CURRENT CONCEPTS ON THE RELATIONSHIP BETWEEN PULPAL AND PERIODONTAL DISEASES

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REZUMAT
Leziunile combinate endo-parodontale reprezintă o adevărat provocare în practica medicala curentă, atât pentru specialistul în endodontie, cât și pentru cel în parodontologie. Existenta simultană a problemelor pulpare și parodontale la asezați dintr-o problema majoră complică diagnosticul și tratamentul planificat pentru dintel respectiv. Acest tip de patologie reprezintă o situație specială datorită complexității anatomiei și fiziologiei pulpare și parodontale. Lucrarea prezintă că trece propune să revizuiască informațiile din literatura de specialitate despre etiologie, căi de diseminare și aspecte clinice în leziunile endo-parodontale. Prognosticul rezervat al acestor afectiuni recomandă o abordare interdisciplinară, cu o bună colaborare între specialiștii în endodontie, parodontologie și microbiologie.

Cuvinte cheie: infecție pulpară, infecție parodontală, migrare bacteriană

ABSTRACT
Combined endodontic-periodontal lesions represent a real challenge in the daily practice, both for the endodontist and periodontologist. The simultaneous existence in the same tooth of pulpal problems and inflammatory periodontal disease can complicate the diagnosis and treatment of the affected tooth. This type of pathology represents a special situation in dentistry due to the complexity of anatomy and physiology of the pulp and periodontium. In this review, current topics relating to etiology, pathways of dissemination and clinical concepts in combined endo-periodontal lesions are summarized. The poor prognosis of these afflictions recommends an interdisciplinary approach with a good collaboration between specialists in endodontology, periodontology and microbiology.

Key Words: pulp infection, periodontal infection, bacterial migration

INTRODUCTION
The dental pulp is closely connected with the periodontal ligament through the apical foramina, accessory canals and dentinal tubules. Due to this relationship, pulp diseases may influence periodontal health and vice versa, and the periodontal infection may affect the pulp integrity. It is estimated that pulpal and periodontal problems are responsible for more than 50% of tooth mortality.¹

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Simon et al. (1972) have classified the lesions based on the primary source of infection, as follows:²
1. Primary endodontic lesions;
2. Primary endodontic lesions with secondary periodontal involvement;
3. Primary periodontal lesions;
4. Primary periodontal lesions with secondary endodontic involvement;
5. True combined lesions.

In addition, Belk and Gutmann (1990) proposed a sixth group of lesions - concomitant pulpal and periodontal lesions.³

ETIOLOGY
It has been demonstrated that the primary etiologic agent in periodontitis is bacterial plaque adhering to the teeth and other hard surfaces in the oral cavity. The pulp may be affected by the invasion of bacteria or their toxins, or by a direct trauma during the restorative therapy.⁴,⁵

Several factors make it difficult to establish a correct etiology of the combined endo-periodontal lesions.
First, the pulpal pain may occur either in a primary endodontic lesion or in a primary periodontal lesion with secondary endodontic involvement. In the last situation, the periodontal symptoms may be masked by the pulpal symptoms. Second, the tests used for the diagnosis of endo-periodontal lesions, such as vitality tests and radiographic examinations, may lead to an incorrect or incomplete diagnosis.

MICROBIOLOGY

The literature contains few descriptions of the periodontal-endodontic lesion microbiology. The profiles of the periodontal pathogens in simultaneous pulpal and periodontal disease associated with the same tooth, were investigated by Rupf et al.6 The periodontal pathogens displayed comparable profiles in pulpo-periapical lesions, except in progressive adult periodontitis. Kobayashi et al. concluded that the periodontal pocket in teeth with advanced periodontitis may be a possible source of root canal infections.7

