Review Article

Endo-Perio Dilemma: A Brief Review

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ABSTRACT

The actual relationship between periodontal and pulpal disease was first described by Simring and Goldberg in 1964. Since then, the term "perio-endo" lesion has been used to describe lesions due to inflammatory products found in varying degrees in both the periodontium and the pulpal tissues. The pulp and periodontium have embryonic, anatomic and functional inter-relationships. The simultaneous existence of pulpal problems and inflammatory periodontal disease can complicate diagnosis and treatment planning. A perio-endo lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. Knowledge of these disease processes is essential in coming to the correct diagnosis. This is achievable by careful history taking, examination and the use of special tests. The prognosis and treatment of each endodontic-periodontal disease type varies. Primary periodontal disease with secondary endodontic involvement and true combined endodontic-periodontal diseases require both endodontic and periodontal therapies. The prognosis of these cases depends on the severity of periodontal disease and the response to periodontal treatment. This enables the operator to construct a suitable treatment plan where unnecessary, prolonged or even detrimental treatment is avoided.

Received: *May 2010*
Accepted: *November 2010*
Dent Res J 2011; 8(1): 39-47

Introduction

The endodontium and periodontium are closely related and diseases of one tissue may lead to the involvement of the other. The differential diagnosis of endodontic and periodontal diseases can sometimes be difficult but it is of vital importance to make a correct diagnosis so that the appropriate treatment can be provided. Endodontic-periodontal lesions present challenges to the clinician as far as diagnosis and prognosis of the involved teeth are concerned. Etiologic factors such as bacteria, fungi, and viruses as well as various contributing factors such as trauma, root resorptions, perforations, and dental malformations play an important role in the development and progression of such lesions. The endo-perio lesion is a condition characterized by the association of periodontal and pulpal disease in the same dental element. The relationship between periodontal and pulpal disease was first described by Simring and Goldberg in 1964.[1] Since then, the term 'perio-endo lesion' has been used to describe lesions due to inflammatory products found in varying degrees in both periodontium and pulpal tissues.

Inter Relationship between pulpal & periodontal tissues

The effect of periodontal inflammation on dental pulp is controversial and conflicting studies abound.[2-10] It has been suggested that periodontal disease has no effect on the pulp before it involves the apex.[5] On the other hand, several studies suggested that the effect of periodontal disease on the pulp is degenerative in nature including an increase in calcifications, fibrosis, and collagen resorption, in addition to the direct inflammatory sequelae.[11,12] Dental pulp and periodontium have embryonic, anatomic and functional inter-relationships.[13] They are ectomesenchymal in origin, the cells from which proliferate to form dental papilla and follicle, which are the precursors of the pulp and periodontium, respectively. They are separated by the formation and development of tooth bud from the

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overlaying ectoderm into enamel and dentine.[14] The embryonic development gives rise to anatomical connections which remain throughout the life of the tooth. The apical foramen decreases in size as the proliferation of the Sheath of Hertwig continues. It remains patent and serves as the communication on which the pulpal tissues rely for nutrition and nervous innervation. As the root develops, ectomesenchymal channels get incorporated, either due to dentine formation around existing blood vessels or breaks in the continuity of the Sheath of Hertwig, to become accessory or lateral canals.[15] The majority of accessory canals are found in the apical part of the root and lateral canals in the molar furcation regions.[16] Tubular communication between the pulp and periodontium may occur when dentinal tubules become exposed to the periodontium by the absence of overlying cementum. These are the pathways that may provide a means by which pathological agents pass between the pulp and periodontium, thereby creating the perio-endo lesion.[15] Blomlof et al.[17] created defects on root surfaces of intentionally extracted monkey teeth with either open or mature apices. The root canals were either infected or filled with calcium hydroxide and replanted back in their sockets. After 20 weeks, marginal epithelial downgrowth was found on the denuded dentin surface of the infected teeth. Noxious elements of pulpal origin including inflammatory mediators and bacterial byproducts may leach out through the apex, lateral and accessory canals, and dentinal tubules to trigger an inflammatory response in the periodontium including an early expression of antigen presentation.[18] Released products are from living bacterial strains including spirochetes as well as of non-living pathogen.[19-23] Fungi and viruses are also implicated.[24-27] In certain cases, epithelial growth is stimulated and affects the integrity of periradicular tissues.[28-33] Jansson et al.[34] assessed the effect of endodontic pathogens on marginal periodontal wound healing of denuded dentinal surfaces surrounded by healthy periodontal ligament. Their results showed that in infected teeth, the defects were covered by 20% more epithelium while the noninfected teeth showed only 10% more connective tissue coverage. They concluded that pathogens in necrotic root canals may stimulate epithelial downgrowth along denuded dentin surfaces with marginal communication and thus augment periodontal disease. The organisms most often involved are probably bacteroides, fusobacteria, eubacteria, spirochetes, wolinellas, selenomonas, campylobacter, and peptostreptococci. Important qualities of cross-infecting organisms may be the ability to survive in highly reduced environments and motility. Precautions should be taken to prevent in vivo seeding of such micro-organisms, particularly in compromised teeth and hosts.[21]

