The Relation of Endodontic-Periodontal Lesion and Therapy (Literature Review)

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ABSTRACT

The correlation between endodontic-periodontal lesion has been documented well by researches. Endodontic lesion originates from pulp, while periodontal lesion originates from periodontal tissues. Anatomically they are connected by apical foramen, lateral canal and accessories, as well as dentin tubules. The correlation appeared as the endodontic defect can be from periodontal lesion, or a periodontal defect is from a pulp tissue. Together they can emerge and form a combination lesion. Endodontic infections have been highly correlated with deeper periodontal pockets and furcation involvement in mandibular, the causal relationship between the two pathoses has not yet been established. This consensus supports the influence of degenerated or inflamed pulp that can happen on the periodontium; but not all researchers agree about the effect of periodontal disease on the pulp. **Conclusion :** The mechanism of endo-perio lesion need to taken care in order to have appropriate diagnostic so that the right therapy would be able to keep the teeth in the oral cavity.

Keywords : endodontic lesion, periodontal lesion, the correlation of endodontic-periodontal lesion, therapy

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INTRODUCTION

Teeth, pulp and supporting tissue are a set of biologic unity. The relation between these structures influence each other either in good or bad condition.¹ The relation between pulp-periodontal disease originally happens through the relation of anatomy and vascular between pulp and periodontium. Pulp and periodontal problems can cause more than 50% of tooth mortality. Death pulp can happen because of caries, restorative procedure, chemis and termal, trauma and periodontal disease. When product from degenerative pulp can reach the periodontium, then inflammation response takes run quickly, which is indicated by bone resorption and tooth mobility. If this happens at apical area, it could form periradicular lesion and this inflammation continues up to crestal alveolar bone that can form retrograde periodontitis or reversed pocket.³

Periodontal disease is a slow progressing disease, that may have a gradually atrophic effect on the dental pulp. Periodontal lesion is especially caused by plaque accumulation. Periodontal treatments such as a deep root planing and/ or curretage, usage of localized medicament, and gum injury or wounding may accelerate further pulpal inflammation and provoke the interrelated disease process.^{3,4}

In recent years, a periodontal disease has relation with pulpal disease and it may cause periodontal lesion that is different from

chronic destructive periodontitis. The effect of periodontal disease on the pulp and the potency for healing certain period lesions after endodontic treatment has been documented in the past. Therefore, the researcher would like to study more the relation of pulp-periodontal disease, the effects of pulp disease on the periodontium, periodontal disease and its effects on the pulp, classification, differential diagnosis and management.

Intercommunication between Pulp and Periodontal Tissues

The interaction process of pulp and periodontal disease can happen through lateral accessory canals, dentinal tubules, palatogingival grooves, periodontal ligament, alveolar bone, apical foramina, and common vasculolymphatic drainage pathways (the most interconnected and evidenced relationship between the two tissues is via the vascular system as illustrated anatomically by the presence of the apical foramen, lateral (i.e. accessory) canals, and dentinal tubules. These communications (if existed) may serve as potential paths for inflammatory reciprocity.⁵

Apical foramen is the most direct route of communication to the periodontium, followed by lateral and accessory canal, mainly in the apical area and to the furcation of molar. A direct pathway between pulp and periodontium typically contains connective tissue and vessels that connect the circulatory

system of the pulp with that of the periodontium. On the other hand, dentinal tubules contain cytoplasmic, in which odontoblastic processes can extend from the odontoblast at the pulp and dentine border to the dentin enamel junction (DEJ) or dentin and cementum junction (CDJ). Harrington and Steiner (2002) reported that pulp chamber could communicate with the external root surface via external dentinal tubules, especially when the cementum is denuded.⁵

Palatogingival grooves are developmental anomalies of the maxillary incisor teeth. But lateral incisors are more often than central incisors (44% versus 0,28% respectively). They usually begin in the central fossa, cross the cingulum, and extend apically with varying distances. Palatogingival grooves are often associated with poor periodontal health due to their inability to keep these areas clean.²

Perforation of the root creates a communication between the root canal system and the periodontal ligament. This may occur as a result of overinstrumentation during endodontic procedures, internal or external root resorption, or caries invading through the floor of the pulp chamber. The prognosis of teeth with the root perforation is usually determined by the location of the perforation, the time left unsealed, the ability to seal the perforation, the chance of building new attachments, and the accessibility of the remaining root canals.²