The microbiology of endodontic infections

Root canal infections are mixed and semispecific infections with a great predominance of obligate and facultative anaerobic bacteria. However, only a small group is commonly isolated from infected dental pulp cavities. The relative proportion of strict anaerobic bacteria to facultative aerobic bacteria increases with the time, and so does the total number of bacteria.8-12 According to recent reports, it is presumed that over 700 bacterial species inhabit the oral cavity and more than half of these cannot be cultivated.13,14 Specific combinations of bacteria can be found in the root canal and they can contribute to ecological shifts of the flora by different mechanisms of interaction.15 Bacteria may not only reside in the main canal, but they also invade the dentinal tubules, making them act as a reservoir for future dental and systemic infections.16,17 Porphyromonas gingivalis, Tannerella forsythensis and Fusobacterium nucleatum are associated with extraradicular biofilm formation and refractory periapical chronic periodontitis.18 The presence of T. forsythensis, P. gingivalis and Treponema denticola was demonstrated in root canal samples collected from patients with carious lesions, necrotic pulp and radiographic evidence of periradicular bone loss.19 T. denticola was highly associated with symptomatic endodontic infections and periradicular bone resorption, whereas Enterococcus faecalis was associated with asymptomatic chronic apical periodontitis and secondary endodontic infections in endodontic failures.20 Black-pigmented bacteria have been isolated from acute abscesses of dental origin, suggesting an active role in the pathogenesis of acute symptoms.21-23

The microbiology of periodontal infections

The periodontal infection is caused by microorganisms colonizing the tooth surface at supra or subgingival level. Living, dying and dead bacteria and their metabolic byproducts form a resistant biofilm by attaching to the tooth, to the epithelial cells and to the underlying exposed tissues.24 The onset of the infectious periodontal disease depends on the simultaneous occurrence of a number of factors for initiation and progression, such as the virulence of periodontal pathogens, the local environment and the host defense mechanisms. It is already documented that approximately 15 host genes are involved in the onset and progression of the periodontal disease in humans.25 Different studies used various criteria such as the association between the pathogens and the disease, the elimination or decrease of species and the parallel remission of disease due to specific treatment, the host response, the virulence factors, associated different bacteria with different periodontal pathoses.26-30 Actinobacillus actinomyctetemcomitans, P. gingivalis, T. forsythensis, Prevotella intermedia, F. nucleatum, Campylobacter rectus, Eikenella corrodens, Peptostreptococcus micros and Eubacterium are the best studied periodontal pathogens.31 However, their proportion in the dental biofilm and their virulence may differ among individuals with periodontal disease.32

PATHWAYS OF DISSEMINATION

Both endodontic and periodontal diseases are caused by mixed anaerobic infections. Bacteria and their inflammatory by-products can penetrate through the pathways connecting the periodontium and the pulp. Kerns and Glickman described six groups of normal and pathological pathways:33 (Fig. 1)

1. The apical foramen may be the most significant direct route of communication between the pulp and the periodontium. The apical foramina represent also a portal of entry towards the pulp from deep periodontal pockets. Pulp infection extends into the periapical tissues causing a local inflammatory response, followed by alveolar bone and sometimes by focal root resorptions.34

2. Lateral and accessory canals appear mainly in the apical area and in the furcation area of molars.
The normal pathways of communication between the endodontium and the periodontium (1 - the apical foramen, 2, 3 - lateral and accessory canals, 4 - dentinal tubules). They have been suggested to be direct pathways between the pulp and the periodontium and contain connective tissue and blood vessels that connect the circulatory systems of the two tissues. The presence of patent accessory canals is a potential pathway for the spread of bacteria and toxic substances from the pulp, resulting in an inflammatory process within the periodontal ligament.

3. Dentinal tubules contain odontoblastic processes that extend from the odontoblasts to the dentin-enamel junction or the dentin-cementum junction. Fluid may flow through patent dentinal tubules. In absence of an intact enamel or cementum covering, the pulp may be considered exposed to the oral environment. The number of dentinal tubules per square millimetre varies from 8,000 to 57,000 and the diameter ranges from 0.5 μm in the peripheral dentin to 3-4 μm near the pulp. The permeability of root dentin depends upon dentin thickness, the number of tubules/mm², and the diameter and patency of the tubules.

4. Palatogingival grooves are developmental anomalies of the maxillary incisor teeth, with lateral incisors more often affected than centrals. Grooves usually begin in the central fossa, cross the cingulum and extent apically to vary distances. Sometimes, a radicular groove is located on the buccal aspect of maxillary central incisors.

