MATHEWS

Research Article

Mathews Journal of Dentistry

Contemporary Aspects of Treatment of Endodonal-Periodontal Lesions

Petrovski Mihajlo*, Veljanovski Darko, Nikolovski Bruno, Mladenovski Marko, Kovacevska Ivona

Faculty of medical sciences, Goce Delcev University, Stip, Republic of N. Macedonia

ABSTRACT

Introduction: Infection in pulp tissue may lead to secondary infection on periodontal tissue. In contrary, severe periodontal disease may initiate or exacerbate inflammatory changes in pulp tissue. The main goal of this research is to present the modern concepts in the treatment of endo-periodontal lesions. **Material and method:** To fulfill the main purpose made adequate literature research on Pub Med for articles relevant to our topic was done. **Results:** Treatment access and prognosis depend primarily on the diagnosis of the specific endodontic and periodontal disease. The main factors to consider for decision-making are pulp vitality and the extent of the periodontal defect. After complete instrumentation, calcium hydroxide should be used as an intracanal medicament. It is an excellent medicament in general, because it is bactericidal, anti-inflammatory and proteolytic; it inhibits resorption; and it favors repair. It is especially effective in endo-perio cases because its temporary obturating action will inhibit periodontal contamination of the instrumented canals. **Conclusion:** Therefore, a primary endodontic lesion draining from attachment apparatus should be initially treated by an endodontic therapy. Primary periodontal disease should only be treated by periodontal therapy. In this case, the prognosis depends on the severity of the periodontal disease and the patient response.

Keywords: periodontium, dental pulp, treatment, endodontic treatment

INTRODUCTION

Communication between pulpal and periodontal tissues is possible because of structures such as apical foramina, lateral or accessory canals and dentinal tubules. Infection in pulp tissue may lead to secondary infection of periodontal tissue. In contrary, severe periodontal disease may initiate or exacerbate inflammatory changes in pulp tissue.

Diagnostic steps must include patient-reported dental history, visual inspection and the association with large restoration and anatomic anomalies such as palatal grooves, radiographic confirmation with tracing the sinus track, results of clinical findings including percussion and palpation, routine periodontal assessment for presence of mobility

Vol No: 08, Issue: 02

Received Date: May 07, 2024

Published Date: May 27, 2024

***Corresponding Author**

Petrovski Mihajlo

Faculty of medical sciences, Goce Delcev University, Stip, Republic of N. Macedonia;

Email: mihajlo.petrovski@ugd.edu.mk

Citation: Mihajlo P, et al. (2024). Contemporary Aspects of Treatment of Endodonal-Periodontal Lesions. Mathews J Dentistry. 8(2):49.

or probing depth and pulp vitality testing.[1] The most common clinical and radiographic features of the endodontic-periodontal lesions reported are periodontal radiolucency and deep periodontal pockets with a nonvital pulp status.[2]

According to this, the presurgical phase or the pre-treatment assessment includes establishing and verifying the nonvital status of the pulp, the extent and severity of the periodontal destruction, and therapeutic prognosis of the planned regenerative procedure. Also it must distinguish an endodontic-periodontal combined lesion from a tooth with a vertical root fracture. A localized deep probing depth with minimal tooth mobility might have a favorable clinical outcome after the regenerative procedure.

Endodontic-periodontal combined lesion is a true challenge from diagnostic and treatment aspect. Its management requires understanding of wound healing process involving both endodontic and periodontal complex.

The treatment strategies and the clinical outcome depend on the extent of the periodontal disease and assessment of the therapeutic prognosis, with the intended regenerative procedure, presence or absence of periapical radiolucency, tooth mobility, properly performed root canal treatment, and appropriate healing time.[2]

The treatment of endodontic-periodontal combined lesions requires both endodontic therapy and periodontal regenerative procedure.

The main goal of this research is to present the modern concepts in the treatment of endo-periodontal lesions.

To fulfill the main purpose made adequate literature research on Pub Med for articles relevant to our topic was done. Articles published in the last two decades (2004-2024) were used, and the type of articles were Clinical Trial, Journal Article, Randomized Controlled Trial, Review, Comparative study and English was chosen as the language and humans as the species.

