

# PREVENTIVE ENDODONTIC CONSIDERATION ON PULP AND PERIAPICAL TRAUMATISM IN FIXED FINAL RESTORATION

(PERTIMBANGAN ENDODONTI PREVENTIF TERHADAP TRAUMA PULPA DAN PERIAPIKAL DALAM RESTORASI AKHIR CEKAT)

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## Abstract

The success of fixed restoration preparation relies on several factors i.e. preparation of abutment tooth which must consider pulp-dentin complex and degree of occlusal traumatism. The thinner remaining dentin thickness during abutment preparation, the easier for inflammation and microbial invasion to occur. Inflammation of the pulp with release of inflammatory mediators such as prostaglandin, interleukin and microbial invasion through dentinal tubules played an important role in bone resorption and periapical lesions. This article was aimed to find out the treatment for periapical abscess resulted from porcelain-fused-to-metal (PFM) fixed restoration on maxillary left canine. A 56 year-old male patient was referred to clinic at Department of Conservative Dentistry, Faculty of Dentistry University of Sumatera Utara. He had a bridge from 23 to 26 on his maxillary left side. He felt discomfort in his maxillary left canine. Radiographic evidence showed radiolucency at apical area and clinical evaluation revealed periapical abscess at gingival mucosa of the tooth. Patient felt tenderness to percussion and palpation. Sequence of the treatment procedures was undertaken as follows: trephination and abscess drainage, root canal treatment using step back technique, obturation using lateral condensation method and eventually restored with composite resin using silane as coupling agent. At two-month evaluations, patient had no dental complaint, no tenderness to percussion and palpation as well as absence of clinical and radiographic periapical abscess. As conclusion, root canal treatment following hermetic root canal obturation and proper final restoration for periapical abscess resulted from PFM fixed restoration will determine the success of the treatment outcome.

**Key words:** Porcelain-Fused-to Metal, fixed restoration, pulp dentin complex, periapical abscess

## INTRODUCTION

Inflammation process may exist prior to any operative procedure or may result from preparation of the tooth as abutment under fixed final restoration i.e porcelain-fused-to-metal (PFM) bridge which may cause pulp-dentin complex disturbance. Periapical and occlusal traumatism may also occur in an intact periodontium or in a periodontium that has been reduced by inflammatory periodontal disease. Manappallil et al. found that when broken stress fixed restorations are used in mouth with weakened supporting tooth structures, deterioration of the bone was more rapid and severe.<sup>1</sup> Pulp-dentin complex plays an important role in tooth preparation for fixed restoration abutment such as crown and bridge

work. Pashley stated that there is a great deal of evidence that dentin and pulp are integrated units of tissue and functionally coupled.<sup>2</sup>

The evaluation of abutment teeth or teeth to be used for support of a bridge is the most important. A structure is as good as the foundation upon which it rests. When the teeth are prepared too small, it will do harm the pulp-dentin complex zone. Additionally when the teeth are used as abutment for bridge, they will receive lateral and vertical forces.<sup>1,3</sup> The force may injure the teeth and inflammation process will begin. In inflammation process, there are inflammatory cells such as neutrophils, lymphocytes, macrophages and plasma cells. In addition, it will result in the activation of sensory nerves and pain signals are transmitted to the central nervous system. Antidro-

mic stimulation of the nerve terminals causes the release of neuropeptides such as substance P, calcitonin gene-related peptide (CGRP) and neurokinin A (NKA) cell by which they will lead to vasodilation and vascular permeability. Inflammatory cells will produce cytokines which is one of the inflammatory mediators that may lead to bone resorption. Macrophages and neutrophils as the inflammatory cells are particularly important source in producing prostaglandins (PGs), leukotrienes (LTs), thromboxanes (TX), interleukins (ILs) and tumor necrosis factors (TNF) with arachidonic acid as precursor molecule.<sup>4</sup> They are important in a number of physiologic and pathologic processes and some play significant roles in inflammation. More over if the inflamed teeth are left untreated, they will become necrosis and even with periapical lesion which can be seen as radiolucence view around the tooth apex. It implies the bone loss which is initiated by periapical inflammation.<sup>5,6</sup> The amount of tissue destruction is directly correlated with the total microbial content in the root canal system and to the length of time, these tissues are exposed to the infecting organism.<sup>7,9</sup>

This case report was aimed to report that pulp necrosis associated with periapical radiolucency can develop in an abutment tooth under a dental bridge by inflammation process resulted from pulp-dentin disturbance, bacterial invasion through dentinal tubules and forces during mastication.

