simring and Goldberg in 1964. The intimate anatomic and vascular connection between the pulp and the periodontium is studied in great detail by the periodontists and the endodontists.^[1] These lesions initially present themselves as just an endodontic lesion and later slowly start showing signs for periodontal involvement. This type of lesion is called by Simon as Class III type of endo-perio lesion.^[2] Periodontal involvement is only for establishing an easy route for drainage of endodontic pathology. There are many cases documented about established secondary periodontal and primary endodontic lesions but few of them document the events that took place during the conversion, when diagnosis is most confusing. Here is a case that presented itself initially as an

endodontic emergency and the bizarre clinical features delayed the correct treatment for several weeks. Its conversion into a secondary periodontic lesion helped in the final diagnosis and correct treatment.

Pulpal-Periodontal Communications

The dental pulp communicates with the periodontal ligament through the various routes that are described as follows.

a. Dentinal tubules: As many as 15,000 dentinal tubules per square millimeter are present on the root surface at the cervical area. Periodontal disease, scaling, root planning, surgical procedures, developmental groove, gap joint at the cemento-enamel junction may lead to exposed dentine. The pulp chamber can thus communicate with the external root surface in case of denuded cementum through this dentinal tubule. Cervical dentinal hypersensitivity can be an example of this phenomenon.

b. Lateral canals and accessory canals: Lateral and accessory canals can be present anywhere along the root, but are mainly seen in the apical third of the

root. De Deus studied 1,140 teeth for accessory canal and found 27.4% exhibited accessory canals. Gutmann evaluated 102 teeth for the presence of accessory canals and found 25.5% of the studied sample demonstrated accessory can in furcation area alone (furcal canals). A large number of accessory canals exist in human teeth, the presence of patent accessory canals is a potential pathway for the spread of bacteria and toxic substances resulting in a direct inflammatory process in the periodontal ligament.

c. Apical foramen: Apical foramen is the prime and most precise route of communication between the pulp and the periodontium. The bacteria, bacterial toxins their other by products and inflammatory mediators can exit easily through the apical foramen causing periapical pathosis and in case of deep periodontal pockets the vice versa may occur.

d. Palatogingival groove: It is a developmental groove, a common anomaly in maxillary central incisors. It begins in the central fossa or across the cingulum, extends varying distances apically. It is located in the midpalatal or mesial or distal regions of the tooth palatally or even buccally. It provide funnel like areas for plaque retention. Periodontal probing is advised for patients with palatogingival grooves. Palatogingival grooves are associated with deep isolated-tubular-shaped periodontal pockets with intrabony defects. They may bleed and suppurate. On radiograph they appear as a-tear drop shaped areal and dark lines parallel or imposed on the root canal can be noticed. These lines are termed as-parapulpal lines (dark vertical line). They are related to the incidence of localized periodontitis with or without pulpal pathosis, depending on the depth, extent and complexity of the groove.

e. Root perforations: Perforations of root are undesirable clinical complications that open up a communication between the root canal system and the periodontal ligament/oral cavity and may lead to treatment failure. They occur due to extensive carious lesion, internal and external resorption, over instrumentation (iatrogenic) and post-preparation (iatrogenic).

f. Vertical root fractures: A vertical root fracture is defined as a fracture of the root that is longitudinally oriented at a more or less oblique angle towards the long axis of the tooth. Root fractures occur accidently and may involve cementum, dentine and

pulp. Mobility of the involved teeth, Pain on biting, pain on selective loading of the cusps, discomfort,

periodontal defect, radiographic bone destruction, abscess formation and a narrow sinus tract-type of probing distance may at instances be present on one side in the line of fracture are all seen.

Pulpal diseases and the Periodontium

Pulpal pathosis involves inflammatory changes. With necrosis of the pulp there is inflammatory response by the periodontal ligament at the apical foramen and at the opening of the accessory canals 25. Necrosis of the pulp, can result in rapid and wide spread destruction of periodontium, the production of radiolucency at the apex of the tooth, in the furcation or at various points along the root. It has been shown that periodontal treatment of teeth with pulpal necrosis and periapical radiolucency resulted in retarded or impaired periodontal healing 26. Retrogradeperiodontitis caused by pulpal disease is a common causeof severe, localised destruction of periodontal tissues. Itssigns and symptoms include periodontal pocket formation, purulent inflammatory exudates, angular bone loss, swelling and bleeding of the gingival tissues and increased tooth mobility

Periodontal diseases and the Pulp

Controversies and conflicts surround the studies done to examine the effects of periodontal inflammation on the pulp. Greater incidence of pulpal inflammation and Degeneration has been reported in periodontally involved teeth than in teeth with no periodontal disease. It has been suggested that periodontal disease has no effect on the pulp till it involves the apex or the periodontal breakdown has been exposed an accessory canal to the oral environment Hence theoretically a deleterious effect of periodontal disease on the pulp can occur and produce pulpitis and is often referred as retrograde pulpitis.

ETIOLOGY

Microbiology

Bacteria: *Actinobacillus actinomycetemcomitans*, *bacteroides frosythus*, *Ekinella corrodens*, *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Prevotella intermedia* and *Treponema denticola* are present in both endodontic sample as well as in teeth with chronic apical periodontitis and chronic adult periodontitis.

Fungi: Various fungal species especially *Candida albicans* are prevalent both in endodontic infections as well as in subgingivally in many cases of adult periodontitis.

