Two types of endo-perio lesions in mouth

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ABSTRACT

Background: Endo-perio lesion is responsible for more than 50% of tooth necrosis. This lesion manifests from embryonic, anatomical, and functional interrelationships between pulp and periodontium. There are few types of endo-perio lesion based on the variations in etiology and pathogenesis.

Purpose: The purpose of this article is to describe the managements of an endo-perio lesion with holistic treatments, including endodontic and periodontal.

Case: A 40 years old female patient came with major complaint on her right maxillary molar. The gingiva in the region had been swollen intermittently followed by a dull pain since the last few months. The additional complaint was on the left maxillary molar which had a similar swelling and pain history, only the frequency was less. The tooth 16 was non-caries, non-vital, positive in percussion and palpation, 2° mobility, and extruded. The gingival was inflamed and there was a wide deep pocket toward apex at palatal. From radiograph, there was an apical radiolucency +6mm in diameter, involving all roots. The tooth was diagnosed as chronic dentoalveolar abscess due to pulp necrosis from traumatic occlusion. Its classification was “true combined lesion”. While tooth 26 is vital with gingival recession at palatal and wide deep pocket at mesial. The tooth was diagnosed as chronic periodontitis, classified as “primary periodontal lesion”.

Case management: The treatments of tooth 16 were elimination of etiology followed by endodontic and periodontal treatments (flap operation). While for tooth 26, only periodontal treatment is indicated. After endodontic treatment, tooth 16 showed a great healing (no subjective complaint, no mobility, normal response in percussion and palpation) but only on endodontic lesion, while the pockets were still deep (8 mm became 6 mm). Only after periodontal treatment with flap operation the pocket depths were reduced greatly (6 mm became 3 mm).

Conclusion: Different type of endo-perio lesion requires different treatment planning. The success of the managements of a combined lesion, as represented by tooth 16, depends on both endodontic and periodontal treatments as well as holistic approaches.

Key words: Endo-perio lesion, traumatic occlusion

INTRODUCTION

Interrelationship between pulpal and periodontal diseases, known as “endo-perio lesion”, has been a controversy for over 100 years.¹ Bender² and Chen³ stated that pulpal and periodontal problems are responsible for more than 50% of tooth necrosis.

This complex lesions are manifested from embryonic, anatomic, and functional interrelationships between pulp and periodontium.⁴,⁵ They are frequently asymptomatic for a long time, resulting in late detection until the symptoms of acute inflammation appear. Sometimes, these lesions are detected intuitively from routine check up.⁵
Frequently, endo-perio lesions are not considered as complex lesions which are related each other and are only assessed from the most evident sign. The result is that the management will be only focused on one point of view, either endodontically or periodontically, whereas these lesions may need comprehensive treatments from both areas. As a consequence, healing will not occur optimally, even failure can be resulted. However, only knowing the relation of both lesions is not enough. The ability in analyzing the primer etiology of these lesions is very important in planning proper treatment and in determining prognosis.

The aim of this article is to differentiate two types of endo-perio lesions founded in one mouth on both contra lateral sides, to discuss the treatment plans of each case, and to describe the managements of one of the endo-perio lesions with holistic treatments, including endodontic and periodontal treatments.

CASE

A 40 years old female patient came with major complaint on her right maxillary molar (tooth 16). The gingival in the region has a repetitious swelling along with mobility and dull pain histories since the last few months. The additional complaint was on the left maxillary molar (tooth 26) which had a similar swelling and pain history, only the frequency was less.

Patient's general condition was good. There was chewing on one side (right) habit since 5 years ago due to extraction of the left mandibular second molar (tooth 37). The right mandibular second molar (tooth 47) was also extracted 15 years ago. From extraoral examination, clicking on both sides of temporomandibular joints (TMJ) was revealed.

