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Endodontic-Periodontic Relationship - A Short Review

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Abstract

The relationship between periodontal and endodontic disease has been a matter of speculation for many years. The endodontium and periodontium influence each other during health, function and disease. This review gives a fair knowledge about several aspects of endo-perio relationship. Their exist three main pathways of communication between the pulp and periodontium. They are the apical foramen, the accessory canals and the dentinal tubules. The apical foramen and the lateral canals come under vascular pathways whereas the dentinal tubule comes under the tubular pathway. The pulp and periodontal tissues are derived from highly vascular mesenchymal tissues of the tooth germ. The apical foramen and lateral canals maintain the vascular connection between these tissues throughout the tooth development.

Key words: *endodontics, periodontics, lesion, relation, pathways*

Introduction

Simring and Goldberg in 1964 first discovered the relationship in between the periodontium and the pulp(1). The periodontium and pulp have embryonic, anatomic and functional interrelationship. Ectomesenchymal cells give rise to dental papilla and dental follicle.They in turn differentiate into periodontium and pulp respectively. This development during the embryonic stage results in anatomical connections,which remain throughout the life.

It has been found that microbiological similarities exist between infected root canals and advanced periodontitis(2,3,4,5).Similarities in the composition of cellular infiltrates also suggest the existence of communication between the pulp and periodontal tissues. A study by Brenda Gomes showing a similarity between the microbiota of periapical lesion and periodontal pocket , before and after CMP, suggests there may be a pathway of infection between the pulp and periodontium(6).

Speaking of these pathways further, apical foramen is the major pathway of communication. Although periodontal disease has damaging effect on the pulp tissue, total disintegration of the pulp will take place only when the bacterial plaque involves the apical foramen, compromising and further arresting the vascular supply. After pulpal necrosis, the bacterial products like enzymes, metabolites, antigens etc. reach the periodontium via the apical foramen, initiating an inflammatory response there.

The frequency of the lateral canals is 17% in the apical third, 1.6% in coronal and 8.8% in the body of the root(7).Bender et al. stated that periodontal endodontic diseases are frequently seen in molars than in the anterior teeth because more number of accessory canals are present in the molars. In first molars, the percentage of lateral canals in the furcation is 46%(8) whereas it is 50 to 60% in a multirooted teeth(9).The radiographic indications of the presence of lateral canals before obturation are presence of a localized thickening

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of periodontal ligament on the lateral root surface or a frank lateral lesion.

The dentinal tubules contain the odontoblastic process that extends from the odontoblast at the pulpal dentin border to the dentinoenamel junction or the cement-dentinal junction. Passage of microorganisms between the pulp and periodontal tissues is seen through these tubules, when the cementum is denuded and the dentinal tubules are exposed.

Non-physiological pathways of communication involve Iatrogenic perforations and vertical root fractures.

The etiopathogenesis of endo-perio lesion can be widely studied under three groups i.e. the anatomic considerations which have been discussed above, the contributing factors like poor endodontic treatment, poor restoration, trauma, resorption, perforation and developmental malformations. The third group is the etiologic factors live pathogens and non living agents. The live pathogens include bacteria, fungi and viruses.

Rupf et al detected Actinobacillus actinomycetemcomitans, Tannerella forsythus, Eikenella corrodens, Fusobacterium nucleatum, Porphyromonas gingivalis, Prevotella intermedia and Treponema denticola in all endodontic samples as well as in teeth with chronic apical periodontitis and chronic periodontitis[10]. A study by Didilescu et al suggested that Fusobacterium nucleatum, Parvimonas micra and Capnocytophaga sputigena may play a role in the pathogenesis of endo-periodontal lesions[11].Spirochetes are another type of microorganisms associated with both endodontic and periodontal diseases. Recent studies demonstrated that the spirochete species most frequently found in root canals are Treponema denticola and Treponema maltophilum[12]. Treponema denticola posess an array of virulence factors associated with periodontal disease and may also participate in the pathogenesis of periradicular disease. Treponema maltophilum is frequently associated with rapidly progressing forms of periodontitis.