## CASE

A 56 year-old man was referred to Department of Conservative Dentistry, Faculty of Dentistry, University of Sumatera Utara with main complaint was discomfort on chewing or percussion and palpation in his left canine.

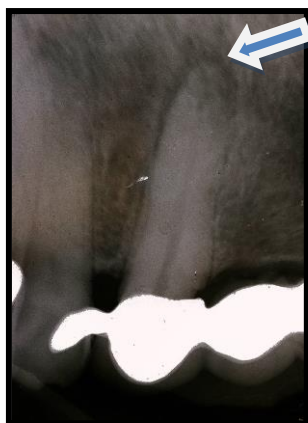


Figure 1. Traumatized tooth with periapical radiolucency

Patient had a porcelain-fused-to-metal (PFM)

bridge from 11 to 23 on his anterior side and he reported that the bridge was made a couple of years ago. Radiographic evaluation revealed that there was radiolucent periapical involvement, widening of periodontal ligament space and disruption of the lamina dura. Clinical finding was acute periapical abscess (Figure 1).

## Case Management

In the first visit, the tooth was trephinated and pus drainage was performed in order to relieve the intra-dental pressure. Apical preparation was serially enlarged to the master apical file (MAF) with circumferential filling. Coronal and mid-root preparation was performed to completion using SS K-file with 1 mm incremental step-back method started from file #25/23,5mm and finished with file #55. Recapitulation was done after each instrument with the MAF. Irrigation was performed with sodium hypochlorite 5.25 % and ethylene diamine tetra acetic acid (EDTA) gel was used as chelating agent. The canal was then dried and an intracanal inter-appointment dressing with pure calcium hydroxide powder mixture and glycerin vehicle were placed. Temporary filling was placed in the access cavity.

Two weeks later, after isolation of the tooth and removal of the temporary filling, calcium hydroxide was removed with copious irrigation of sodium hypochlorite. Radiographic view showed the improvement of periapical radiolucency. Patient felt no discomfort feeling and clinical symptoms were found (Figure 2). #40 gutta percha cone was applied as master apical cone.



Figure 2. Master cone of gutta percha

A week later root canal was then dried and filled with gutta percha and endomethasone sealer by using cold lateral condensation technique. The orifice was then sealed with glass ionomer cement (GIC) as lining material. Radiographic view reveal-

ed that there was diminution of the periapical radiolucency (Figure 3).



Figure 3. Filled root canal

In the fourth visit, the filled root canal was observed with no clinical symptoms, tenderness to percussion and palpation. Patient had no discomfort feeling. Final restoration was performed with composite resin filling by using silane agent as intermediate material between the PFM bridge and the adhesive material. Radiographic examination revealed regeneration of the periradicular tissues (Figure 4).

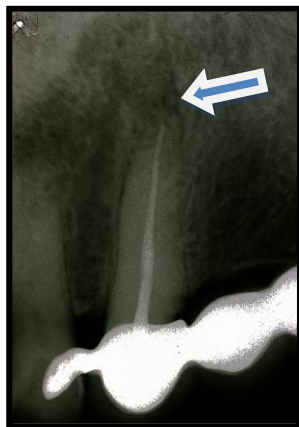


Figure 4. Diminution of periapical radiolucency

## DISCUSSION

Pulp necrosis may develop when the abutment teeth are prepared to dentin-pulp complex disturbance and will undergo exudative process to be periapical abscess. The thickness of remaining dentin is proportional to the possibility of the microbial invasion and inflammation. In other words, the thinner the remaining dentin thickness, the easier for the microbial invasion and the inflammation to take place. In this case, the defense mechanism of the pulp was weakening because the preparation of

abutment did not consider the totality of pulp dentin complex. In addition, functional stress during mastication would cause weakened pulpal defense. Inflammation process will begin and it will lead to pulp necrosis if the teeth are left untreated. Inflammation involves inflammatory cells such as macrophage and lymphocyte which in turns will produce chemical inflammatory mediators such as prostaglandins ( $\text{PGD}_2$ ,  $\text{PGE}_1$ ,  $\text{PGE}_2$ ,  $\text{PGF}_2$ ,  $\text{PGI}_2$ ), cytokines (IL, TNF, IFN), leukotrienes (LTs) and it will also affect the pain sensory nerve to produce several neuropeptides such as substance P, CGRP and NKA cells. These inflammatory mediators will cause bone resorption, pain, vasodilation, increased vascular permeability and neurovascular disorder. A primary endodontic lesion draining through the attachment apparatus should therefore be treated initially by endodontic therapy (trephination). It was performed for pain relief by reducing intrapulpal pressure once the periapical abscess occurred. This opening established a pathway for drainage of the inflammatory exudates.<sup>6,10</sup> In this case, calcium hydroxide was used as the intracanal inter appointment dressing to disinfect the root canal system further and to evaluate the improvement of the surrounding tissues at the following appointment, at which time it was decided to fill the root canal. Ion  $\text{OH}^-$  and  $\text{Ca}^{+2}$  released from calcium hydroxide in relatively big amount could promote the alkaline environment of root canal and result in sterilization and calcification.<sup>11,12</sup>