Oral hygiene was moderate. There were gingival recessions and absolute pockets on both sides of posterior maxilla and on anterior mandible, attrition on almost all teeth, anterior deep bite, and multiple diastemas. Teeth 18, 28, 37, and 47 were missing. The antagonist and adjacent teeth were malpositioned (extruded or tipping) due to unreplaced missing teeth.

Tooth 16 was non-caries, vitality (-), percussion and palpation (+), 2° mobility, 1 mm extruded on mesio-buccal cusp. Gingival was inflamed and recessed 4 mm on palatal surface. Palatal pocket was wide and 8 mm in depth. Buccal pocket was 3 mm. There was an overhanging and overcontour composite resin filling on cervico-palatal of the crown, accompanied with abundance of plaque retention. After the filling was removed, the surface of the tooth was looked smooth without cavity.

The radiograph (Figure 3) showed a radiolucent lesion at apex involving all roots with diameter ± 6 mm. The decreasing of density on alveolar crests between teeth 14, 15 and 14 were unfavorable.
From collected data, the tooth was diagnosed as pulp necrosis with chronic dentoalveolar abscess caused by traumatic occlusion along with chronic periodontitis. The classification of endo-perio lesion was “true combined lesion” according to Rotstein and Simon’s classification. The differential diagnosis was primary periodontal lesion with secondary endodontic involvement, which means pulp necrosis with chronic dentoalveolar abscess caused by retrograde pulpal infection originated from primary periodontal lesion.

Tooth 26 was non-caries, vitality (+), no mobility, percussion and palpation (-), and there was 4 mm gingival recession on palatal, wide pockets on mesial, 6 mm in depth that decreased gradually as it went to distal. Thus, total attachment loss on mesial was 10 mm. Radiograph of tooth 26 (Figure 7) showed angular bone lost on mesial up to apical third. The mesial root was thin and pointed. There was widened periodontal ligament along root surface with condensed lamina dura thorough lateral surface of distal root. The decreasing of density on alveolar crest between teeth 24, 25, and 26 indicated that there was chronic periodontitis. The contact point on mesial surface of tooth 26 was unfavorable. Disto-lingual cusp of tooth 36 acted as plunger cusp.

Tooth 26 was diagnosed as chronic periodontitis exacerbated by food impaction. The differential diagnosis was chronic periodontal abscess. The classification of endo-perio lesion was primary periodontal lesion.

CASE MANAGEMENT

The treatment plans for tooth 16 were endodontic with onlay as the final restoration followed by periodontal treatment (debridement with flap operation). The prognosis was doubtful because of the severity of periodontal lesion.
At first visit, prior to access, reduction of occlusal plane of tooth 16 was performed. Canal preparation was done with crown down system using Protaper® hand files. For root canal dressing, calcium hydroxide (Calcyl®) was used. For obturation, Protaper® gutta percha cone and Endomethasone® sealer were used. The overcontour and overhanging composite resin filling on cervico-palatal was removed to facilitate plaque control. Pocket was rinsed with concentrated povidone iodine (Betadine®) and 3% H2O2 solution to eliminate anaerobic bacteria. Patient was instructed to improved oral hygiene. Two percent chlorhexidin gluconate solution (Minosep®) was administrated for 2 weeks. Antibiotic was not administrated because the infection was chronic.

On a recall visit, 7 months post endodontic treatment, tooth 16 has become asymptomatic and functional even though the patients had not undergone periodontal treatment on both teeth, except deep scaling on the very beginning due to lack of cooperativeness and financial problem. However, the tooth was negative in percussion and palpation; no tooth mobility; buccal pocket was remain, palatal pocket was reduced but still deep, from 8 mm became 6 mm; and there was bleeding on probing, indicated remaining inflammation on gingival tissue. The radiograph showed the size reduction of apical lesion, and body of lesion was less radiolucent, indicated that there had been bony septum formation in apical lesion. On this visit, scaling and root planing on inflamed site was performed.