Coming to Fungi, yeast colonization associated with radicular pathosis has been seen in untreated root caries, dentinal tubules, failing root canal treatments, apices of teeth with asymptomatic apical periodontitis and in periapical tissues.Reduction in specific strains of bacteria in Root Canal treatment creates a low nutrient environment which may allow fungal overgrowth. Fungi may gain access from oral cavity during treatment as a result of poor asepsis. Colonization of Candida albicans and other species like Candida glabrata, Candida guillermondii, Candida incospicia and Rodotorula mucilaginosa were detected in root canal, which can penetrate into dentinal tubules. It has been found that approximately 20% of chronic periodontitis patients harbor subgingival yeast, mostly Candida albicans.

Gingival herpes virus has been associated with increased occurrence of subgingival Porphyromonas gingivalis, Tannerella forsythus, Porphyromonas intermedia, Prevotella nigrescens, Treponema denticola and Actinomyces actinomycetem comitans . Hence viruses may play a role in promoting overgrowth of pathogenic periodontal bacteria. Cytomegalo and Epstein Barr viruses play a major role in pathogenesis of symptomatic periapical lesion by the production of cytokines and chemokines thereby leading to tissue destruction[13].

The non living etiologic agents are extrinsic and intrinsic. The extrinsic agents are the foreign bodies which are associated with inflammation of the periradicular tissues. They may be dentin and cementum chips, amalgam, root canal filling materials, cellulose fibers from absorbent paper points, gingival retraction cords, leguminous foods and calculus like deposits. The clinical reaction may be acute or chronic. The intrinsic agents are cholesterol, Russell bodies, Rushton hyaline bodies, epithelium and Charcot – Leyden Crystals (CLC).The Russell bodies are found in inflammatory pulpal tissue of carious primary teeth. The Rushton Hyaline bodies are found in some odontogenic cysts. The epithelial rests of malassez found along the lateral and apical periodontal ligament stimulate the epithelium to proliferate and line the lesion.The presence of CLC is seen within a periapical lesion that failed to resolve after orthograde root canal treatment.

Classification

By SIMON,GLICK AND FRANK IN 1972[14]:

1. Primary endodontic disease
2. Primary periodontic disease

3. Primary endodontic disease with secondary periodontal involvement

4. Primary periodontal disease with secondary endodontic involvement

5. True combined lesions

TORABINEJAD AND TROPE IN 1996:

1. endodontic origin,
2. periodontal origin,
3. combined endo-perio lesion,
4. separate endodontic and periodontal lesions,
5. lesions with communication,
6. lesions with no communication

BY THE WORLD WORKSHOP(1999)

1. endodontic-periodontal lesion,
2. periodontal-endodontic lesion,
3. combined lesion.

The classification by Simon et al[14] is the most accepted and important classification till today.First comes the primary endodontic disease. It is caused by caries, restorative manipulations, traumatic injury. The pulp sensibility tests show completely absent of signs (necrosis, except multirooted teeth), may be aware of minimal discomfort. Probing shows normal sulcus or narrow drainage to the sulcus (fistulas resulting from pulpal disease) .Radiolucency at apical, lateral and furcation area is seen. Prognosis is good after RCT. The sinus tract extending into the gingival sulcus or furcation region resolves at an early stage, if the pulp which is necrosed is removed and the root canals are well sealed[15].

The primary periodontal disease shows wide pockets on probing that do not necessarily extend towards the apex. The clinical findings include attachment loss, gingival bleeding, tooth mobility, calculus and plaque. Radiographs show horizontal or vertical bone defect and furcation lesion. Pulp tests are normal. Pulp is vital in primary periodontal disease .Treatment wholly depends on periodontal therapy.

