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Endodontic-periodontal lesion: A two-way traffic

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Abstract

Endodontic-Periodontal lesions are complicated disease entity, frequently encountered in day-to-day dental practice, difficult to diagnose and treat. The success of management of a combined periodontal and endodontic lesion depends on the elimination of both of the disease processes. In the case of a combined endo-perio lesion, the endodontic therapy results in healing of the endodontic component, while the prognosis of tooth would finally depend on the healing of the periodontal lesion. This literature review aims to assess the causes and consequences of periodontal-endodontic lesions, as well as its clinical, radiographic and microbiological aspects, and presents a comprehensive outline of diagnosis of perio-endo lesions and a sequential treatment protocol to the same.

Keywords: Retrograde pulpitis, retrograde periodontitis, pulp, periodontium.

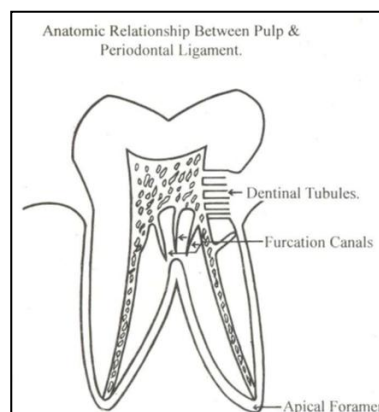
Introduction

The tooth, the pulp within it and its supporting periodontal tissues should be viewed as one biological unit. The interrelationship among these structures influences themselves during health and disease [1]. The periodontal-endodontic lesions are characterized by the co-existence of the pulpal and periodontal disease in a same tooth, which makes complex its diagnosis, as well as management, because a single lesion often present signs of endodontic and periodontal involvement. This suggests that one disease might be the cause or consequence of the other, or had originated from two different and independent processes which were mingled by their advancement [2]. The relation between the pulp and the periodontium was a matter of concern since long back. Effects of periodontal disease on pulp was first depicted by Turner and Drew (1919) [1] The reverse was first narrated by Simring and Goldberg (1964) [2] Cahn (1927) and Sicher (1936) [3] first pointed the communicating channels between pulp and periodontal tissues.

Pathways of Communication

The periodontium and pulp have embryonic, structural and functional interrelationship. From ecto Mesenchymal cells develop the dental papilla and follicle, which differentiate into periodontium and the pulp respectively. This developmental origin results anatomical connections, which remain throughout life [4]. Three main pathways have been attributed to the development of periodontal-endodontic lesions (Rotstein & Simon, 2004) [5].

- Apical foramen
- Lateral & Accessory canals
- Dentinal tubules



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The apical foramen represent the principal route of communication between pulp and periodontal ligament. Bacteria themselves, or their products, and inflammatory factors can exit through apical foramen, resulting peri-apical pathosis; or reverse happens in deep periodontal pockets.

Lateral and accessory canals present a possible route for spread of pathogens from pulp to periodontal tissues, and vice versa. De Deus (1975), studying 1140 human teeth, observed that, about 17% of all teeth presented lateral canals in apical third, about 9% in middle third and 2% in coronal third of root [3]. Gutmann (1978), studying 102 human teeth, noticed 25.50% sample presented lateral canals in furcation area alone [4]. Kirkham (1975), studying 1000 human teeth with advanced periodontal involvement, observed only 2% of lateral canals associated with periodontal pocket.

Exposed dentinal tubules, in area where dentine is devoid of cementum, can act as pathway for communication between pulp and periodontium. Exposures take place from development defects, wear defects, restorative procedures and periodontal therapies. The density of dentinal tubules varies from 15000 per Sq. mm. at C.D. Junction cervically, while 8000 near apex; to 57000 at pulpal end. Again, the diameter ranges from 1µm in periphery to 3µm toward pulp. Dentine exposure occurs at C.E. Junction in 18% of teeth in general and 25% of anterior Teeth.

Palato-gingival grooves are found in maxillary lateral incisor, extending varying distance apically from cingulum. Radiographically they appear as 'tear drop shaped area' and para-pulpal line' (dark vertical line parallel to canal). They provide funnel like area aiding into plaque retention. These are related to deep 'tubular' periodontal pocket, with localized periodontal disease, with/without pulpal pathosis, depending on their depth and extent.