At next recall visit, 11 months post endodontic treatment, the clinical condition of tooth 16 still remained; only the BOP was no longer exist. From radiograph there was more significant healing, marked by reduction of apical radiolucent. Eighteen months after obturation, the patient finally willed to undergo flap operation on tooth 16. On flap opening, the alveolar bone at palatal site remained only at apical one third, along with much granulated tissue. Flap was performed with envelope design, including incision at palato-cervical side of tooth 14 to 17.

On a recall visit one month later, the tooth 16 was asymptomatic and functional. The gingival was not inflamed; palatal pocket has been reduced from 6 mm to 3 mm. But there was a quite great progression on untreated tooth 26. The mesial...
pocket has become deeper, from 8 mm to 10 mm, along with 2 degree of mobility. The tooth was still asymptomatic and thermal test showed normal reaction. However there was a possibility that the tooth has been chronically inflamed.

**DISCUSSION**

Tooth 16 was diagnosed as pulp necrosis with chronic dentoalveolar abscess along with chronic periodontitis. Non-vital pulp status was confirmed with cold test using ethyl chloride spray on cotton pellet that was applied on well-isolated tooth surface. The result was negative. Vitality then rechecked with cavity test which also gave negative result. The diagnosis of chronic dentoalveolar abscess was set based on history about an intermittent gingival swelling, on examination that showed 2° mobility, positive in percussion and palpation, and on an apical radiolucent appearance. Generalized chronic periodontitis was confirmed by the presence of generalized shallow pockets (except on tooth 16 and 26) and reduction of alveolar ridges with moderate oral hygiene.

The etiology of pulp necrosis of tooth 16 was hardly estimated as traumatic occlusion. The missing of tooth 47 caused mesial tipping of tooth 48 and distal movement of tooth 46. There was severe attrition on occlusal surface of tooth 46. Tooth 16 was 1 mm

**Figure 12.** One week post flap operation, the gingival recession became 6 mm and pocket became 4 mm. OH was good. The palatal gingival had purplish color, which was a normal reaction. There was no subjective complaint.

**Figure 13.** One month after flap operation. There was no subjective complaint. The gingival color was normal, BOP(-), the recession became 7 mm and pocket reduced to 3 mm.

**Figure 14.** One month after flap operation. The size reducing of apical radiolucent lesion has became more obvious, particularly on palatal root.

**Figure 15.** Untreated tooth 26, 20 months after initial visit. The radiograph showed no significant progression, but clinically the tooth was worsened.
extruded on mesio-buccal cups. The altered tooth positions caused disharmony of occlusion. The distal movement of tooth 46 created cup to cusp occlusion against teeth 16 and 15. According to literature, traumatic occlusion can be resulted from gradual alteration of occlusion caused by attrition, drifting movement, and extrusion combined with parafunction habits such as bruxism or chenching. In this case, the patient had chewing habit on one side (right) due to unreplace missing tooth 37.

Other possibility of the etiology of pulp necrosis of tooth 16 was retrograde infection originated from extended periodontal infection involving apex. In this stage, periodontal pathogens were penetrated into pulp through apical and/or lateral foramen, along with micro-vascularization disturbance on the apex. Inflammation effect of periodontal towards pulp, however, was still a controversy. Chaker stated that the spreading direction of inflammation products is following the vein stream which is from pulp to periodontium.

The existence of composite resin on the palato-cervical surface may also compromise the health of the pulp. The prolonged acid etching may irritate the pulp. But it cannot be assumed as a single cause of the necrotic pulp because the filling was placed on a shallow non-cavity surface.

If pulp necrosis of tooth 16 was considered as the result of traumatic occlusion then the classification of endo-perio lesion on this tooth is “true combined lesions” (according to Rotstein and Simon's), while if the pulp necrosis was considered as the result of retrograde pulp infection originated from extended periodontal infection then the classification becomes “primary periodontal disease with secondary endodontic involvement.” The comparison of the two possible differential diagnoses can be seen in table 1.

