Biomechanical aspects of external root resorption in orthodontic therapy

Allan Abuabara

DDS, Specialist in Dental and Maxillofacial Radiology. Health Division, Joinville, Santa Catarina, Brazil

Correspondence: Dr. Abuabara Rua Quintino Bocaiúva, 102, apto 206, Joinville, SC, Brazil. 89204-300 E-mail: allan.abuabara@gmail.com

Received: 8-12-2006 Accepted: 4-05-2007

ndexed in:	
-Index Medicus / MEDLINE / Pub	Med
 EMBASE, Excerpta Medica 	
-SCOPUS	
-Indice Médico Español	
-IBECS	

ABSTRACT

External apical root resorption is a common phenomenon associated with orthodontic treatment. The factors relevant to root resorption can be divided into biological and mechanical factors. Some mechanical and biological factors might be associated with an increased or decreased risk of root resorption during orthodontic treatment. For mechanical factors, the extensive tooth movement, root torque and intrusive forces, movement type, orthodontic force magnitude, duration and type of force are involved. For biological factors, a genetic susceptibility, systemic disease, gender and medication intake have been demonstrated influence root resorption. Orthodontic therapy of patients with increased risk of root resorption should be carefully planned. Medical history, medication intake, family history, tooth agenesis, root morphology, oral health and habits must be considerate if we do not want jeopardize our patients by severe root resorption. To monitor apical root resorption the standard procedure is a radiographic examination after 6 months of treatment. In teeth with enhanced risk, a 3-month radiographic follow-up is recommended. The administration of anti-inflammatory drugs might suppress root resorption induced by orthodontic therapy, although none study was enough conclusive to indicate a protocol for patients with enhanced risk. In the event of multiple external root resorption, the diagnostic procedure should focus on the exclusion of the local factors and its associations (such as magnitude, duration and type of orthodontic force; periodontal disease; root form) that might lead to external root resorption. Systemic disorders associated with phosphorus-calcium metabolic alterations shall be suspected. This review searched the current knowledge of the mechanical and biological aspects of root resorption in orthodontic tooth movement.

E610-3

Key words: External root resorption, orthodontic tooth movement, risk factors.

CONCEPT

Orthodontic tooth movement is based on force-induced periodontal ligament and alveolar bone remodeling. Necrosis of periodontal ligament on the pressure side with formation of a cell-free hyaline zone followed osteoclast resorption of the neighbouring alveolar bone and bone apposition by osteoblasts on the tension side are the welldescribed typical histological characteristics of these processes. The resorption process of dental hard tissues seems to be triggered by the activity of cytokines as well as that of bone. Immune cells migrate out of the capillaries in the periodontal ligament and interact with locally residing cells by elaborating a large array of signal molecules (1). External root resorption (ERR) is a problem observed in association with orthodontic movement. The factors relevant to ERR can be divided into biological and mechanical factors (2). For mechanical factors, the extensive tooth movement, root torque and intrusive forces, movement type, orthodontic force magnitude, duration and type of force are involved. For biological factors, a genetic susceptibility, systemic factors (hormone unbalance), teeth agenesis, and medication intake have been demonstrated influence in root resorption (2-4). This review searched the current knowledge of the mechanical and biological aspects of root resorption in orthodontic tooth movement.

Abuabara A. Biomechanical aspects of external root resorption in

orthodontic therapy. Med Oral Patol Oral Cir Bucal. 2007 Dec 1;12(8):

© Medicina Oral S. L. C.I.F. B 96689336 - ISSN 1698-6946

EPIDEMIOLOGY

External apical root resorption is a common phenomenon associated with orthodontic treatment and can begin in the early leveling stages of orthodontic treatment. The prevalence among the researches varies widely. Means values ranging from 0.5 to 3 mm of root shortening during treatment have been reported (2). A frequency of severe apical root resorption of 5-18% has been reported (5). Killiany (6) reported root resorption of > 3 mm to occur at a frequency of 30%, with only 5% of treated individuals found to have > 5 mm of root resorption. Resorption occurs primarily in the maxillary anterior teeth, averaging over 1.4 mm. The worst resorption is seen in maxillary lateral incisors (7). Orthodontic patients with detectable root resorption during the first six months of active treatment are more likely to experience resorption in the following six-month period than those without (8).

One of the difficulties in assessing the causes of ERR is to separate the contribution made by genetic factors from those due to environmental factors, such as treatment and drugs. Family clustering of ERR has been suggested. Direct evidence for a genetic component was demonstrated with use of the sib-pair model and estimated the heritability to be 70% (3). No evidence was found for a sex or age difference in susceptibility.