The combination of sodium hypochlorite solution 5,25% as irrigant and EDTA gel were very good at eliminating smear layer and organic debris as well as penetrating to the dentinal tubules. Osteogenesis was expected so that healing of lesion and bone would occur. Even though calcium hydroxide was applied only in one visit however, healing could already be seen through radiography in the second visit, it implied that root canal debridement and bacteria elimination held a very vital role in tissue repair as well as bone healing.<sup>13,14</sup>

In conclusion, remaining dentin thickness must be an important consideration because it plays a very important role during abutment tooth preparation for fixed final restoration such as bridge work and crown. The thickness will determine the possibility of inflammation and bacterial infection through the dentin tubules. Endo-perio lesion was a lesion involving the tissue destruction of dental pulp and periodontal tissue. Endodontic treatment must be performed in inflamed or necrotic abutment teeth under a bridge and successful healing can be achieved by performing adequate root canal treatment with great emphasis on disinfection and sealing of the root canal system. Intentional endodontic may be consi-

dered as a treatment option in preparing an abutment tooth for fixed final restoration.

## References

1. Manappallil JJ. Classification system for conventional crown and fixed partial denture failures. *J Prosthet Dent* 2008; 99: 293-8.
2. Pashley D. Pulpodentin complex. In: Hargreaves KM, Goodies HE (eds). *Seltzer and Bender's Dental Pulp*, 4<sup>th</sup> Ed. Carol Stream: Quintessence Publishing Co Inc, 2002; 63-93.
3. Kelleher M, Tiernan J. Crown and bridge restoration. *Clinical Risk* 2004;10: 60-4.
4. Didilescu A, Iliescu R, Rusu D. Current concepts on the relationship between pulpal and periodontal disease. *TMJ* 2008; 58(1-2): 98-103.
5. Reit C, Petersson K, Molven O. Diagnosis of pulpal and periapical disease. In: Bergenholtz G, Horsted-Bindslev P, Reit C (eds). *Textbook of endodontology*. West Sussex: Blackwell Publishing Ltd, 2003: 9-18.
6. Vera J, Trope M, Barnett F, Serota KS. Endodontic management of the endodontic-periodontal lesion. *Endodontic Practice* 2006: 40-4.
7. Rupf S, Kannengießer S, Merte K, Pfister W, Sigusch B, Eschrich K. Comparison of profiles of key periodontal pathogens in periodontium and endodontium. *Endodontol Dent Traumatol* 2000; 16 (6): 269-75.
8. Siqueira JF Jr. Endodontic infections: concepts, paradigms and perspective. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002; 94 (3): 281-93.
9. Matsuo T, Shirakami T, Ozaki K, Nakanishi T, Yumoto H, Ebisu S. An immunohistological study of the localization of bacteria invading root pulpal walls of teeth with periapical lesions. *J Endod* 2003; 29 (3): 194-200.
10. Walker MR. The pathogenesis and treatment of endo-perio lesion. *CPD Dentistry* 2001; 2(3): 91-5.
11. Lin S, Tilinger G, Zuckerman O. Endodontic-periodontic bifurcation lesions: a novel treatment option. *The Journal of Contemporary Dental Practice* 2008; 9(4): 1-8.
12. Gastal MT, Silveira CF, Silveira LF, Nova Cruz LE, Martos J. *Rev. Clin. Pesq. Odontol* 2008; 4(3): 201-6.
13. Carrotte P. Endodontics: Part 9. Calcium hydroxide, root resorption, endo-perio lesions. *British Dental Journal* 2004; 197 (12): 735-43.
14. European Society of Endodontology. Quality guidelines for endodontic treatment: consensus report of the European Society of Endodontology. *Int. Endod. J* 2006; 39: 921-30.