The primary endodontic lesion if not treated will lead to secondary periodontal disease. This is primary endodontic lesion with secondary periodontal involvement. The clinical findings include plaque and calculus at the gingival margin due to drainage. The adjacent teeth are not necessarily involved.The pulp tests show absence of response.A solitary wider pocket is seen on probing extending towards the apex. The radiographs show widening of the periodontal ligament space extending from apical to cervical region. Angular defects can be seen. The prognosis depends on the periodontal therapy, assuming the endodontic procedures are adequate[16]. With endodontic therapy alone, only part of the lesion may heal which indicates the presence of secondary periodontic involvement.

A severe primary periodontal lesion if untreated eventually leads to secondary endodontic involvement. This is primary periodontal lesion with secondary endodontic involvement. The clinical findings include history of extensive periodontal disease. Initially there is pain from inflamed pulp. On progression, pulp loses its vitality. The pulp tests are abnormal or absent. On probing , wide pocket is seen, sometimes extending apically. The radiographic findings show angular bone loss from cervical region towards apex.The prognosis depends on the periodontal therapy once the endodontic therapy has been solved.

Then come the true combined lesions which show history of extensive periodontitis. Pulp tests are absent. Probing reveals wide and conical pocket.Radiographic findings show extensive bony radiolucencies which may/may not communicate. Findings of true combined lesions may be similar to that of vertical fractured tooth.

The effect of periodontal inflammation on the pulp is controversial and conflicting studies are present[17,18,19,20]. It has been suggested that periodontal disease has no effect on the pulp, at least until it involves the apex[18]. The effect of periodontal lesions on the pulp can result in atrophic and degenerative changes like dystrophic mineralization, fibrosis, formation of reparative dentin, inflammation and resorption[21,22].

The atrophic changes result in decrease in the cells' size, more collagen depositions, impaired nutrition to the pulp cells. Hence they slowly degenerate.The death of the cell is so gradual that morphologic evidence

sometimes appears to be lacking.Disruption of blood flow results in localized areas of coagulation necrosis in pulp.These areas are eventually walled off from the rest of the healthy pulp tissue by collagen and dystrophic mineralization. With slowly progressing periodontal disease, before pulpal irritation occurs, deposition of cementum is seen obliterating the lateral canals. This may explain why, not all periodontally involved teeth demonstrate pulpal atrophy and canal narrowing. Sometimes, because of the mobility of the periodontally involved teeth, pressure atrophy may also occur.

The pathogenic bacteria and inflammatory products of the periodontal disease infect the pulp via the apical foramen and lateral canals and evoke an inflammatory reaction in the pulp. This is called Retrograde pulpitis. Coming to inflammatory changes, when periodontal disease approaches the apex, the inflammatory products act on the periodontal ligament and the surrounding alveolar bone. Localized apical granuloma is a commonly encountered periodontal lesion produced by the pulp disease. It is because of transport of bacterial products via the root apex, with the formation of vascular granulation tissue. Eventually, resorption of the alveolar bone and sometimes the root itself may occur.

Resorption is seen on sides of the roots adjacent to granulation tissue.When the periodontal lesions are deep, resorption may also be found within the root canals, often opposite lateral canals, and at the apical foramen.This is called peripheral inflammatory root resorption (PIRR).

The effects of endodontic infection on periodontium is seen in many findings of intrapulpal infection tending to promote epithelial down-growth along a denuded dentin surface. Also, in experimentally created periodontal defects, the epithelium was 20% more around infected teeth than non infected teeth. Non infected teeth had more of connective tissue covering than infected teeth. Hence, it is important to treat the pulpal infections first, before undertaking periodontal procedures.

The periodontal treatment procedures also have some effect on pulp.Deep root planning, improper or overusage of the instruments and periodontal injury or wounding may speed up the pulpal inflammation and initiate the interrelated disease process[23].Improper root planning and scaling results in removal of cementum and superficial dentin getting exposed. It leads to microbial colonization and bacterial invasion resulting in inflammatory lesions in the pulp and dentinal hypersensitivity.

Conclusion

Endodontic –periodontic disease requires multidisciplinary involvement. The signs and symptoms have to be evaluated thoroughly and based on that, treatment protocol has to be planned. Improper management may result in failure in healing of either endodontic or periodontic lesion or both the lesions.

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