

## POLYMICROBIAL ORIGIN OF ENDOPERIODONTAL LESIONS: LITERATURE REVIEW

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**Abstract:** Endoperiodontal injury is a common condition in dental offices that, due to the complexity of clinical aspects, can induce simulations of one disease and not another, making diagnosis difficult. Faced with changes that simultaneously affect endo and periodontal tissues, the dentist must have knowledge about etiological factors, dental and orofacial anatomy, pulp and periodontal diseases, predominant pathogens, communication routes, classifications of primary diseases, primary with secondary involvement or combined, assessment techniques to – through anamnesis and intraoral and complementary exams – reach the correct diagnosis that facilitates the best outcome for the patient, whether through a therapeutic or surgical approach. The apical foramen, lateral or accessory canals, dentinal tubules devoid of cementum (due to scaling or root planning or due to congenital absence) are pathways for fluid communication to occur between the pulp and periodontium, which may contribute, for example, to the development of dental diseases. primary origin. The endodontic and periodontal relationships discussed, since 1948, by Johnson and Orban were later proven by research carried out by Seltzer and his team, when changes in periodontal tissues were found after induction of a certain endodontic condition. The present study aims to review the literature regarding the polymicrobial origin of these lesions, considering that the lack of knowledge about the respective agents contributes to mistaken diagnoses and decision-making that can lead the dentist to respond legally, even if their responsibility as an endodontist or periodontist is half.

This literature review examined articles published in Portuguese and English on the platforms PubMed, SciELO, ResearchGate, Google Scholar and LILACS, between 2000 and 2023, using the DeCS descriptors

in Portuguese and English “regenerative endodontics”, “periapical tissue”, “dental pulp diseases”, “dental pulp necrosis”, “regenerative endodontics”, “periapical tissue”, “dental pulp diseases”, “dental pulp necrosis” articulated with the Boolean operators “AND”, “OR” and “NOT”. Exclusion criteria include articles published outside the established period, theses, dissertations, monographs and animal studies.

**Keywords:** regenerative endodontics; periapical tissue; dental pulp diseases; necrosis of the dental pulp.

## INTRODUCTION

The endodontic and periodontal relationships discussed since 1948, by Johnson and Orban, are justified by the proximity between pulp and periodontium, later proven by research by Seltzer, when injuries to periodontal tissues were observed after induction of endodontic damage (FACHIN et al. 2001).

Endoperiodontal injury is an inflammatory process that affects the pulp and periodontal tissues, common in dental offices and which, due to the complexity of clinical aspects and advanced stages, can induce periodontal disease to the detriment of pulp disease (and vice versa) or simulate a primary endodontic and non-periodontal lesion (the opposite being true), making the diagnosis and prognosis of the case difficult. Due to the difficulty in establishing the differential diagnosis – based on anamnesis and the combination of clinical-visual exams (with pulp sensitivity test, percussion, palpation, probing) with imaging exams to complete the clinical information – it is important to move forward with an approach interdisciplinary approach to detect the origin of the problem, choose the best approach and eliminate the risk of recurrence (FACHIN, 2001; KIRCHHOFF, 2013; GONÇALVES, 2020).

Considering that more than 50% of tooth losses derive from endodontic and periodontal imbalances and that a mistaken assessment of this pathological context contributes to mistaken decision-making and poor prognoses, the present study aims to review the literature regarding polymicrobial origin and present a list of signs that contribute to differential diagnoses (SINGH, 2011; KERNS, 2011; GONÇALVES, 2020; TRAVASSOS, 2022).

## METHODS

This literature review examined articles published in Portuguese and English on the platforms PubMed, SciELO, ResearchGate, Google Scholar and LILACS, between 2000 and 2023, using the DeCS descriptors in Portuguese and English “regenerative endodontics”, “periapical tissue”, “dental pulp diseases”, “dental pulp necrosis”, “regenerative endodontics”, “periapical tissue”, “dental pulp diseases”, “dental pulp necrosis” articulated with the Boolean operators “AND”, “OR” and “NOT”. Exclusion criteria include articles published outside the established period, theses, dissertations, monographs and animal studies.

## RESULTS AND DISCUSSION

The intimate histological relationship between the pulp and periodontal tissues begins from the embryonic stage, with odontogenesis, as both tissues derive from the ectomesenchyme<sup>1</sup> – a type of mesenchyme originating from neural crest cells located under the oral epithelium – and which gives rise to tissues: pulpal, from the dental papilla, and periodontal, from the dental follicle (BATH-BALOGH, 2012).