Literature Review

Pulpal Disease & Periodontal Health

Pulpal lesion involves in inflammatory changes, causing inflammatory response of the periodontal ligament at the apical foramen and at the opening of the accessory canals [7], resulting rapid and wide spread destruction of PDL, with the production of radiolucency peri-apically, or in the furcation or at various points along the root. This is referred to as 'Retrograde periodontitis' with signs and symptoms including deep localized periodontal pocket, purulent inflammatory exudates, angular bone loss, swelling and bleeding of the gingival tissues and increased tooth mobility. Osseous destruction involves furcal area much earlier, because of [1] greater incidence of furcal canal and [2] furcal bone, being thinner, resorbes faster (Moss, 1965).

In animal model, change in periodontal ligament were found after pulpotomy and placement of caustic agent in pulp chamber (Seltzer *et al*, 1967) [7]. Periodontal therapy of a tooth with pulpal disease and peri-apical radiolucency results in poor periodontal healing (Ehnevid *et al*, 1993) [8]. In animal model, free autogenous soft tissue graft failure rate was greater, when graft was placed over untreated teeth with diseased pulp (Perlmutter *et al*, 1987). Hence, precious pre-assessment of pulpal status is critical for a successful periodontal therapy.

Periodontal Disease & Pulpal Health

Controversies and conflicts exist regarding the effects of periodontal inflammation on the pulp. Theoretically periodontal pathosis can adversely affect pulp, producing retrograde pulpitis (Simring & Goldberg, 1964) [2]. A greater

incidence of pulpal inflammation and degeneration was reported in periodontally involved teeth than teeth with no periodontal involvement, in a study on 85 human teeth by Bender & Seltzer (1972) [11]. Periodontal disease has seen to exert no effect on pulp until the pocket has extended to the apex (Czarnecki & Schilder, 1979); or periodontal damage has opened an accessory canal to oral environment (Rubach & Mitchel, 1965) [26]. If the microvasculature of apical foramen remains intact, pulp maintains its vitality (Langeland *et al*, 1974) [12].

The effects of periodontal inflammation on pulp is atrophic in nature; including calcification, increase in collagen content, formation of reparative dentine and narrowing of canal spaces; or resorptive; in addition to direct inflammatory sequel. (Mandi *et al*, 1974) [5] Root planing may exert same effects on pulp and has been shown to increase the rate of formation of reparative dentine. (Hattler & Listgarten, 1984).

Microbiota

The oral cavity contains more than 600 species of microorganisms, and the gram negative anaerobic ones are directly related to both the peri-apical and periodontal lesion, among which the endodontic is less complex than periodontal pathogen [13].

Bacteria: *Aggregatibacter actinomycetemcomitans*, *Bacteroides frolythus*, *Ekinella corrodens*, *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Prevotella intermedia* and *Treponema denticola* are seen to exist in both endodontic and periodontal infection [12].

Fungi: *Candida albicans* is prevalent in both in endodontic lesion as well as sub-gingival plaque [6,7]

Viruses: Recent study indicates Cytomegalo virus, Epstein-Barr virus, herpes virus could be involved in pathogenesis of periodontal and endodontic disease [8,9].

Kobayashi *et al*. [15] detected microorganisms common to canal and p pockets were detected from endodontic samples in 15 devitalized teeth, without caries and with periodontal advancement including: *Eubacterium* and *Fusobacterium spp*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Peptostreptococcus spp*, *Capnocytophaga spp*, *Actinomyces spp* and *Streptococcus spp*.

The similarity between the endodontic and periodontal microbiota indicates the possibility of the occurrence of cross infection between the root canal and periodontal pocket.