Classification of perio-endo lesions

There are four types of perio-endo lesions and they are classified due to their pathogenesis.

1. Endodontic lesions: an inflammatory process in the periodontal tissues resulting from noxious agents present in the root canal system of the tooth.
2. Periodontal lesions: an inflammatory process in the pulpal tissues resulting from accumulation of dental plaque on the external root surfaces.
3. True-combined lesions: both an endodontic and periodontal lesion developing independently and progressing concurrently which meet and merge at a point along the root surface.
4. Iatrogenic lesions: usually endodontic lesions produced as a result of treatment modalities.

Pathogenesis

A. Endodontic lesions

When the pulp becomes inflamed/infected, it elicits an inflammatory response of the periodontal ligament at the apical foramen and/or adjacent to openings of accessory canals. These inflammatory lesions cause localized oedema and a resulting increase in intra-pulpal pressure and cell death.[5] Increased damage associated with an inflammatory exudate cause local collapse of the venous part of the local microvasculature. This causes local tissue hypoxia and anoxia resulting in localized necrosis, the chemical mediators of which cause further localized oedema, completing the cycle.[35]

Carious lesions or any direct exposure to the oral cavity of dentine or pulp may ingress bacteria to contaminate an otherwise sterile lesion. This is predominantly a mixed anaerobic infection.[6] Endodontic lesions are most frequently initiated and sustained by the apical foramen, followed by accessory and lateral canals, and most infrequently by dentinal tubules. A periapical lesion may perforate the cortical bone close to the apex, elevate the periosteum and overlying soft tissues, and drain into the gingival sulcus,[36] and form pseudopockets that simulate periodontal disease without neces-

sarily permanent damaging of the cementum and its fibres.[15] If the acute periapical drainage becomes chronic and drainage through the gingival sulcus continues a downgrowth of epithelium along the tract can result in a periodontal pocket in which secondary periodontal disease may complicate the lesion.[37] Simon, Glick and Frank[38] further categorized endodontic lesions into two subcategories: 1. primary endodontic lesions, when a sinus tract has formed to establish drainage, and 2. primary endodontic lesions with secondary periodontal involvement, when plaque formation occurring in the sinus tract with progression to periodontitis and associated calculus formation.

B. Periodontal lesions

Plaque and calculus initiate periodontal lesions. Inflammatory mediators cause destruction of gingival connective tissue, periodontal ligament and alveolar bone. Alteration of the root surface occurs by loss of the outer cementoblast layer and results in shallow resorptive lesions of cementum. Endotoxins produced by plaque bacteria also have an irritant effect on overlying soft tissue, preventing repair.[39] Although periodontal disease has been shown to have a cumulative damaging effect on the pulp tissue, total disintegration of the pulp will only be a certainty if bacterial plaque involves the main apical foramina, compromising the vascular supply.[15,36] The presence of an intact cementum layer is important for the protection of the pulp from pathogenic agents produced by the plaque bacteria.[36] Therefore, the exposure of dentinal tubules by the removal of cementum due to rigorous scaling and root-planning for the treatment of periodontal disease will allow bacterial invasion of the tubules. This would increase the likelihood of cumulative damage to the pulp.[15] Results from microbiological and immunological studies support the suggestion that the source of endodontic infection in perio-endo lesions with periodontal origin is the periodontal pocket bacteria.[40]

Categorization of periodontal lesions into two sub- categories: (Simon, Glick and Frank)[38]

1. Primary periodontal lesions, when there is a progression of periodontal lesion to involve the apex of a tooth while the pulp is vital (there may be some pulpal degenerative changes).