After a continuous process of tooth

development, distinct structures are formed with anatomical proximity and in continuous biological integration. Perhaps, therefore, in endoperiodontal lesions there is facilitated dissemination of various pathogens and their respective metabolic products between the pulp and periodontal tissues. However, endoperium lesions do not only develop from anatomical factors, they also originate from pathological factors. Toxins and necrotic substances from microorganisms that spread throughout the root thirds – destroy both pulp and periodontal tissue – can circulate freely through lateral canals, accessory canals, apical foramen and dentinal tubules due to the absence of root cementum due to excessive scaling and root planning, root resorption triggered by different agents, perforations during endodontic treatments or fractures caused by trauma (FACHIN, 2001; COHEN, 2011; LACEVIC et al, 2015, PENONI, 2016).

Although the endodontic microbiota is less complex than the periodontal one, the literature indicates that in endoperium lesions there is a wide variety of microorganisms that inhabit both tissues, some of which are difficult to control. With the purpose of assisting the dentist, general practitioner or specialist, and in order to achieve a good prognosis in favor of the patient, several studies focus on the study of pathogens associated with the classifications of these injuries (FAGUNDES et al, 2007; ROTSTEIN, 2017).

The “Primary endodontic lesions” are conditions that only require endodontic treatment for remission of the lesion, which may or may not be symptomatic in the patient. Its etiology may be due to biological (microbial), chemical (adhesive system of restorative procedures) or physical (trauma) agents. The inflammatory process

1. When dental epithelium, basement membrane and proteins interact with undifferentiated ectomesenchymal cells of the dental papilla, differentiation occurs into odontoblasts, cells that deposit dentin, and contribute to the conversion of the dental papilla into dental pulp. But, also when periodontal tissues originate from fibrocellular tissues, cementum, periodontal ligaments and alveolar bone also derive from ectomesenchyme.

that migrates from the necrotic pulp to the periodontium can lead to the formation of a drainable fistula or cause bone loss proven by periapical radiographs. Clinical signs may be sensitivity to pressure, sensitivity to percussion, pain, tooth mobility and gingival edema with the possibility of abscess (which is why this condition can be confused with periodontal abscess). The microorganisms present in these primary endodontic lesions are: *Enterococcus faecalis*, *Eubacterium brachy*, *Filifactor alocis*, *Fretibacterium fastidiosum*, *Mogibacterium timidum*, *Parvimonas micra*, *Parvimonas micra*, *Streptococcus constellatus*, *Tannerella forsythia*. *Anaeróbios estrictos*: *Campylobacter rectus*, *Lactobacillus acidophilus*, *Porphyromonas gingivalis*, *Propionibacterium acnes*, *Veillonella parvula*, *Slackia exigua*, *Anaeróbios facultativos*: *Bactérias microaerófilas*: *Aggregatibacter actinomycetemcomitans* (COHEN, 2011; GAMBIN, 2018; POURHAJIBAGHER, 2018).

Specifically, “primary endodontic lesions with secondary periodontal involvement” are conditions that require combined treatment of the endodontic lesion and, subsequently, the periodontal lesion. This type of injury can occur due to the lack of treatment for primary endodontic inflammation that progresses to the destruction of periodontal tissues, as different degrees of concentration and potency of agents trapped internally in necrotic spaces can potentiate different degrees of reactions. If, on the one hand, in the hard tissues there is necrotic pulp and canals, on the other, in the soft tissue region there may be an accumulation of subgingival calculus in the periodontal pockets and continuous drainage from an exteriorized fistula that communicates through the mucosa with the cavity. oral. Fachin stated in his article, published in 2001, that irritating agents in root canals demonstrate the potential to cause – after dissemination through lateral

canals, accessories and apical foramen – pathological changes in the periodontal region. Figueiredo, in 2000, further advises that endoperiodontal lesions, in addition to having a more complex root canal microbiota, can also be more pathogenic than those that are exclusively endodontic. The predominant microorganisms in this classification are: *Enterococcus faecalis*, *Eubacterium brachy*, *Filifactor alocis*, *Fretibacterium fastidiosum*, *Mogibacterium timidum*, *Parvimonas micra*, *Streptococcus constellatus*, *Tannerella forsythia*; (FIGUEIREDO, 2000; FACHIN, 2001; COHEN, 2011; GAMBIN, 2018; POURHAJIBAGHER, 2018).