CONTEMPORARY ASPECTS OF TREATMENT OF EDNODONAL-PERIODONTAL LESIONS

The relationship between periodontal and pulpal disease are based on their common embryological development, primarily because the pulp and the periodontium are derived from a common mesodermal source. The developing tooth bud pinches off a portion of mesoderm that becomes pulp, while the remaining mesoderm develops into periodontium. In the course of root development, strands of mesodermal tissue may get trapped and later become lateral and accessory canals.[3] These offshoots also may result from dentin formation around existing blood vessels or a loss of continuity of Hertwig's root sheath during dentin formation. In time, most of these communications are sealed by cementum or secondary dentin, leaving the pulp basically dependent on the apical foramen for metabolic exchange. Some of these paths of communication remain life-time, however, these-in addition to the apical foramen and dentinal tubules-are pathways through which etiologic agents may pass between the pulp and the periodontium.

Pathogenesis of this condition is very simple. Infection in pulp tissue may lead to secondary infection or periodontal tissue breakdown. In contrary, severe periodontal disease may initiate or exacerbate inflammatory changes in pulp tissue.

Classification of endodontic–periodontic lesions, according to Caranzza[4]is presented as:

- Primary pulpal infection, leading to chronic periradicular periodontitis by which a periapical radiolucency can develop and migrate cervically.
- Primary periodontal infection, lead to extensive breakdown of alveolar bone that migrates from cervical area to the apex.
- Both primary pulpal and primary periodontal infection, occur simultaneously in an "independent" endodontic–periodontic lesion, exhibiting the characteristics of both.
- Primary pulpal and primary periodontal infections, that occurs extensively in "combined" endo-perio lesion.

In 2014, Al-Fouzan [5] presented a new endodontic-periodontal interrelationship classification, based on the primary disease and its secondary effect. The classification is as follows: (1) Retrograde periodontal disease (a)Primary endodontic lesion with drainage through the 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 and (5) Iatrogenic periodontal lesion.

In context of therapy, endodontic treatment is highly predictable and according to numerous authors has high successful rate when is appropriately performed. If majority of bony support has been lost from chronic periodontitis, regardless of predictability of endodontic therapy, tooth may have hopeless prognosis. Regeneration, root resection and hemisection are indicated as a part of strategic treatment of multirooted teeth [6].

At the beginning it must be noted that, if the root canal system is infected, endodontic treatment should be commenced prior to any periodontal therapy in order to remove the intra-canal infection before any cementum is removed. With this method several complications are avoided and a favorable situation for tissue repair is provided. The endodontic treatment can be completed before periodontal treatment except where there is a "combined endodontic-periodontal lesion with communication"-in these cases, the root canals should be medicated until the periodontal treatment has been completed and the overall prognosis has been reassessed as being favorable. The use of non-toxic intra-canal therapeutic medicaments is essential to destroy bacteria and to encourage tissue healing [7].

According to Kim et al,[8] even by using contemporary methods and techniques such as usage of an operating dental microscope during the treatment, endodontic-periodontal lesions often have a lower success rate than isolated endodontic lesion. In endodontic-periodontal lesions, the pulpal and periodontal tissues are mainly connected through the apical foramen. Thus, any pulpal inflammation may extend from the apical foramen into the periapical tissue. This typically results in local periapical inflammation that is associated with bone resorption.

The most important issue in therapy of these types of lesions is the medicaments that can be use for treatment. Calcium hydroxide has good antibacterial activity, and biocompatibility and its application after the root canal procedure could inactivate any exotoxins by hindering the increase in cytokine chemical inflammatory mediators [9].

After complete instrumentation, calcium hydroxide should be used as an intracanal medicament. It is an excellent medicament in general, because it is bactericidal, anti-inflammatory and proteolytic; it inhibits resorption; and it favors repair. It is especially effective in endo-perio cases because its temporary obturating action will inhibit periodontal contamination of the instrumented canals via patent channels of communication. When the etiology is purely endodontic, this regimen usually will resolve the pseudopocket within a few weeks.

According to Alshawwa, et al. [10] the obturation procedure of the root canals could be done safely by performing a successful root canal disinfection and preparation.