2. Primary periodontal lesions with secondary endodontic involvement; periodontal disease causes a resultant pulpal necrosis as it progresses apically.

C. Combined lesions

The pathogenesis of a true-combined lesion is identical to the pathogenesis of primary endodontic and periodontal lesions. These lesions are often indistinguishable from an advanced primary endodontic lesion with secondary periodontal involvement and/or a primary periodontal lesion with secondary endodontic involvement.[39] True combined endodontic-periodontal disease occurs with less frequency. It is formed when a coronally progressing endodontic disease joins an infected periodontal pocket progressing apically.[2,38] The radiographic appearance of combined endodontic-periodontal disease may be similar to that of a vertically fractured tooth. A fracture that has invaded the pulp space, with resultant necrosis, may also be labeled a true combined lesion and yet not be amenable to successful treatment.

D. Iatrogenic lesions

These include root perforations, overfilling of root canals, coronal leakage, trauma, chemical induced root resorption, intra-canal medicaments and vertical root fractures.[39] Root perforations are undesirable clinical complications that may lead to periodontal lesions. When root perforation occurs, communications between the root canal system and either peri-radicular tissues or the oral cavity may often reduce the prognosis of treatment. Root perforations may result from extensive carious lesions, resorption, or from operator error occurring during root canal instrumentation or post preparation.[41,42] At the site of perforation, an inflammatory reaction in periodontal ligament produces a degradation of surrounding tissues and formation of a lesion which can progress as a conventional primary endodontic lesion.[36]

The overfilling of root canals produces a lesion in exactly the same way as the lesion originating adjacent to the apical foramen rather than the lateral wall or pulpal floor. Coronal leakage is the leakage of bacterial elements from the oral environment along the restoration's margin to the endodontic filling. Studies have indicated that this factor may be an important cause of endodontic treatment failure.[43-46] Root canals may become recontaminated by microorganisms due to delay in placement of a coronal restoration and fracture of the coronal restoration and/or the tooth.[43] Madison and Wilcox[44] found that exposure of root canals to the oral environment allowed coronal leakage to

occur, and in some cases along the entire length of the root canal. Ray and Trope[45] reported that defective restorations and adequate root canal fillings had a higher incidence of failures than teeth with inadequate root canal fillings and adequate restorations. Nevertheless, even popular permanent restorative materials may not always prevent coronal leakage.[47] Cemented full crowns[48-49], as well as dentin-bonded crowns[50] also leaked. Dental injuries or trauma may take on many shapes but generally can be classified as enamel fractures, crown fractures without pulp involvement, crown fractures with pulp involvement, crown-root fracture, root fracture, luxation, and avulsion.[51] Treatment of traumatic dental injuries varies depending on the type of injury and it will determine pulpal and periodontal ligament healing prognosis.[52-57] The most common cause of vertical root fracture in endodontically treated teeth is the excessive force used during lateral condensation of gutta-percha. Widening of the periodontal ligament along one or both sides of the root, or bone loss in solitary tooth are the major radiographic findings. Mild pain or discomfort and swelling are the major clinical symptoms, and solitary pocket around one aspect of the suspected tooth is the major clinical sign. Certain chemicals used in dentistry have the potential to cause root resorption. Clinical reports[58-63] have shown that intracoronal bleaching with highly concentrated oxidizing agents, such as 30-35% hydrogen peroxide, can induce root resorption. The irritating chemical may diffuse through the dentinal tubules and when combined with heat, they are likely to cause necrosis of the cementum, inflammation of the periodontal ligament, and subsequently root resorption.[62,64] Replacement resorption or ankylosis occurs following extensive necrosis of the periodontal ligament with formation of bone onto a denuded area of the root surface. This condition is most often seen as a complication of luxation injuries, especially in avulsed teeth that have been out of their sockets in dry conditions for several hours. Certain periodontal procedures have been reported to induce replacement root resorption. The potential for replacement resorption was also associated with periodontal wound healing. Granulation tissue derived from bone or gingival connective tissue may induce root resorption and ankylosis. Vertical root fractures are most often caused when a tooth, often weakened due to undermining by caries, previous restorative treatment or a non-vital pulp becomes traumatised.[39] Leaching of the root canal contents or bacterial contamination of the fracture line may cause an inflammatory lesion in periodontal tissues.[36] The continuing lesion mimics those due to root perforation or over-filling of root canals.