5. Perforations of the roots create a communication between the root canal system and the periodontal ligament. This usually occurs as a result of iatrogenic factors (overinstrumentation), internal or external resorption (Figs. 3, 4) or caries penetrating through the floor of the pulp chamber. The closer the perforation is to the gingival sulcus / periodontal pocket, particularly into the coronal third of the root or the furcation region, the greater is the probability of a periodontal lesion by migration towards apical of the gingival epithelium.
CLINICAL CONCEPTS

Primary endodontic lesions with secondary periodontal involvement

A causal relationship between root canal infection and periodontal disease requires a patent route to the periodontium, an infected root canal system and sufficient virulence of the pulpal bacteria to promote marginal periodontitis. In the absence of treatment, the pulpal infection will continue, leading to destruction of the periapical alveolar bone and progressing into the area of furcation. Due to drainage through the gingival sulcus and accumulation of plaque in the purulent pocket, periodontal disease progresses and the further apical migration of the attachment may occur. Extensive pulpal infection spreading through patent lateral canals or dentinal tubules in the absence of an intact cementum may alter the periodontium, too. Radiographs show evidence of both pulpal and periodontal disease.

A typical situation: the upper first molar displays signs of periodontal disease with loss of attachment of mesio-buccal root. Radiographic examination reveals the presence of periapical and periodontal radiolucency, and a complete obturation of three canals. Usually, in this case, the symptoms are misinterpreted and the diagnosis is of localized periodontitis; consequently, the applied treatment consists of scaling and root-planning, associated with local antiseptics and irrigations with chlorhexidine. Healing does not occur because of the endodontic origin of this condition, due most probably to the second mesio-buccal canal, very often ignored during the endodontic therapy. (Fig. 5)
Therefore, the correct diagnosis is of primary endodontic lesion with secondary periodontal involvement.

Primary endodontic lesions with secondary periodontal involvement may also be the results of iatrogenic factors, such as root perforations, root fracture or misplacement of posts during coronal restorations. Primary endodontic lesions with secondary periodontal involvement may also be the results of iatrogenic factors, such as root perforations, root fracture or misplacement of posts during coronal restorations. Primary endodontic lesions with secondary periodontal involvement may also be the results of iatrogenic factors, such as root perforations, root fracture or misplacement of posts during coronal restorations. Primary endodontic lesions with secondary periodontal involvement may also be the results of iatrogenic factors, such as root perforations, root fracture or misplacement of posts during coronal restorations.

**Primary periodontal lesions with secondary endodontic involvement**

The effect of periodontitis on the healthy pulp has been reported in several studies. Periodontal disease can affect the pulp through dentinal tubules, lateral canals or apical foramina. Lateral canals and dentinal tubules may be also infected through a temporary opening to the oral environment during surgical flap procedures. When the pulp is affected, the patient reports acute pain and clinical signs of pulpal inflammation. Radiographically, these lesions may be indistinguishable from the primary endodontic lesions with secondary periodontal involvement. (Fig. 6)

**True combined lesions**

Pulpal and periodontal diseases may occur independently and concomitantly in and around the same tooth. A necrotic pulp or a failing endodontic treatment, plaque, calculus and periodontitis are present in varying degrees. True combined endo-periodontal lesions present radiographic evidence of bone loss surrounding a substantial area of the root. Periodontal probing reveals a pocket extending to the apical region of the tooth. However, a fully documented diagnosis of a true combined lesion is hard to establish.

**Concomitant pulpal and periodontal lesions**

In this case, periodontal and pulp diseases coexist, but with different etiology. Radiographically, the endodontic lesion and a noncommunicating periodontal pocket are observed.

**CONCLUSIONS**

The tooth anatomy and the etiology of endodontic-periodontal lesions offers 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.

**REFERENCES**


**Figure 6.** Primary periodontal lesions with secondary endodontic involvement lesion. Initial x-ray of the defect (A) and initial probing of the PD (B). Intra-operative aspect of the bone (C). Six months after x-ray (D) with the root canal treatment completed and the bone defect filled.