Treatment access and prognosis depend primarily on the diagnosis of the specific endodontic and periodontal disease. The main factors to consider for treatment decision-making are pulp vitality and the extent of the periodontal defect. Diagnosis of primary endodontic disease and primary periodontal disease usually presents no clinical difficulty. In primary endodontic disease, the pulp is infected and non-vital. When the pulp becomes inflamed/infected, it elicits an inflammatory response of the periodontal ligament. On the other hand, when a tooth with primary periodontal disease is present, the pulp is vital and responsive to testing. However, primary endodontic disease with secondary periodontal involvement, primary periodontal disease with secondary endodontic involvement, or true combine diseases are clinically and radiographically very similar. If a lesion is diagnosed and treated as a primarily endodontic disease due to lack of evidence of marginal periodontitis, and there is soft-tissue healing on clinical probing and bone healing on a recall x-ray, a valid retrospective diagnosis can then be made. The degree of healing that has taken place following root canal treatment will determine the retrospective classification. In the absence of adequate healing, further periodontal treatment may be indicated.

The prognosis and treatment of each endodontic–periodontal disease type varies. Primary endodontic disease should only be treated by endodontic therapy. Good prognosis is to be expected if treatment is carried out properly with a focus on infection control. Primary periodontal disease should only be treated by periodontal therapy. In this case, the prognosis depends on the severity of the periodontal disease and the patient response. Primary endodontic disease with secondary periodontal involvement should first be treated with endodontic therapy. It has been suggested that periodontal disease has no effect on the pulp before it involves the apex [11]. On the other hand, several studies suggested that the effect of periodontal disease on the pulp is degenerative in nature including an increase in calcifications, fibrosis and collagen resorption, in addition to the direct inflammatory sequelae. It appears that the pulp is usually not severally affected by periodontal disease until the periodontal tissue breakdown has opened an accessory canal to the oral environment [12]. At this stage, pathogens leaking from the oral cavity through the accessory canal into the pulp may cause a chronic inflammatory reaction, followed by pulp necrosis. However, if the mircovasculature of the apical foramen remains intact, the pulp may maintain its vitality [13]. The effect of periodontal treatment on the pulp is similar and scaling and root planning, curettage as well as periodontal surgery may not induce severe inflammatory changes of the pulp. Treatment results should be evaluated in 2–3 months and only then periodontal treatment should

be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition [14]. It also reduces the potential risk of introducing bacteria and their byproducts during the initial phase of healing. In this regard, it was suggested that aggressive removal of the periodontal ligament and underlying cementum during interim endodontic therapy may adversely affect periodontal healing. Areas of the roots that were not aggressively treated showed unremarkable healing [15].

Consequently, the prognosis for treatment of primary endodontic disease with secondary periodontal involvement depends primarily on the severity of periodontal involvement, periodontal treatment, and patient response. Jansson et al. [16] assessed the effect of endodontic pathogens on marginal periodontal wound healing of denuded dentinal surfaces surrounded by healthy periodontal ligament. Their results showed that in infected teeth, the defects were covered by 20% more epithelium while the noninfected teeth showed only 10% more connective tissue coverage. They concluded that pathogens in necrotic root canals may stimulate epithelial downgrowth along denuded dentin surfaces with marginal communication and thus augment periodontal disease. It was found that endodontic infection in mandibular molars was associated with more attachment loss in the furcational areas [17]. These authors suggested that endodontic infection in molars associated with periodontal disease might enhance periodontitis progression by spreading pathogens through accessory canals and dentinal tubules. In contrast to these findings, Miyashita et al. [18] failed to observe a correlation between a reduced marginal bone support and endodontic status.

When a restorative post is present in the involved root, the possibility of root fracture also should be considered in the differential diagnosis. A vertical root fracture usually will produce periodontal breakdown similar to pulpal-periodontal lesions. Surgical exposure often will be necessary, either to confirm the presence of a fracture or to perform the apicoectomy if no fracture is present. In teeth that have not been treated endodontically, the presence of extensive caries or of deep restorations certainly suggests endodontic etiology, although either of these might be only incidental findings in a tooth with periodontal involvement [19].

The success rate of joined endodontic-periodontal lesions without a regenerative procedure is between 27% and 37% [20]. These rates are much lower than the success rate of 95% with conventional nonsurgical root canal therapy [21]. The use of barrier membranes and bone-grafting materials during treatment encourages the growth of surrounding lost tissues such as the periodontal ligament, bone cementum, and connective tissue while preventing unwanted cell types such as epithelial cells [22].

