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Endodontic-periodontal continuum: An enigma for dentistry

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Abstract
Endodontic-periodontal lesions are a challenge to everyday clinical practice. The key factor to achieve success in treatment of endodontic periodontic lesions is elimination of the disease process in both areas. .In cases where there is combined lesion, the endodontic treatment helps in elimination of existing inflammation and healing of the endodontic component while the tooth survival and prognosis depends on healing of periodontal lesion. This article aims to discuss clinical, microbial, and the radiological aspects involved in diagnosis and sequential treatment protocol of perio-endo lesions.

Keywords: retrograde pulpitis, retrograde periodontitis, pulp, periodontium

Introduction

The tooth along with its pulp and supporting periodontium forms a single biological unit. The close connection between these structures has an impact on states of health and disease of the tooth. The periodontal -endodontic lesions show co-occurrence of pulpal and periodontal lesions on the same tooth. The diagnosis is complicated by the fact that a single lesion presents signs of periodontal as well as endodontic involvement, indicating that one disease caused the other or both disease processes originated from independent sources and mingled with advancement of the disease.

The impact of periodontal disease on the pulp was first noted by Turner and Drew (1919) [1].The influence of pulp on periodontal ligament on the other hand was first explained by Simring and Goldberg (1964) [2]. Cahn (1927) and Sicher (1936) then identified the presence of communication between the pulp and periodontal tissues [3].

Pathways of communication

The periodontium and pulp both have structural and functional relationships owing to similar developmental origin. The ectomesenchymal cells of the developing tooth bud differentiate into dental papilla and follicle which then give rise to the pulp and periodontium respectively These anatomical connections, which remain throughout life [4]. Three main pathways are linked to the development of periodontal-endodontic lesions (Rotstein& Simon, 2004) [5].

- Apical foramen
- Lateral & Accessory canals
- Dentinal tubules

The apical foramen forms a two-way communication between pulp and periodontal ligament. Bacteria or bacterial products can exit the apical foramen leading to peri-apical disease. The reverse occurs in case of deep periodontal pockets.

Spread of pathogens can occur from pulp to periodontal tissue and vice versa through lateral and accessory canals. DeDeus (1975), observed 1140 human teeth, and found 17% of all teeth have lateral canals in the apical third, 9% in middle third and 2% in the coronal third of root[3]. Gutmann (1978), found 25.50% teeth presented lateral canals in the furcation alone[4]. Kirkham (1975), among 1000 human teeth with advanced periodontal involvement, found only 2% of lateral canals to be associated with periodontal pocket.

Developmental defects, wear defects, periodontal treatment and restorations can lead to dentinal areas denuded of cementum, exposing the dentinal tubules. These exposed areas form a pathway of communication between pulp and periodontium. Dentinal tubules at the cemento-dental junction vary in density from 15000 per sq. mm cervical to about 8000 per sq. mm at the apex and 57000 per sq. mm close to the pulp. Dentinal tubules are tapered structures larger near the pulp with diameter approximately 3µm and slender in the periphery about 1µm. Dentine exposure occurs at C.E. Junction in 18% of all teeth and 25% of anterior teeth. The anatomy of maxillary lateral incisor with its palato-gingival grooves extending apically from cingulum makes it conducive to plaque retention. These are associated with deep tubular pockets, localised periodontal disease with pulpal pathosis, depending on depth and extent of the periodontal pocket. Radiographically they present a 'tear drop shaped area'.

Literature Review

Pulpal disease & periodontal health

Inflammation of the pulp causes inflammatory responses of the periodontal ligament at the apical foramen and accessory canals opening[7], resulting in the wide spread and rapid destruction of periodontal ligament, which appears as a radiolucency in peri-apical areas, furcations and other parts around the root. Signs and symptoms such as deep localized pockets, purulent inflammatory exudate, angular loss of bone, increasing mobility of the tooth along with gingival swelling and bleeding indicate 'retrograde periodontitis'. The furcation area shows osseous destruction earlier because of (1) greater incidence of furcal canal and (2) furcal bone, being thinner, resorbs faster (Moss, 1965).

After pulpotomy and placement of caustic agent in pulp chamber, Seltzer *et al*, (1967) [7] found changes in the periodontal ligament, in animal model. Ehnevid *et al*, (1993) [8] found that there is poor healing of periodontal tissues in teeth with pulp infection and associated periapical lesion. In the animal study, Perlmutter *et al*, (1987) found the rate of failure of the free autogenous soft tissue graft was greater, when graft was placed over untreated teeth with diseased pulp. Assessment of pulp status is a pre-requisite to success of periodontal treatment. Hence, proper pulpal evaluation is necessary prior periodontal therapy.