The “Primary periodontal lesions” are conditions that require periodontal treatment for remission of the lesion because they present inflammation, calculus accumulation, more widespread bone and attachment loss, tooth mobility, as well as periodontal abscess. However, its solution depends on the extent of the destruction, the host’s ability to react and its adherence to treatment. The classified microorganisms are: *Eikenella corrodens*, *Fusobacterium nucleatum*, *Fusobacterium nucleatum*, *Parvimonas micra*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Treponema denticola*, (COHEN, 2011; GAMBIN, 2018; POURHAJIBAGHER, 2018).

“Primary periodontal lesions with secondary endodontic involvement” are conditions that require combined treatment of the periodontal lesion and, subsequently, the endodontic lesion of the respective tooth element. Generally, treatment focuses on deep pockets that generate severe pain and discomfort in the patient, and secondarily on interrupting microbial migration to the pulp, whether through the foramen, lateral canals or dentinal tubules lacking protection from the root cementum. Research points to the presence of the following microorganisms: *Bacteroidace-*

*ae sp.*, *Fretibacterium fastidiosum*. (COHEN, 2011; GAMBIN, 2018; POURHAJIBAGHER, 2018).

The “Concomitant pulp and periodontal lesions” occur simultaneously, with different etiological factors interfering in this condition in which one of the pathologies is independent of the other. Requiring concomitant treatment of pathologies, the prognosis may remain unfavorable even with the elimination or prevention of etiological factors. We report the following microorganisms: *Actinomyces odontolyticus*, *Aggregatibacter aphrophilus*, *Campylobacter concisus*, *Campylobacter rectus*, *Corynebacterium matruchotii*, *Enterobacter asburiae*, *Fusobacterium canifelinum*, *Haemophilus parainfluenzae*, *Mogibacterium timidum*, *Neisseria elongata*, *Neisseria bacilliformis*, *Peptostreptococcus stomatitis*, *Porphyromonas endodontalis*, *Tannerella forsythia*, *Veillonella rogosae* (COHEN, 2011; GAMBIN, 2018; POURHAJIBAGHER, 2018).

Given the diverse and complex list of microorganisms, didactically subdivided by type of endoperiodontal lesion, the dentist must also know the stages of these lesions, given that the failure to eliminate pathogens from root canals or periodontal pockets or, even, the virulence of certain microorganisms<sup>2</sup> can contribute to treatment failure (DUQUE et al., 2018; POURHAJIBAGHER, 2018).

When faced with changes that simultaneously affect pulpal and periodontal tissues of the same dental element, the dentist must: i) know the patient's past clinical

2. *Aggregatibacter actinomycetemcomitans*, *Campylobacter rectus*, *Fusobacterium nucleatum*, *Lactobacillus acidophilus*, *Porphyromonas gingivalis*, *Propionibacterium acnes*, *Slackia exigua* and *Veillonella parvula*.

3. Auschill explains that 90% of restorations do not present failures as long as the cavity preparation has respected the criteria: contour, resistance (to chewing forces), retention (to avoid displacement of the restoration), convenience (of instruments and methods), removal of unsupported structures, finishing and cleaning (to receive restorative material) (AUSCHILL, 2009). However, in 2021, Silva Neto demonstrated that 1/3 of cases may present postoperative sensitivity. Remembering Auschill's lessons, depending on the size of the cavity (extension and depth) the risk of sensitivity can quadruple. This means that during the preparation of extensive and deep cavities that will have composite resin restorations proportional to the damage, there must be subsumption of protocols that preserve the dentinopulpal complex, such as the use of new rotating instruments, cooling the tooth during removal of composites with a high-speed pen, maintaining cavity humidity during preparation, respecting the photopolymerization and use of universal adhesive, eliminating the need for acid etching of deep dentin (CUNHA, 2007; MUÑOZ, 2013; DE AQUINO, 2021).

history, trying to verify location, duration or reports of pain; ii) evaluate the presence of carious lesions, extensive or unsatisfactory restorations<sup>3</sup>, bacterial plaque in the cervical region, signs of trauma, swollen periodontium, deep periodontal pockets, fistulas or swelling; iii) perform periodontal examination; iv) test pulp sensitivity, percussion and palpation; v) request imaging exams to observe periradicular radiolucency, bone losses (horizontal, vertical, closer or further from the root apex), bone defects such as fenestrations or dehiscences, resorptions or complete clinical information; vi) have in-depth scientific knowledge about etiological factors, dental and orofacial anatomy, pulp and periodontal diseases, predominant pathogens, communication routes, classifications of primary diseases, primary with secondary involvement or combined in order to achieve the correct diagnosis with a positive outcome for the patient by therapeutic or surgical approach. Apical foramen, lateral or accessory canals, dentinal tubules devoid of cementum (due to scaling, root planning or congenital absence) are pathways that allow the communication of fluids and microorganisms between the pulp and periodontium, and may cooperate with the development of initial lesions. (FACHIN et al. 2001; ROTSTEIN and SIMON, 2004; GRECCA et al. 2012; ROSTEIN, 2004; LINDHE, 2010; KIRCHHOFF, 2013; GONÇALVES, 2020; TRAVASSOS, 2022). KIRCHHOFF, 2013).