A vertical root fracture can produce a "halo" effect around the tooth radiographically. Deep periodontal pocket and localized destruction of alveolar bone are often related to long-standing root fractures. The fracture root can mimic a radiographic profile of occlusal trauma, with localized loss of lamina dura, altered trabecular pattern, and a widened periodontal ligament. The fractured site provides a portal of entry for irritants from the root canal system to the surrounding periodontal ligament. Vertical root fracture has contributed to the progression of periodontal destruction in the presence of apparently succesful endodontic tooth therapy and overall periodontal site stability.^{2,6}

Influence of Pulpal Pathologic Condition on the Periodontium

Etiology factors like pulp disease and procedure treatment of root canal can cause disparity at periodontium like : irritant from necrosis pulp, procedure treatment of endodontic and material root canal. Irritant from necrosis pulp can result disparity in periodontium. Pulpa degeneration results in necrotic debris, bacterial by products, and other toxic irritans that can move toward the apical foramen, causing periodontal tissue destruction apically and potentially migrating toward the gingival margin, which is called as Retrograde periodontitis.⁷

Procedure treatment of endodontic can result periodontal lesion. After extirpasi

pulp, sweeping and forming of root channel can push debris into periodontium so that it causes acute inflammation of periodontium in the form of cementum and bone resorption. Mistakes during endodontic procedure (like elementary perforation of pulp room, root perforation, vertical fracture when obturasi) can cause damage of periodontium.⁵

The medicaments (e.g. high concentration of calcium hydroxide, corticosteroids, antibiotics) used for root canal therapy can also irritate the periodontal attachment apparatus. pervasive to pass dentine tubulus and causes sementum necrosis.^{5,8}

The ability of the periodontium to regenerate lost attachment apparatus on pulpless teeth has been questioned, especially if these teeth contain a root canal filling and have been denuded of cementum. Although endodontic infections have highly correlated with deeper periodontal pockets and furcation involvement in mandibular molars, the causal relationship between the two tissues has not yet been established. Miyashita and coworkers (1998) suggested that endodontic treatment should occur before treatment of furcation lesions (i.e. bone regeneration) to ensure successful results.⁹

Influence of Periodontal Inflammation on the Pulp

Infection from a periodontal pocket may spread to the pulp through accessory

canals, which occur most often in the furcation and closer to the apex of teeth. In addition, bacterial products and toxins may also gain access to the pulp via exposed dentinal tubules. The pulpa reaction is not only influenced by the stages of periodontal disease but also by the type of periodontal treatment such as scaling, root planing, and administration of medication. Inflammation lesions of varying severity and necrotic pulp tissues are usually found in teeth with large canal or in cases where periodontal breakdown has extended to the apex.³

The Periodontal disease and periodontal treatments can influence pulp necrosis, like progressive periodontal disease and invasive treatment of periodontium. Progressive periodontal disease can result migration junctional epithelial towards apical, opening surface of root and the entry of bacterial plaque. Open root channel can continue toxic product into pulp so that it causes atrophic, degenerative, inflammatory and resorptive. Accumulation of plaque at root in apical area can cause inflammation and pulpa necrosis.^{9,10,11}

The invasive periodontal treatment like curettage will destroy venous in apical and causes pulp necrosis. Scaling and root planing will throw away sementum layer which may open lateral dentinal tubules and root channel. This is still disputed so that this situation may cause changes of pulpal pathosis.

Classification and Treatment

According to Simon and coworkers (1972 cit Ammons and Harrington, 2006), endodontic-periodontal lesion is classified into to five classes, they are primary endodontic lesions, primary endodontic lesions with secondary periodontal involvement, primary periodontal lesions, primary periodontal lesions with secondary endodontic involvement, true combined lesions (Figure 1).¹²

Fig.1 Endodontic and periodontic pathways.
A. Endodontic lesions. B. Periodontal lesions.
C. True combined endodontic and periodontic lesions
(Kerns and Glickman, 2006)

1. Primary Endodontic Lesions

Disease processes of the dental pulp frequently involve inflammatory changes. Caries, restorative procedures and traumatic injuries are the most common causes. Typically, endodontic lesions resorb bone apically, laterally and destroy the attachment apparatus adjacent to a non vital tooth. Inflammatory processes in the periodontium occurred as a result of root canal infection may not only happen at the apex of the root, but they may also appear along the lateral aspects of the root, and in furcation areas of two and

three rooted teeth. The emergence of these processes may or may not be associated with clinical signs of inflammation, such as pain, tenderness to pressure and percussion, increased tooth mobility, and swollen marginal gingiva, stimulated periodontal abscess.^{2,3}