Periodontal disease & pulpal health:

Theoretically periodontal pathosis can produce retrograde pulpitis (Simring & Goldberg, 1964) [2]. Teeth that have periodontal involvement show more incidence of pulp inflammation and degeneration, in a study on 85 human teeth by Bender & Seltzer (1972) [11]. The pulp is affected when the periodontal pocket extends to the apical third of root, disrupting its blood supply through apical foramen (Czarnecki & Schilder, 1979); or periodontal damage has opened an accessory canal to oral environment (Rubach& Mitchel, 1965) [26].

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. Periodontal inflammation causes changes simulating pulp atrophy which includes increased collagen, reparative dentin formation, calcification and narrowing of the canal space (Mandi *et al*, 1974) [5] Root planing has also been shown to increase the rate of formation of reparative dentine (Hattler & Listgarten, 1984).

Microbiota

Periodontal and periapical lesions are both directly caused by gram negative anaerobic bacteria. The disease process in the periodontium shows more complexity than the disease process in the root canal [12].

Bacteria: Aggregatibacter actinomycetemcomitans, Bacteroides frosythus, 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 Cytomegalovirus, 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 andp pockets were detected from endodontic samples in 15 devitalized teeth, without caries and with periodontal advancement including: Porphyromonas gingivalis, Prevotella intermedia, Peptostreptococcus spp, Capnocytophaga spp, Actinomyces spp, Streptococcus spp, Eubacterium and Fusobacterium spp.

Cross infection between root canal and periodontal pocket can be attributed to similarity in disease causing microbiota.

Classification of Endodontic-Periodontal Lession

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 endodontically 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) [12] modified the primary endodontic lesions, 'since it has no periodontal relationship; and proposed a new endo-periodontal interrelationship classification as follows:

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 periapical 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 periapical disease but in fact have 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 periapical lesion with/without sinus tract
   b) Chronic periapical abscess with sinus tract passing through a/a.
   c) Root fracture
   d) Root resorption
   e) Replantation
   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 resorption, 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:

1. Endodontic-periodontal lesion
2. Periodontal-endodontic lesion
3. Combined lesion

Primary Endodontic Lesion

- Pathogenesis

A primary endodontic lesion presents a necrotic pulp and a chronic periapical abscess with a sinus tract draining through periodontal ligament space or gingival sulcus.

- Clinical Features
  - Appears as an 'isolated' periodontal problem in relation to the affected tooth only, without a generalized periodontal disease.
  - H/O pulpitis.
  - negative pulp vitality test
  - A sinus tract, originating from apex, is often present in sulcus.

- Treatment
  - Root canal treatment in mutiple sittings and evaluate healing of tissues from progressively from start of treatment to end of treatment
  - Periodontal therapy isn't required usually.

- Prognosis

A sinus tract heals soon after canal debridement. Healing completes within 3-6 months. Such cases show good prognosis.

Primary endodontic lesion with secondary periodontal involvement

- Pathogenesis

Occurs when secondary periodontal problem is seen in a tooth with primary endodontic lesion. If the endodontic lesion with a sinus tract isn't detected and treated early; plaque and calculus deposited in draining sinus tract create 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 –post canal debridement is achieved

- Prognosis

Predictable prognosis of endodontic treatment, though regeneration of periodontal tissue depends upon the extent of tissue destruction.

Primary Periodontal Lesion

- Pathogenesis

Develops as a result of periodontal pocket extending to the root apex. Here plaque is the main causative factor.

- Clinical Features
  - Generalized chronic periodontitis often with sinus tract, probing extenfs to apex of affected tooth.No pulp exposure from caries, trauma or fracture. Patient reports minimum or no pain.
  - Positive pulp vitality test.

- Treatment
  - Surgical/ non-surgical periodontal therapy.
  - Periodic re-evaluation to check for retrograde pulp infection

- Prognosis

The extent of periodontal damage and response of periodontium to therapy dictates the prognosis of such cases

Primary periodontal lesion with secondary endodontic involvement

- Pathogenesis

Arises as retro-infection of pulp, when periodontal lesion extends to apex.