Classification of endodontic-periodontal lesion

Simon, Glick & Frank (1972) [10] classified endodontic-periodontal lesion, based upon origin of the disease and its spread, as following:

1. Primary endodontic lesion (when the lesion is entirely endodontic in origin)
2. Primary endodontic lesion with secondary periodontal involvement (when periodontal defect develops in endodontic ally affected teeth)
3. Primary periodontal lesion (when the lesion is entirely periodontal in origin)
4. Primary periodontal lesion with secondary endodontic involvement (when endodontic problem arises in periodontally diseased teeth)
5. True combined lesion (when both endodontic and periodontal disease develop independently and unite)

Stock (1988) ^[11] modified Simon *et al* classification and omitted Class V lesions as he argued that both Class II and Class IV lesions become combined lesion in advanced stage. Similarly, one can argue on including ‘primary periodontal lesion’ in the classification

Khalid Al-Faujan (2014) modified the primary endodontic lesions, ‘*since it has no periodontal relationship*’; and proposed a new endo-periodontal interrelationship classification as follows ^[12]:

1. Retrograde periodontal disease
 - a) Primary endodontic lesion with drainage through periodontal ligament
 - b) Primary endodontic lesion with secondary periodontal involvement
2. Primary periodontal lesion
3. Primary periodontal lesion with secondary endodontic involvement
4. Combined endodontic-periodontal lesion
5. Iatrogenic periodontal lesion

Here, the primary endodontic lesion with/without periodontal involvement secondarily is referred as retrograde periodontitis, (as such periodontal disease begins from apex and then extends coronally- just reverse to common periodontal disease).

Weine (1982) ^[13] presented a different classification depending on clinical presentation as follows:

1. Symptoms clinically and radiographically simulate periodontal disease, but are in fact owing to pulpal lesions.
2. That has both pulpal and peri-apical disease and periodontal disease concomitantly
3. That has no pulpal disease but requires endodontic therapy plus root amputation in order to gain periodontal healing
4. Symptoms clinically and radiographically simulate pulpal and peri-apical disease but in fact has periodontal origin.

Grossman (1988) followed the oldest classification by Oliet and Pollock (1968) and classified the endo-perio lesions according to treatment need, as follows:

1. Teeth requiring endodontic therapy only – it includes

- a) Necrotic pulp and peri-apical lesion with/without sinus tract
- b) Chronic peri-apical abscess with sinus tract passing through a/a.
- c) Root fracture
- d) Root resorption
- e) Replnatation
- f) Intentional endodontic therapy
- g) Radisectomy
- h) Incomplete closure of apex

2. Teeth requiring periodontal therapy only – it includes

- a) Occlusal trauma causing reversible pulpitis
- b) Occlusal trauma plus inflammation of gingiva resulting in pocket
- c) Overzealous periodontal therapy causing pulpal sensitivity
- d) Deep and extensive infra-bony pocket, extending beyond apex, sometimes coupled with root desorption, yet with a vital pulp.

3. Teeth requiring both endodontic-periodontal procedures – it includes:

- a) Any type 1 lesion causing irreversible reaction to a/a, and hence require periodontal therapy
- b) Any type 2 lesion causing irreversible reaction to pulp, and hence require endodontic therapy

World workshop for classification of periodontal diseases (1999) (14) has presented a new classification depending on origin of disease as follows:

- a) Endodontic-periodontal lesion
- b) Periodontal-endodontic lesion
- c) Combined lesion

Primary Endodontic Lesion

• **Pathogenesis**

- It arises as sequel of pulpitis from dental caries, wear defects, trauma and fracture.
- A primary endodontic lesion presents a necrotic pulp and a chronic peri-apical abscess with a sinus tract draining through periodontal ligament space or gingival sulcus.

• **Clinical Features**

- The lesion presents ‘isolated’ periodontal problem in relation to the affected tooth only, without a generalized periodontal disease.
- H/O pulpitis.
- A sinus tract, originating from apex, is often present in sulcus.
- Negative pulp vitality test.

• **Treatment**

- Endodontic therapy – must be performed in multiple appointment, to reevaluate healing process between the beginning and completion of treatment.
- Periodontal therapy isn’t required usually.

• **Prognosis**

- They exhibit good prognosis. Radiographic and clinical healing occurs rapidly.
- A sinus tract heals soon after canal debridement. Healing completes within 3-6 months.