In the dental arch, according to Hargreaves,

Cohen and Askel, endoperiodontal lesions are more prevalent in multirrooted posterior elements because they have: i) a greater number of pathways – lateral and accessory canals, apical foramen, dentinal tubules devoid of root cementum protection – which they provide communication between endodontic and periodontal tissues when compared to the group of anterior teeth, as well as, ii) greater volume of permeable areas of cementum and dentin (ASKEL et al., 2014).

Knowing that bacteria expel toxins that reach the periodontal ligament space – destroying soft and hard tissues adjacent to the element – it is important to note that the prognosis of these injuries primarily depends on the triad intensity of the offending factor, the host's immunological response and duration of disease (which may involve, to a greater or lesser extent, bone and support structures), secondarily, also depends on knowledge about etiology, pathophysiology, planning and multidisciplinary treatment carried out by dental surgeons to eliminate or prevent recurrences generated by aggressive agents. The success of endodontic treatment can be noted when pain, edema, fistula, periapical bone radiolucency, symptoms on percussion and palpation are eliminated, but, equally, when during follow-up, to monitor the results obtained, endoperiodontal therapy is followed by good hygiene oral performed by patients instructed by oral health professionals (FIGUEIREDO, 2000; FACHIN, 2001; LINDHE, 2010; COHEN, 2011; POURHAJIBAGHER, 2018; GONÇALVES, 2020; TRAVASSOS, 2022).

Several experts, including Harrington, Bergenholtz and Hasselgren, advocate starting the approach with endodontic treatment with canal instrumentation, antimicrobial irrigation of the root canals, application of intracanal medication (biocompatible, capable of raising the pH and inducing the formation

of mineralized tissue – hydroxide calcium P.A.) and, in more complex cases, antibiotic prescription (amoxicillin associated with potassium clavulanate) so that the definitive filling can be performed in the next session. Periodontal treatment with scaling and root planning removes microbial layers from the root, cementum and superficial dentin, which may leave dentinal tubules exposed to continuous subgingival colonization, generally by gram-negative bacteria. Thus, for periodontal healing, considering the most modern periodontal treatments, one can opt for the application of enamel-derived matrix biomaterial (EMD) – with the aim of assisting in the recovery of hard and soft tissues, in addition to inducing tissue neof ormation. periodontal. (BERGENHOLTZ, 1997; FACHIN, 2001; GONÇALVES, 2020; REIS, 2022).

## CONCLUSION

The general dentist, endodontist or periodontist must identify the extent of the lesion, intensity of the offending factor, signs (presence of pain or insensitivity), location, radiographic aspects of bone loss, duration of the disease, etiological factors and communication routes in order to correctly diagnose and draw up the appropriate treatment plan for the specific case, mitigating the toxic/necrotic content of the internal and external environments of the dental element.

The multidisciplinary approach to endoperiodontal lesions can begin with endodontic treatment with canal instrumentation, irrigation of root canals, placement of intracanal medication so that the definitive filling can be performed in the subsequent session.

Periodontal treatment with scaling and root planning that removes microbial layers from the root, cementum and superficial dentin relies on the application of enamel-

derived matrix biomaterial (EMD) – to assist in the recovery of hard and soft tissues, in addition to inducing neoformation of periodontal tissues. However, according to Reis, in a recently published work, the new periodontal approach is far from being content with controlling periodontal disease or reestablishing the periodontium because, specifically, its focus seeks to reestablish the region of periodontal ligaments and form new cementum structures. and alveolar bone

(REIS, 2022).

Finally, the multidisciplinary intervention that seeks to guarantee “better opportunities” and “reduce costs” for the patient is inspired by a concept from Economic Theory that goes beyond merely economic value, as it observes other values. Early diagnosis of these injuries and multidisciplinary intervention can lead to greater treatment effectiveness, while reducing social costs for public health and, consequently, for the patients themselves.

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