Treatment

Diagnosis and preliminary considerations

The most important factor in the treatment is a correct diagnosis which is achieved by careful history taking, examination and the use of special tests.[16] Specific things to look for in the history include past disease, trauma and pain. The teeth are examined for abnormalities such as caries, defective restorations, erosions, abrasions, cracks, fractures, and discolorations. A discolored permanent tooth may often be associated with a necrotic pulp. A ‘‘pink spot’’ detected in the tooth crown may indicate an active internal resorption process. A conclusive diagnosis for pulpal disease cannot be achieved by visual examination alone. It therefore must always be accompanied by additional tests. Visual examination is dramatically improved by the use of enhanced magnification and illumination. 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.[65] Diagnosis of primary endodontic disease and primary periodontal disease usually presents no clinical difficulty. In primary endodontic disease, the pulp is infected and non-vital. On the other hand, in a tooth with primary periodontal disease, the pulp is vital and responsive to testing. However, primary endodontic disease with secondary periodontal involvement, primary periodontal disease with secondary endodontic involvement, or true combined diseases are clinically and radiographically very similar. If a lesion is diagnosed and treated as a primarily endodontic disease due to lack of evidence of marginal periodontitis, and there is soft-tissue healing on clinical probing and bone healing on a recall radiogragh, a valid retrospective diagnosis can then be made. The degree of healing that has taken place following root canal treatment will determine the retrospective classification. Radiographs are essential for detection of anatomic landmarks and a variety of pathological conditions. In addition, radiographs are of utmost importance

for documentation and legal purposes. 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, periradicular radiolucencies, thickened periodontal ligament, and alveolar bone loss. Advanced lesions and true-combined lesions are difficult to differentiate between and therefore, where doubt exists they should be considered as an endodontic lesion in origin.[65]

Treating primary endodontic lesions

Primary endodontic diseases usually heal following root canal treatment. The sinus tract extending into the gingival sulcus or furcation area disappears at an early stage once the affected pulp has been removed and the root canals well cleaned, shaped, and obturated. A review of patient, 4-6 months post-operatively should show healing of the periodontal pocket and bony repair.[66] Surgical endodontic therapy has been shown to be unnecessary even in the presence of large periradicular radiolucencies and periodontal abscesses.[67] Invasive periodontal procedures should be avoided as this may cause further injury to the attachment, possibly delaying healing.[15] Primary endodontic lesions with secondary periodontal involvement will not completely resolve with endodontic treatment alone. Root/re-root canal treatment is instituted immediately and the cleaned and shaped root canal filled with calcium hydroxide paste. 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 canals are eventually filled with a conventional obturation when there is clinical evidence of improvement.[15] The prognosis for primary endodontic lesions is good but worsens in the advanced stages of secondary periodontal involvement.

Treating primary periodontal lesions

Determining the prognosis depends upon the stage of periodontal disease and the efficacy of periodontal treatment. Primary periodontal lesions are treated by hygiene phase therapy in the first instance. Subsequently, poor restorations and developmental grooves that are involved in the lesion are removed as these are difficult areas to treat successfully. Periodontal surgery is performed after the completion of hygiene phase therapy if deemed necessary. Periodontal treatment removes the noxious stimuli and secondary mineralization of dentinal tubules allows the resolution of pulpal hypersensitivity.[67] If pulpal inflammation is irreversible root/re-root treatment is carried out followed by periodontal treatment; in some cases surgical intervention is advantagous.[66] The prognosis of periodontal lesions is poorer than that of endodontic lesions and is dependent on the apical extension of the lesion. As the lesion advances, the prognosis approaches that of a true-combined lesion.