CONCLUSION

According to aforementioned, it can be concluded that the primary endodontic disease with secondary periodontal involvement should first be treated with an endodontic therapy. Prognosis depends on the severity of periodontal involvement, periodontal treatment and patient response. Therefore, a primary endodontic lesion draining from attachment apparatus should be initially treated by an endodontic therapy. Primary periodontal disease should only be treated by periodontal therapy. In this case, the prognosis depends on the severity of the periodontal disease and the patient response. Communication between pulpal and periodontal tissues is possible because of structures such as apical foramina, lateral or accessory canals and dentinal tubules. Infection in pulp tissue may lead to secondary infection of periodontal tissue. In contrary, severe periodontal disease may initiate or exacerbate inflammatory changes in pulp tissue.

REFERENCES

1. Meng HX. (1999). Periodontic-endodontic lesions. Ann Periodontol. 4(1):84-89.
2. Oh SL, Fouad AF, Park SH. (2009). Treatment strategy for guided tissue regeneration in combined endodontic-periodontal lesions: case report and review. J Endod. 35(10):1331-1336.
3. Velickova N, Petrovski M, Milev M. (2021). Општа и орална хистологија со ембриологија - учебник. UGD, Stip.
4. MNHTPKF C. (2014). Carranza's clinical periodontology.
5. Al-Fouzan KS. (2014). A new classification of endodontic-periodontal lesions. Inter J Dent. 2014.
6. Jivoinovici R, Suciu I, Dimitriu B, Perlea P, Bartok R, Malita M. (2014). Endo-periodontal lesion-endodontic approach. J Med Life. 7(4).
7. Abbott P. (1998). Endodontic management of combined endodontic-periodontal lesions. J New Zealand Society Periodontol. 15-28.

8. Kim E, Song JS, Jung IY, Lee SJ, Kim S. (2008). Prospective clinical study evaluating endodontic microsurgery outcomes for cases with lesions of endodontic origin compared with cases with lesions of combined periodontal–endodontic origin. J Endod. 34(5):546-551.

9. Mohammadi Z, Dummer PMH. (2011). Properties and applications of calcium hydroxide in endodontics and dental traumatology. Int Endod J. 44(8):697-730.

10. Alshawwa H, Wang JF, Liu M, Sun SF. (2020). Successful management of a tooth with endodontic-periodontal lesion: A case report. World J Clin Cases. 8:5049.

11. Czarnecki RT, Schilder H. (1979). A histological evaluation of the human pulp in teeth with varying degrees of periodontal disease. J Endod. 5(8):242-253.

12. Rubach WC, Mitchell DF. (1965). Periodontal disease, accessory canals and pulp pathosis. The Journal of Periodontol. 36(1):34-38.

13. Langeland K, Rodrigues H, Dowden W. (1974). Periodontal disease, bacteria, and pulpal histopathology. Oral Surg Oral Med Oral Pathol. 37(2):257-270.

14. Chapple IL, Lumley PJ. (1999). The periodontal-endodontic interface. Dent Update. 26(8):331-341.

15. Blomlöf L, Lindskog S, Hammarström L. (1988). Influence of pulpal treatments on cell and tissue reactions in the marginal periodontium. J Periodontol. 59(9):577-583.

16. Jansson L, Ehnevid H, Blomlöf L, Weintraub A, Lindskog S. (1995). Endodontic pathogens in periodontal disease augmentation. J Clin Periodontol. 22(8):598-602.

17. Rotstein I, Simon JH. (2006). The endo-perio lesion: a critical appraisal of the disease condition. Endod Topics. 13(1):34-56.

18. Miyashita H, Bergenholtz G, Gro¨ndahl K. (1998). Impact of endodontic conditions on marginal bone loss. J Periodontol. 69: 158–164.

19. Solomon C, Chalfin H, Kellert M, Weseley P. (1995). The endodontic-periodontal lesion: a rational approach to treatment. J Am Dent Associat. 126(4):473–479.

20. Tsesis I, Rosen E, Taschieri S, Strauss YT, Ceresoli V, Del Fabbro M. (2013). Outcomes of surgical endodontic treatment performed by a modern technique: an updated meta-analysis of the literature. J Endod. 39(3):332-339.

21. Imura N, Pinheiro ET, Gomes BP, Zaia AA, Ferraz CC, Souza-Filho FJ. (2007). The outcome of endodontic treatment: a retrospective study of 2000 cases performed by a specialist. J Endod. 33(11):1278-1282.

22. Bashutski JD, Wang HL. (2009). Periodontal and endodontic regeneration. J Endod. 35(3):321-328.