PATHOGENISIS AND CLINICAL PICTURE

Genetic susceptibility

Interleukin-1 (IL-1) and tumour necrosis factor (TNF) are pro-inflammatory cytokines known to induce synthesis of various proteins that, in turn, elicit acute or chronic inflammation. Al-Qawasmi et al. (9) identified linkage disequilibrium between the IL-1B gene and ERR in orthodontically treated individuals. Another candidate gene for ERR is TNFRSF11A, which encodes the receptor activator of nuclear factor-kappa B (RANK), located in the same region as do familial expansile osteolysis and a form of familial Paget disease of bone (10). RANK is a member of the TNF-receptor superfamily and, together with the RANK ligand, mediates signaling leading to osteoclastogensis (11). Another candidate gene for ERR in orthodontic treatment is tissue non-specific alkaline phosphatase (TNSALP), the product of which plays an important role in mineralization and cementum formation. Mice lacking a functional TN-SALP gene have defective acellular cementum formation along the molar roots and delayed tooth eruption (12). Previous studies implicate TNF-alpha in bone remodeling in vitro and in vivo (13), supporting its inclusion as a candidate gene for EARR. TNF, found in human gingival sulcus, is elevated during tooth movement (14).

In two large studies of more than 860 cases, Sameshima & Sinclair (7, 15) showed that adult patients present more resorption than children only in mandibular anterior segment; Asian patients presented significantly less root resorption than white or Hispanic patients. Ngan et al. (16) investigated the genetic contribution to orthodontic root resorption. Monozygotic dizygotic twins were evaluated and the results indicated a genetic component to root resorption.

Influence of systemic diseases and medications intake

A study determined whether there is an association between excessive root resorption and immune system factors (17). The prevalence of root resorption found was 10.3%. Allergy, root morphology abnormality and asthma showed be highrisk factors for the development of excessive root resorption during orthodontic tooth movement. The modifying effect of several pharmacological agents on orthodontic ERR also has been examined. Among them, L-thyroxine has been shown to have an inhibitory effect and clinical application has been attempted (18). Similar effects have been shown for prednisolone in rats, in which low doses of corticosteroids during orthodontic treatment decrease root resorption (19). Studies have been published describing anti-inflammatory properties of tetracyclines (and their chemically modified analogues) unrelated to their antimicrobial effect. A significant reduction in the number of mononucleated cells on the root surface was observed. Such cells have been related to ERR (20).

The bone turnover can influence orthodontic treatment. High bone turnover (i.e. hyperthyroidism) can increase the amount of tooth movement compared with the normal or low bone turnover state and adult patients. Low bone turnover (i.e. hypothyroidism) can result more root resorption, suggesting that in subjects where a decreased bone turnover rate is expected, the risk of root resorption could be increased (21). Bisphosphonates, potent inhibitors of bone resorption, causes a significant dose-dependent inhibition of root resorption in rats after force application. These results prompt that a thorough case history regarding possible pathophysiological conditions influencing bone metabolism should be performed on an individual patient basis. In subjects where increased bone turnover rates are expected, the reactivation of the appliance could be performed more frequently. However, in patients where decreased bone turnover rates are expected, the reactivation should be carried out less frequently and the risk of root resorption should be carefully evaluated (21).

These results suggest that the administration of anti-inflammatory drugs might suppress root resorption induced by orthodontic therapy. Owman-Moll & Kurol (22) conducted a vast review investigating some factors that might be associated with orthodontically induced root resorption. A series of studies were analyzed and the table was reproduced (Table 1).

Local determinants

The tooth movement type, orthodontic force magnitude, duration and type of force also can alter the severity of root resorption. The intrusion of teeth causes about four times more root resorption than extrusion (23). Another study showed that the teeth activated with the superelastic wire (constant force) moved significantly more than the teeth with the steel wire (intermittent force). The depth of the resorption lacunae did not differ significantly between the groups, however, perimeter, area, and volume of the resorption lacunae on the teeth of the "superelastic group" were **Table 1.** Some local and systemic factors which might be associated with the risk of root resorption during orthodontic treatment. Increased (+), decreased (-) or (+/-) when the results of the studies are conflicting. Table modified of Owman-Moll & Kurol (22).

Factors	Influence
Dental Health	
Root morphology, i.e. pipette shaped, blunt,	
abrupt root deflection, narrow root,	+
taurodontism	
Tooth agenesis	+
Pre-treatment root resorption	+
Endodontically treated teeth	+/-
Oral Health	
Gingivitis, periodontal disease	+
Medical Health	
Allergies, asthma, arthritis, hypothyroidism	+
and diabetes	
Phosphorus-calcium metabolic alterations	+
Habits	
Nail-biting, lip/tongue dysfunction	+
Medication	
Aspirin	-
Tetracyclines (and their chemically modified	_
analogues)	

140 per cent greater than on the teeth of the "steel group" (24). Teeth with abnormal root shape (pipette, pointed, or dilacerated) (7), long, narrow, and deviated roots (25) appear be more susceptible to suffer ERR.

Root resorption in hypofunctionally non-occluding teeth during tooth movement also was evaluated (26). Root resorption was significantly greater in these teeth than in those with a normal periodontium. The results suggest that orthodontic movement of non-occluding teeth should be performed with caution. Since the hypofunctional periodontium exhibited progressive atrophic changes in all functional structures, this might have accelerated the root destruction resulting from the mechanical stress of orthodontic force (26). Also it was demonstrated that periodontal disease, habits (nail-biting, lip/tongue dysfunction) (22) and multiple aplasia (four or more teeth) (5), in particular in teeth with an abnormal root form, increases the risk of ERR during orthodontic treatment.