Viruses: Recent data suggests that a number of common types of viruses such as *Cytomegalo virus*,

Epstein-Barrvirus, herpes virus may be involved in pathogenesis of periodontal and endodontic disease ranging from an increase in periodontal pathogens in periodontal pockets to involvement in pulpal and periapical pathologies

CLASSIFICATION OF ENDO-PERIO LESIONS

1. BASED ON POSSIBLE PATHOLOGIC RELATIONSHIPS (Guldener and Langeland):

a. Endodontic - periodontal lesions: In endodontic-periodontal lesions, pulpal necrosis precedes periodontal changes. A periapical lesion originating in pulpal infection and necrosis may drain to the oral cavity through the periodontal ligament and adjacent alveolar bone. This may present clinically as a localised, deep, periodontal pocket extending to the apex of the tooth. Pulpal infection also may drain through accessory canals, especially in the area of the furcation & may lead to furcal involvement through loss of clinical attachment and alveolar bone.

b. Periodontal - endodontic lesions: In periodontal-endodontic lesions, bacterial infection from a periodontal pocket associated with loss of attachment & root exposure may spread through accessory canals to the pulp, resulting in pulpal necrosis. In the case of advanced periodontal disease, the infection may reach the pulp through the apical foramen. Scaling & root planning removes cementum & underlying dentin and may lead to chronic pulpitis through bacterial penetration of dentinal tubules.

c. Combined lesions: Combined lesions occur when pulpal necrosis and a periapical lesion occur on a tooth that also is periodontally involved. Combined lesions could display interesting correlations between specific microbiota of endodontic lesion and periodontal pockets.

2. BASED ON ETIOLOGY, DIAGNOSIS, PROGNOSIS AND TREATMENT {Simon's Classification }:

1. Primary endodontic lesion: Necrotic pulp draining coronally through the periodontal ligament into the gingival sulcus with acute exacerbation of chronic apical lesion. The necrotic pulp may drain through the apical foramen, lateral canal or through the accessory canals at the furcal area. The pocket that forms is narrow and has little or no local factors. Radiographs with gutta-percha cone tracing the sinus tract point towards the origin of the lesion. Root canal treatment is the treatment of choice.

Prognosis is excellent with complete and rapid resolution of the lesion occurs.

2. Primary periodontal lesion: It is chronic periodontitis progressing apically along the root surface. It is characterized with wide periodontal pocket along with presence of local factors, a vital pulp, minimal or no pain, periodontal pockets in multiple teeth. Periodontal therapy is the available treatment option. Root canal therapy must not be carried as the pulp is vital. Only Periodontal therapy should be carried out. Prognosis is related to the amount of attachment loss, the effectiveness of the periodontal treatment accomplished and the patient response.

3. Primary endodontic with secondary periodontal involvement: Primary endodontic lesion with a draining abscess through the periodontium if left untreated over a period of time may lead to local factors accumulating in the sinus tract and a creation of secondary periodontal problem. It may also be caused due to root fractures, iatrogenic perforations by improper placement of pins and post. Evidence of both pulpal and periodontal disease can be seen in radiographs. Root canal therapy is carried out and certain time is allowed for periodontal tissues to heal. After this evaluation period of 2-3 months periodontal therapy is carried out if required. Prognosis depends on the amount of attachment loss and severity of periodontal disease.

4. Primary periodontal with secondary endodontic involvement: Retrograde pulpitis can occur when the periodontal disease exposes lateral canal to oral environment or involves the apical canal. In such a case primary periodontal lesion with secondary endodontic involvement can be observed. Patients reports with severe pain, signs of pulpal disease concomitant with deep pocketing and history of extensive periodontal disease. Microbiota of the root canal shows strong correlation with that of periodontal pockets. Radiographically these lesions are similar to primary endodontic lesions with secondary periodontal involvement. Both periodontal and endodontic therapies are required. Prognosis depends on the severity of the periodontal disease and periodontal response to treatment.

5. True combined lesions: Pulpal pathosis progressing coronally and periodontal pathoses progressing apically may develop independently around the same tooth and concomitantly unite. They are relatively infrequent and when occur may have significant periodontal involvement with considerable attachment loss. Clinically necrotic

pulp or failing endodontic treatment with presence of local factors (plaque and calculus), deep pockets and periodontitis are present in varying degrees. Radiographically these lesions appear similar to that of the tooth with vertical fractures. Immediate sealing of root perforations, root canal therapy, advanced endodontic surgery, periodontal therapy with procedures such as hemisection, root resection may be required treatment options. Prognosis is guarded and depends on the amount of destruction caused by periodontal disease

6. Concomitant pulpal and periodontal lesions: This additional group of lesions was proposed by Belk and Guntmann. Pulpal and periodontal diseases can co-exist with different aetiologies. Thus the lesions will consist of an endodontic lesion and a non communicating periodontal lesion. In this situation both the diseases should be treated individually.

3. **BASED ON THERAPY** {Grossman's Classification}:

- a. Teeth that require endodontic therapy alone.
- b. Teeth that require periodontal therapy alone
- c. Teeth that require endodontic as well as periodontal therapy

4. **BASED ON ENDODONTIC THERAPY** {Rateitschak *et al*):

- a. Type I: It is primarily of endodontic origin and the pulp is usually dead.
- b. Type II: It is basically periodontal disease periodontal disease, which sometimes affects the pulp, and the pulp is usually normal or sometimes damaged by ascending pulpitis.
- c. Type III: It is a combined case of a root canal problem and periodontal disease, and the pulp is usually dead.

Investigations

Proper history taking, thorough examination, radiographic evaluation, pulp vitality testing, pocket probing, fistula tracking essential aids in proper diagnosis. When you are unable to establish a diagnosis, consider the lesion to be of pulpal origin, as endodontic treatment may correct both lesions.

CONCLUSION

Endo-perio lesions primarily occur by way of the intimate anatomic and vascular connections between the pulp and the periodontium. Diagnosis is often challenging because these diseases have been primarily studied as separate entities and may mimic clinical characteristics of each other. A patient study of behavior of the lesion and a thorough differential diagnosis can help us reach the correct diagnosis.

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