Finally, the endo-perio lesion of tooth 16 was classified as “true combined lesion” because the possibility of pulp necrosis due to traumatic occlusion was higher than due to retrograde infection. The reason was that there were abundance of proofs in supporting traumatic occlusion, including one side chewing habit, attrition facets on occlusal surfaces, and unreplaced missing tooth 47 that caused malpositioned adjacent teeth.

The overcontour and overhanging composite resin filling on palato-cervical retained plaque and food debris, thus deteriorating existing periodontitis. This filling was removed to facilitate plaque control on that area. The surface of the bare palatal root was smooth and non-cavity, thus refilling was not necessary. Besides of the pressure of abscess, the inflamed periodontal can also produce dull pain due to activation of C nerve fibers on periodontium and due to the production of inflammation mediators.

The tooth 16 needed both endodontic and periodontal treatments. However the endodontic procedure was completed prior to periodontal in order to eliminate etiology factor in the root canals leading to periapical healing. In both recall visits the probing examinations showed only slight reduction (2 mm) on palatal pocket. It was in accordance with the theory that stated that the endodontically treated tooth with combined lesion will deliver improvement only on apical lesion, while periodontal lesion will still remain.

According to Rotstein and Simon, the prognosis of combined lesion was based on the severity of periodontal lesion. In this case, the lesion was very deep extended to apex, thus the prognosis was doubtful. Bruseth et al concluded that a tooth with an endo-perio lesion that later treated endodontically has a significant chance of 19% to lose that tooth within the first two years.

Tooth 26 was classified as “primary periodontal lesion” because it fulfilled the characteristics as followed: the presence of plaque and calculus accumulation, wide and deep pocket toward apex. The pulp vitality was assured by cold test. The respond was sharp brief pain only on stimulus application, therefore it was concluded that tooth 26 was vital and healthy. The tooth was still vital possibly because the periodontal lesion involving mesial root has not reached the apex, while there were no lesions on distal and palatal roots. Therefore, periodontal pathogens have not invaded pulp yet through apical foramen and pulp vascularization has not disturbed. If periodontal lesion reaches the apex, vascular supply may be disturbed and periodontal pathogen may penetrate into pulp resulting retrograde infection followed by pulp necrosis. If the progression of the disease come to this stage then it would be classified as “primary periodontal lesion with secondary endodontic involvement.”

The main predisposing factor on this tooth was food impaction due to the angular appearance of alveolar bone loss on mesial where the contact point was unfavorable, accompanied by the presence of plunger cups (disto-lingual cusp of tooth 36).
The fist phase of treatment was eliminating etiology and predisposing factors, including hygiene stage: deep scaling and root planing to eliminate periodontal pathogens; occlusal adjustment (by selective grinding) and occlusal force distribution (by fabricating removable partial denture for the missing tooth 37) to eliminate traumatic occlusion and to improve TMJ condition; and grinding of plunger cups and correction of contact point to eliminate food impaction. After a period of re-evaluation for 4-6 weeks, the next was correction phase, that was debridement (subgingival scaling and root planing) facilitated by flap opening. The alternative treatments were endodontic treatment prior to mesial root resection, followed by coronal splinting. As a conclusion, the success of treatment is depend on proper diagnosis, appropriate treatment plan, careful treatment procedures, patient's respond towards treatment, and reevaluation phase. In this case, with the presence of endo-perio lesion, traumatic occlusion, and TMD, cooperation among dental disciplines was necessary, including conservative dentistry, periodontology, and prostodontics, in order to give optimal outcome for the patient. From the case of tooth 16 discussed above, it was proven that only with endodontic treatment alone, an optimal healing cannot be achieved. Subsequent periodontal treatment is needed. Traumatic occlusion and TMD have to be overcome by collaborating with prostodontics. Otherwise, the healing process of tooth 16 would be disturbed with recurrence risk and the progression of periodontal lesion on tooth 26 would be continued, while TMD would be worsening. Finally, the cooperation from patient is very important. Unfortunately due to financial limitation and lack of priority on dental treatment the patient is not very cooperative to do holistic treatment.

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