Primary Endodontic Lesion with Secondary Periodontal Involvement

• **Pathogenesis**

- It arises when periodontal problem develops on teeth with PEL.
- If the primary endodontic lesion with a sinus tract isn’t detected and hence, treated early; plaque and calculus is often deposited in draining sinus tract; creating a secondary periodontal problem

• **Clinical Features**

- Negative pulp vitality test.
- Presence of plaque and calculus, in the way of sinus tract.

• **Treatment**

○ **Endodontic therapy**

- Periodontal therapy – should not be employed until complete debridement of canal is achieved.

- **Prognosis**
 - Prognosis of endodontic therapy is usually predictable.
 - Regeneration of periodontal tissue depends upon the extent of tissue destruction.

Primary Periodontal Lesion

- **Pathogenesis**
 - The lesion develops as sequelae of progressing periodontal problem extending to the apex.
 - Plaque represents the prime etiologic factor.
- **Clinical Features**
 - Patient presents with generalized chronic periodontitis, sometimes having a sinus tract, with periodontal probing even extending to apex of the affected tooth, showing no pulpal exposure through caries/trauma/fracture. Patient can experience minimal or no pain.
 - Positive pulp vitality test.
- **Treatment**
 - Surgical/ non-surgical periodontal therapy.
 - Re-evaluation must be done periodically to check for retro-infection of pulp.

Prognosis

The prognosis is entirely dependent on periodontal therapy and hence, extent of periodontal damage.

Primary Periodontal Lesion with Secondary Endodontic Involvement

- **Pathogenesis**
 - It arises as retro-infection of pulp, when periodontal lesion extends to apex.
 - It may also follow the path through a lateral canal. Cervical abrasion and SRP also can add to such problem.
- **Clinical Features**
 - Negative/ altered pulp vitality test (as pulp can be necrotic/ partially vital, especially in multi-rooted teeth)
- **Treatment**
 - Surgical/ non-surgical periodontal therapy
 - Endodontic therapy
- **Prognosis**
 - The prognosis depends upon periodontal therapy and hence, extent of periodontal damage.
 - Healing of peri-apical lesion isn't predictable owing to periodontal communication

True Combined Lesion

- **Pathogenesis**
 - Here pulpal and periodontal lesions develop and unite independently.
 - It develops when an endodontic lesion progressing coronally joins with a pre-existing periodontal defect progressing apically.
- **Clinical Features**
 - Features are similar to that of primary periodontal lesion. In addition, there must be some caries, trauma, fracture, wear defects, deep restoration or history of endodontic therapy. So patient often has severe pain.
 - Negative pulp vitality test.

- **Treatment**
 - Endodontic therapy and periodontal therapy.
 - Root resection can be in need with regenerative therapy.
- **Prognosis**
 - Prognosis of lesion is related to extent of periodontal damage.
 - Though response of endodontic therapy is predictable, the tooth shows hopeless prognosis, if majority of osseous support is lost from periodontal lesion.

Diagnosis

The correct diagnosis of the periodontal-endodontic lesions is fundamental to set the treatment plan and assess the prognosis. Diagnosis of primary endodontic disease and primary periodontal disease usually present no clinical difficulty. In primary periodontal lesion, the pulp is vital and responds to pulp vitality test. In primary endodontic lesion, the pulp is infected and non-vital and doesn't respond. 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^[1]. Accurate diagnosis can be achieved by detailed history taking. According to the studies of Goldman and Schildert^[22], cases of caries, traumas, defective restorations and wear defects, which can develop a pulp necrosis, indicate the endodontic origin of the lesion. Absence of these and presence of calculus, plaque, inflammation of marginal tissue and generalized periodontitis, indicate periodontal lesion^[23].

Treatment Sequence

Before the doing an advanced restorative treatment for an endodontic-periodontal lesion, the prognosis of the involved tooth should be evaluated thoroughly. Whether there is a functional need for the tooth, whether the tooth is restorable after the lesion will heal and whether the patient is suitable for a lengthy, costly and invasive treatment are crucial factors that should be taken into account. If any of those appears negative, extraction is the treatment of choice^[15].