The suppurative process may cause a sinus tract along the periodontal ligament space or through patent channels (including the apical foramen and lateral accessory canals). This usually results in a narrow opening of the sinus tractus into gingiva sulcus and pocket can be easily traced with a guta-percha cone or periodontal probe, such a tract that can readily be probed down to the tooth apex, where no increased probing depth would otherwise exist around the tooth. In multirooted teeth, a periodontal ligament sinus tract can drain off into the furcation area and a grade III, "through and through" furcation defect resulting from periodontal disease.^{2,3}

Because the primary lesion is an endodontic problem that has merely manifested itself through the periodontal ligament, complete resolution is usually anticipated after conventional endodontic therapy without any periodontal treatment.²

2. Primary Endodontic Lesions with Secondary Periodontal Involvement.

When a lesion of endodontic origin is not treated, usually pathosis will continue, leading to destruction of the peri-apical alveolar bone and progressing into the interradicular area,

causing breakdown of surrounding hard and soft tissues. Primary endodontic lesions can continue up to gingiva sulcus or furcation, usually at asymptomatic chronic phase. Prognosis bases on the success of treatment of endodontic and periodontic. Primary endodontic lesion with secondary periodontal involvement can happen as a result of perforation root during root canals therapy, fracture tooth root which is taken care of by endodontic or restoration with crown. The symptom is acute, with periodontal abscess causes pain, oedem, exudate, forming of pocket and tooth mobility. Chronic responses sometime happen without pain at all.⁸

When these occur, not only the diagnosis but also the prognosis as well as the treatment may become more difficult. Diagnostically, these lesions have a necrotic root canal and plaque or calculus accumulation, demonstrable by a probe and radiograph. Radiographs may show generalized periodontal disease with angular defects at the initial site of the endodontic involvement.³

3. Primary Periodontal Lesions

Periodontal disease begins in the sulcus and migrates to the apex as deposits of plaque and calculus produce inflammation, causing loss of surrounding alveolar bone and supporting periodontal soft tissues. This leads to a loss of clinical attachment and formation of a periodontal abscess during the acute phase of destruction. The progression of

periodontal disease to the formation of osseous defects and subsequent radiographic appearance along lateral aspects of root and in furcation areas is well known. These defects may or may not be in association with trauma from occlusion, which can often be the cause of an isolated periodontal problem. Osseous lesions of periodontal origin are usually associated with tooth mobility and the affected teeth respond positively to pulp testing. The bony lesion is usually more widespread and generalized than lesions of endodontic origin.³

The prognosis for those teeth affected by periodontitis worsens as the disease process and periodontal destruction progresses. Treatment depends on the extent of the periodontitis and on the patient's ability to comply with potential long-term treatment and maintenance therapy. Because this is purely a periodontal problem, the prognosis depends exclusively on the outcome of periodontal therapy.²

4. Primary Periodontal Lesions with Secondary Endodontic Involvement.

It is still questionable whether progressive periodontitis has effect on pulp vitality. The pulp has good defence as far as blood supply passes apical fully. Periodontal disease related to plaque accumulation seldom generates a change of pulpal pathosis. Pulpal damage can happen if periodontal pocket has reached apical foramen.³

5. True Combined Lesions.

Pulpa-periodontal disease may occur independently or concomitantly in and around the same tooth. The prognosis at multirrooted teeth with combined pulp and periodontal lesions depend largely on the extent of the destruction caused by the periodontal disease component. A necrotic pulp or a failing endodontic treatment, plaque, calculus and periodontitis will present varying degrees.²

According to Grossman (1998), factors influencing true combined lesion are anatomical factor, trauma and others. Anatomical factor is tooth crowding triggers trauma, like food impaction and occlusion trauma, multirrooted teeth accessory lateral canals, cervical enamel projection into furcation, big lateral canals at coronal or middle root.¹

Trauma can generate periodontal pocket or furcation involvement. If there is big lateral canals at pocket area, the pulp usually will be open so that it causes periodontal problems and becomes pulpitis irreversible. This trauma can also cause crown fracture, root migration, necrosis or periapical diseases. Furthermore, it can generate cellular changes resulting internal and external resorption related to root perforation. Tooth trauma can happen because of cavity preparation, orthodontic treatment, malocclusion and parafunctional habits.³