- 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

Prognosis again depends on extent of damage and response of periodontium to therapy. Healing of periapical lesion isn't predictable owing to periodontal communication

True Combined Lesion

- Pathogenesis

Independant development of pulpal and periodontal lesions, where endodontic lesion progressing coronally joins with a pre-existing periodontal defect progressing apically.

- Clinical Features
  - Similar to that of primary periodontal lesion. Plus there is some caries, trauma, fracture, wear defects, deep restoration or history of endodontic therapy. Patient often reports severe pain.
  - Negative pulp vitality test.

- Treatment
  - Endodontic therapy and periodontal therapy.
  - Root resection and 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

Treatment plan and prognosis of tooth depends on correct diagnosis of the periodontal-endodontic lesion.

Primary lesions do not present difficulty in diagnosis. The pulp remains vital and responds to pulp vitality test in case of primary periodontal lesion. On the other hand primary endodontic lesions present pulp infection and loss of pulp vitality.

Primary endodontic disease with secondary periodontal involvement, primary periodontal disease with secondary endodontic involvement, or true combined diseases show overlapping features clinically and radiographically [1]. A detailed history help may help in accurate diagnosis. Studies by Goldman and Schildert show presence of caries, traumas, defective restorations can cause pulp necrosis, indicating the endodontic origin of the lesion [22]. While presence of plaque, calculus, marginal tissue inflammation and generalized periodontitis indicate periodontal origin of lesion [23].

Treatment sequence

Thorough evaluation of the tooth should precede treatment of the tooth .The functional need of the tooth, potential for restoration after the lesion is healed, length and cost of the invasive treatment along with patient compliance are all important factors to be considered into account. If any of those appears negative, extraction is the treatment of choice [15].

Conventional root canal treatment can be used in cases where the pulp is non-vital and infected. Surgery is required only if a large peri-apical lesion continues to persist after root canal treatment. Johnson and Orban showed that periodontal disease that remained after unsuccessful endodontic therapy cleared up after successful endodontic therapy [31].

Primary endodontic lesions that remain even after extensive endodontic treatment can actually have a secondary periodontal involvement or 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 [30].

In secondary periodontal involvement, root canal treatment is done 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. Patient recall and evaluation is done for eight to twelve weeks before rendering periodontal treatment.

Primary periodontal lesions should be treated first by non-surgical periodontal therapy. Deeper pockets and angular bone defects may require periodontal surgery, in the form of pocket surgery and ressective and regenerative procedure.

Periodontal lesions with early secondary endodontic involvement often have reversible pulpal hypersensitivity, which can be treated by periodontal therapy. Periodontal procedure removes pathogens, and promote secondary mineralization of dentinal tubules, which allows the resolution of hypersensitivity. In case of irreversible pulp damage, root canal treatment is carried out, followed by periodontal treatment.

Approach toward treatment of True combined lesions is initially same 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 of the root to be retained.

Lesions that are iatrogenic, 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].

Discussion

The apical foramen, accessory canals and dentinal tubules of

the root create multiple pathways of communication between pulp and periodontium. The impact of pulpal disease on periodontal health is well established but the influence of periodontal disease on pulpal health still remains unclear.

Ribach and Mitchell[26] support claimed through their studies that the periodontal disease affects the pulp through accessory canals near the apical foramen and the furcation. Bacteria from periodontal pocket can be translocated into the pulp via dentinal tubules during aggressive periodontal treatment as reported by Adriaens *et al*. [27]

This idea was refuted by Seltzer *et al*. [7], because even after removal of the superficial cementum during SRP in vital teeth, the pulp tissue will be protected through forming reparative dentin. In fact, dentinal fluid moves inside outword protecting against the diffusion of bacterial toxins from the surface of the dentine.

Langeland *et al*. stated that the pulp would be affected by the periodontal disease only when the apical foramen is involved [12]. Czarnecki and Schilder also supported this [6].

Primary endodontic and periodontal diseases have a clear path of treatment, however true combined lesions are complex to treat and have an unpredictable outcome. Cases in which endodontic treatment is performed before periodontal therapy show better outcome. Prognosis depends mostly on the extent and severity of periodontal destruction and effectiveness of the periodontal therapy.

Conclusion

Based on existing literature, the correct diagnosis and treatment of periodontal -endodntic lesions is incumbent on thorough understanding of the different routes of communication between pulp and periodontium. This knowledge is a key factor in achieving successful management of periodontal -endodontic lesions in the clinic.

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