When the pulp is non-vital and infected, conventional endodontic therapy alone will resolve the lesion. Endodontic surgery is not necessary, even with the presence of large peri-radicular radiolucency's and abscesses, but can be of need when large peri-apical radiolucency remains even after the non-surgical endodontic therapy. Johnson and Orban showed that periodontal disease that remained after unsuccessful endodontic therapy cleared up after successful endodontic therapy^[31].

If primary endodontic lesions persist, despite extensive endodontic therapy, then it may also have secondary periodontal involvement or it can be a true combined lesion. Hiatt and Amen suggested that persistent periodontal disease resolves only after definitive periodontal therapy is accompanied by successful endodontic therapy^[33].

In case of secondary periodontal involvement, root canal treatment is employed immediately and the cleaned and shaped canal is filled with calcium hydroxide paste, which has anti-microbial, anti-inflammatory and proteolytic property, inhibiting resorption and favouring repair. It also prevents periodontal contamination of instrumented canals via patent channels connecting the pulp and periodontium, before periodontal treatment removes the contaminants. Treatment results should be evaluated after two to three months and only then should periodontal treatment be considered.

Primary periodontal lesions should be treated first by non-surgical periodontal therapy. Periodontal surgery, in the form of pocket surgery and resective and regenerative procedure, is required for deeper pockets and angular bone defects.

Periodontal lesions with early secondary endodontic involvement presents with reversible pulpal hypersensitivity, which can be treated purely by periodontal therapy. Periodontal procedure removes noxious stimuli, and subsequent secondary mineralization of dentinal tubules allows the resolution of hypersensitivity. If pulpitis is irreversible, root canal treatment is carried out, followed by periodontal treatment. Decision of periodontal surgery depends upon destruction of periodontal structure

True combined lesions are treated initially as for primary endodontic lesions with secondary periodontal involvement. Root resection or hemi-section often yields good prognosis following palliative periodontal therapy and root canal treatment to root to be saved

Iatrogenic lesions like perforation during root canal treatment or preparation of the canal for post and core, require a surgical approach or sealing with a ZOE, GIC, or MTA immediately.

The prognosis can also be improved by enhancing bony support, achieved by regenerative procedure like GTR [32]. These advanced treatment options are based upon responses to basic periodontal and endodontic therapy over along time period.

Discussion

It is evident that both the pulp and the periodontium are closely linked together, through the apical foramen, accessory canals, and dentinal tubules of the root, and one can interfere on the integrity of the other. Though the effect of pulpal disease on periodontal health is well established, reverse evokes controversy and conflicts till today.

Authors like Ribach and Mitchell [26] affirmed that the periodontal disease affects the pulp through accessory canals in the furcation and apical foramen. Adriaens *et al.* [27] reported that the bacteria coming from the periodontal pockets can infect the pulp through the dentinal tubules during aggressive periodontal therapy.

Seltzer *et al.* [7] contraindicated this idea, because even with the removal of the cementum during SRP in vital teeth, the pulp tissue will be protected against the harmful agents through forming reparative dentin. Moreover, a dentinal fluid move towards the exterior protecting against the diffusion of bacterial toxins from the dentinal surface.

Langeland *et al.* opined that the pulp would be affected by the periodontal disease only when the apical foramen is involved [12], which was supported by Czarnecki and Schilder too. [6]

Treatment and prognosis of primarily endodontic and primarily periodontal disease are very straightforward. However, the most guarded prognosis is experienced with true combined lesions, and the treatment is more complex. Endodontic therapy is usually more predictable and employment before periodontal procedures imparts better effect on periodontal healing, and the prognosis largely rests with the severity and extent of the periodontal damage and the efficacy of periodontal therapy.

Conclusion

Based on the literature review, it can be concluded that differentiating the origins of the periodontal-endodontic lesions, including all possible routes of communication between the pulp and the periodontium, is of extreme importance to a dentist. Through this knowledge, the dentist

would obtain the correct diagnosis and treatment plan, achieving greater chances of success in the management of the periodontal-endodontic lesions.

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