Other factors are in the form of iatrogenic mistakes, for example furcation perforation in the root canal therapy, root perforation or apical root which is bent in the instrumentation. Besides that there is some possibility of systematical factor like diabetes may cause combined lesion.¹

Diagnosis of Comparative Endodontic-Periodontal Lesions

To avoid from faulty diagnosis, prognosis and maintain a correct treatment plan, it is necessary to evaluate some examination, such as subjective symptoms, radiographic and other clinical tests.^{1,9,10,12}

1. Subjective symptoms

Subjectively, a complete history of detailed location, intensity and frequent pain as well as drugs used to eliminate the pain can be helpful to find the source of disparity. The subjective symptoms are usually pain and edematous. The pain at the pulp lesion and periradicular is local phenomenon and tends to generate exciting pain. In chronic periodontal disease, the process is flattening and there is a little pain. But in the acute phase, it can be followed with moderate pain, for example in the form of periodontal pocket or ulcerative gingivitis.^{10,12}

Edematous in the tooth with pulp lesion, generally can be seen in the meeting area of mucogingiva in gingival mucosa. An edematous face can happen due to endodontic lesion; and it seldom happen in periodontal abscess,

which usually tends to happen in attached gingiva.^{10,12}

2. Radiographic examination

Radiographic examination is important to diagnose comparative endodontic and periodontal lesions. Periapical lesion destroys periodontium in apical area; sometimes it may continue up to cervical. Periodontal lesion is usually connected with the loss of angular bone extending to apical area. It is usually not isolated in one tooth, while endodontic lesion often happens only in one tooth.

The radiographic figure of endodontic-periodontal lesion is erratic. Therefore, radiographic diagnosis itself is just inconceivable, and it must be followed by clinical test.^{10,13}

3. Clinical test

Clinical test must be performed to determine the origin of disparity, so that it can ascertain the correct prognosis and definitive treatment. The clinical test includes vitality test, probing, palpation, percussion, and visual inspection. The results of vitality test is usually certifiable, although it is not wholly reliable. Primary endodontic lesion is always connected with necrosis pulp; whereas in primary periodontal lesion, usually the pulp reacts normally to the thermal test. Electrical test shows that the pulp is still vital. Sometimes there is a real combination lesion. In this case,

necrosis pulp is caused by deep caries, restoration or trauma.^{9,10}

Cavity test can ensure vitality pulp in the cases at which radiographic or other vitality tests cannot be concluded. The defect coming from periodontal diseases usually shows positive cavity test (vital pulp), while the defect coming from the pulp usually shows negative test.¹⁰

Probing. Because of limited procedural inspection of the pulp and the confused picture of radiographic test in endodontic lesion or periodontal diseases, probing is used as a comparative test. The probing defect resulted from endodontic lesion is usually narrow, tight and extends to apical foramen or to root canals. Sometimes periodontal pure lesions look like narrow, tight defect coming from endodontic. In this case, endodontic lesion looks like periodontal lesion.²

Palpation and percussion. Palpation on tooth tissue with periodontal lesion or periradicular lesion is very few. Palpation on coronal gingiva at periodontitis or palpation at apical lesion of tooth is useful to have comparative diagnoses of periradicular and periodontal lesion. Positive percussion has shown inflammation on periodontium. Because periodontal lesion or endodontic lesion has caused the inflammation, so that percussion cannot be used to differentiate the diseases.³

Table 1. Clinical and radiographic endodontic-periodontal lesions (Walton, Torabinejad, 1996).

Lesion type	Limited to teeth	Caries or restoration	Vitality test	Probing defect	Vertical resorbtion	Palpation or percussion	Treatment
Endodontic source	+	+	-	+	-	±	Endodontic
Periodontic source	-	-	+	-	+	±	Periodontic
Combined	±	±	-	-	+	±	Endodontic and periodontic

Visual inspection of teeth and gingiva add supplementary information to support the results of the previous tests. In the primary endodontic lesion, there must be some reasons for the dead pulp which can be caused by caries, wide restoration, tooth fracture, trauma history, the crown which has changed its colour. Primary periodontic lesion is shown when there is no damage of clear corona in its relation with periodontal pocket, plaque, calculus and gingivitis or periodontitis.