Treating true combined lesions

Primary endodontic lesions with secondary periodontal involvement may also occur as a result of root perforation during root canal treatment, or where pins or posts have been misplaced during coronal restoration. Symptoms may be acute, with periodontal abscess formation associated with pain, swelling, pus exudate, pocket formation, and tooth mobility. A more chronic response may sometimes occur without pain, and involves the sudden appearance of a pocket with bleeding on probing or exudation of pus. True-combined lesions are treated initially as primary endodontic lesions with secondary periodontal involvement. The prognosis of a true-combined perio-endo lesion is often poor or even hopeless, especially when periodontal lesions are chronic with extensive loss of attachment.[8] Root amputation, hemisection or separation may allow the root configuration to be changed sufficiently for part of the root structure to be saved. The prognosis of an affected tooth can also be improved by increasing bony support which can be achieved by bone grafting[68] and guided tissue regeneration.[69] This is due to the most critical determinant of prognosis being a loss of periodontal support. Cases of true combined disease usually have a more guarded prognosis than the other types of endodontic–periodontal problemsThus, the prognosis of combined diseases rests with the efficacy of periodontal therapy.[70-76]

Treating iatrogenic lesions

Although the first priority is to close the iatrogenic communication, the aim is to produce a seal.[67]

Root perforations are treated according to their aetiology. The outcome of the treatment of root perforations depends on the size, location, time of diagnosis and treatment, degree of periodontal damage as well as the sealing ability and biocompatibility of the sealer. It has been recognized that the success of the treatment depends mainly on immediate sealing of the perforation and appropriate infection control. Several materials such as MTA, Super EBA, Cavit, IRM, glass ionomer cements, composites, and amalgam have been recommended to seal root perforations.[70-74] Palatal perforations are difficult to manage, even surgically, and frequently lead to extraction. The successful treatment of root perforations depends principally on early detection and sealing. Although the prognosis is deemed poor, it appears that a successful outcome can frequently be achieved.[67] Lesions attributable to over-filling of root canals and intra-canal medicaments can usually be resolved by periradicular surgery, probably accompanied by a retrograde root filling. Teeth with lesions caused by vertical root fractures have a hopeless prognosis and should be extracted.[15] A review of the literature examined the factors associated with long-term survival of endodontically treated teeth,[75] and concluded that: (1) post space preparation and cementation should be performed with rubber-dam isolation, (2) the post space should be prepared with a heated plugger, (3) a minimum of 3 mm of root canal filling should remain in the preparation, (4) the post space should be irrigated and dressed as during root canal treatment, (5) leak-proof restorations should be placed as soon as possible after endodontic treatment, and (6) endodontic retreatment should be considered for teeth with a coronal seal compromised for longer than 3 months.[75]

Conclusion

A perio-endo lesion can have a varied pathogenesis which ranges from quite simple to relatively complex one. Having enough knowledge of these disease processes is essential in coming to the correct diagnosis. It is important to remember that the recognition of pulp vitality is essential for a differential diagnosis and for the selection of primary measures for treatment of inflammatory lesions in the marginal and apical periodontium. Diagnosis of teeth with necrotic pulps can be difficult to establish. The entire dentition should be examined for possible causes of pain before commencing treatment. Some periodontal lesions of endodontic origin can heal following root canal treatment alone.[77] The endodontic treatment can be completed before periodontal treatment is provided when there is no communication between the disease processes. However, when there is a communication between the lesions of the two diseases, then the root canals should be medicated until the periodontal treatment has been completed and the overall prognosis of the tooth has been reassessed as being favorable. The use of non-toxic intracanal therapeutic medicaments is essential to destroy bacteria and to help encourage tissue repair.[78] Because the primary aetiology is infection, endodontic treatment is directed at control and elimination of the root canal flora by working in a sterile way. Based on current knowledge, the best available method for obtaining clean, microbe-free root canals is instrumentation with antimicrobial irrigation reinforced by an intracanal dressing with calcium hydroxide.[79] The presence of a combined endodontic-periodontal lesion will always result in a compromised situation following treatment. Even with apparently successful treatment, the tooth will still be compromised as there is likely to be some gingival recession and loss of periodontal attachment and bone support. It is of utmost importance that the patient maintains good oral hygiene and obtains regular professional care for this region. The tooth anatomy and the etiology of endodontic-periodontal lesions offer a strong base for establishing a correct diagnosis. Due to the complexity of these affections, an interdisciplinary approach with a good collaboration between endodontists, periodontologists and microbiologists, is recommended.

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