DISCUSSION

Irritancies from root canal system can pass lateral canals or apical foramen and cause pathologic changes in periodontium. However, whether periodontal diseases influence the pulp through the same canal it is still being disputed. The different concentration and irritant potency in the necrosis pulp compared with periodontium irritancies can explain different inflammation at every tissue.¹⁰

The ability of periodontium to regenerate attachment apparatus on the disfunctioned pulp is still questionable, especially on the tooth with root canal and denuded cementum. Jansson and Ehnevid (1998) reported that after endodontic treatment teeth do not respond as well as tooth which treated periodontally.¹⁴ Sanders et al. (1983) found that 60% of bone regeneration at periodontal defect happened to teeth which are not treated endodontically, compared with 33% of bone regeneration at teeth with endodontic treatment.¹⁵ But on a research with monkeys, it was reported that any periodontium has a potency to regenerate after periodontal surgery, regardless its pulp status (vital, filling, having medicament or open pulp). Another study reported that the pulp status has a small influence on initial cementogenesis and filler root canal has not changed sementum deposition.¹³

According to Miyashita et al. (1998), even though endodontic infection has relation with deep periodontal pocket and furcation involvement in mandible molar, its cause-effect between two pathoses is not yet proven. It is suggested that endodontic treatment is better performed before furcation lesion treatment (bone regeneration) to get good results.⁹

The result of the consensus supports the influence of pulp degeneration or inflammation pulp on the periodontium; and not all researchers agree with the effect of periodontal diseases on the pulp. The change of inflammation and necrosis pulp must be observed carefully, especially in the area nearby lateral canals at open root because of periodontal diseases.³

Chaker (1974, cit Grossman 1998) stated that periodontal diseases usually will not cause pulp diseases, because inflammation would follow vein drainage and venous blood flows out into the pulp and goes into periodontium passing apical foramen. Czarnecki and Schilder (1979, cit Grossman 1998) also reported that there was no causal relation between periodontal diseases and the pulp. The overall role of lateral or accessory canals in endodontic-periodontal is not clear yet, whether every lateral or accessory canal can cause pulpitis irreversible.³

Grossman (1998) mentioned that periodontal diseases can cause a change of pathologic pulp, especially through lateral or

accessory canal. There is a pathologic reaction on the pulp at near open lateral canal; however, not every open lateral canal can cause pulpitis irreversible, because the pulp is a tissue having the ability to maintain and improve open lateral canals.¹

Some clinicians suggest that the treatment of initial combined lesion depends on the starting sources of the diseases. Other clinicians suggest that endodontic treatment is partly performed through preparation and root canal medication, followed by periodontal therapy until a good result is obtained. Then endodontic procedure is finalized. Grossman (1988) suggests that endodontic treatment is performed to precede periodontal therapy, regardless of causative diseases.¹

Determination of treatment procedures depends on many factors. If the pulp test is normal to all vitality tests (such as electrical test, thermis test, or cavity test) and the pulp does not react abnormally and functions well without causing pain, then the treatment can be limited on periodontal treatment only. The result of the treatment is observed accordingly until it shows curative result; when needed, endodontic treatment can be started.¹

When traditional endodontic and periodontal treatments prove insufficient to stabilize an affected tooth, the clinician must consider alternative treatment, like resection or regeneration. Resective techniques focus on eliminating the diseased roots or teeth; and regenerative efforts are aimed at restoring lost

biologic structure. Root resection is the removal of a root with accompanying odontoplasty, before or after endodontic treatment. The indications of root resection often includes root fracture, perforation, root caries, dehiscence, fenestration, external root resorption involving one root, severe impaired endodontic treatment of a particular root, severe periodontitis affecting only one root, and severe grade II or III furcation involvement. Recently the concepts of guided tissue regeneration (GTR) or guided bone regeneration (GBR) have been used to promote bone healing after endodontic surgery.^{2,3}

CONCLUSION

Endodontic and periodontic lesions are resulted from the close interrelationship of pulp tissues and the periodontium. The major pathways of communications between the two types of tissues are the apical foramina, lateral and accessory canals and dentinal tubules. The differential diagnose of endodontic and periodontic lesion is not always straightforward and requires clinical data accumulation from a number of diagnostic tests to obtain a correct diagnosis.

When examining and treating the combined or individual lesion in endodontics and periodontics, the clinician must bear in mind that successful treatment depends on the correct diagnosis. Lesions with combined cause will require both endodontic and

periodontal therapies, and endodontic therapy should usually be completed first. In addition, root resection and regenerative technique offer alternative approaches, thus enhancing the clinician's ability to deal with these complex clinical problems.

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