MANAGEMENT AND PROGNOSIS

The diagnosis is uncertain during the first months of treatment. After 5–6 months a reliable radiographic diagnosis of apical root resorption can be performed (27). To evaluate root shape and position most clinicians order panoramic or periapical radiographs in addition to the cephalometric radiograph. Periapical radiographs are an important part of orthodontic records. They are useful to compare pretreatment and post-treatment root resorption. Maxillary incisors are affected more frequently and to a greater degree than the rest of the teeth during active treatment (7). Also, root resorption of the upper incisors during the initial 6-9 months of treatment with fixed appliances gives a high risk for continued resorption during the subsequent treatment. Therefore, it would be prudent to take periapical radiographs periodically during treatment (8).

Although there are advantages of the panoramic film (less radiation exposure, less patient chair time, less operator time, and better patient cooperation) root shape is much harder to assess on panoramic films. In cases where the apices are obscured or other factors are present that might suggest higher risk for root resorption or vertical bone loss, periapical films should be ordered. Digital images have more advantages comparing to the conventional radiographs due to a higher quality, reduction in radiation of approximately 70% and no chemicals. Moreover brightness, contrast and saturation can be modified on the digital images which can make ease the identification of anatomic tissues and pathologies (28). When root resorption is detected during active treatment, a decision must be made as to whether to continue, modify or discontinue the treatment. Extremely heavy forces should be avoided, since they have been shown to produce greater resorption activity.

The potential benefit of treating young teeth was investigated by Mavragani et al. (29). Roots that were incompletely developed before treatment reached a significantly greater length than those that were fully developed at the start of treatment. The results of this study show a definite advantage for younger teeth with regard to post-treatment root length. This finding may influence treatment planning strategy.

COMPLEMENTARY EXPLORATION

Currently, there are no reliable markers to predict either which patients will develop ERR or the severity of ERR following orthodontic tooth movement. The association of a specific IL-1B allele and ERR, which accounts for approximately 15% of the total ERR variation seen in orthodontic patients, has emerged as a potential genetic marker (9). Multigenic nature, such as IL-1B gene, TNF family gene and TNSALP, appear to influence ERR, although to what extent is not yet known.

After 3 months, apical root resorption can be detected in only a few teeth. The number increase significantly after 6 months. To monitor apical root resorption the standard procedure is a radiographic examination after 6 months of treatment. In teeth with enhanced risk, such as teeth with blunt and pipette-shaped apices, a 3-month radiographic control follow-up is recommended (27).

Orthodontic therapy of patients with increased risk of root resorption should be carefully planned. Habits, medical history, medication intake (principally diseases and drugs that might affect bone turnover), family history, tooth agenesis, root morphology, oral health must be considerate if we do not want jeopardize our patients by severe root resorption. Results showed potential advantage for younger teeth with regard to post-treatment root length. The administration of anti-inflammatory drugs might suppress root resorption induced by orthodontic therapy, although none study was enough conclusive to indicate a protocol for patients with enhanced risk.

In the event of multiple ERR, the diagnostic procedure should focus on the exclusion of the local factors and its associations (such as magnitude, duration and type of orthodontic force; periodontal disease; root form) that might lead to ERR. Systemic disorders associated with phosphorus-calcium metabolic alterations such as hypothyroidism, hyper- or hypoparathyroidism, hypophosphatemia, sarcoidosis (as an occasional cause of hypercalciuria), Paget's disease, osteogenesis imperfecta, calciuria and a history of nephrolithiasis shall be suspected (30).

REFERENCES

1. Jäger A, Zhang D, Kawarizadeh A, Tolba R, Braumann B, Lossdörfer S, et al. Soluble cytokine receptor treatment in experimental orthodontic tooth movement in the rat. Eur J Orthod. 2005 Feb;27(1):1-11.

2. Brezniak N, Wasserstein A. Root resorption after orthodontic treatment: Part 2. Literature review. Am J Orthod Dentofacial Orthop. 1993 Feb;103(2):138-46.

3. Harris EF, Kineret SE, Tolley EA. A heritable component for external apical root resorption in patients treated orthodontically. Am J Orthod Dentofacial Orthop. 1997 Mar;111(3):301-9.

4. Levander E, Malmgren O, Stenback K. Apical root resorption during orthodontic treatment of patients with multiple aplasia: a study of maxillary incisors. Eur J Orthod. 1998 Aug;20(4):427-34.

5. Mirabella AD, Artun J. Prevalence and severity of apical root resorption of maxillary anterior teeth in adult orthodontic patients. Eur J Orthod. 1995 Apr;17(2):93-9.

 Killiany DM. Root resorption caused by orthodontic treatment: an evidence-based review of literature. Semin Orthod. 1999 Jun;5(2):128-33.

7. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part I. Diagnostic factors. Am J Orthod Dentofacial Orthop. 2001 May;119(5):505-10.

8. Artun J, Smale I, Behbehani F, Doppel D, Van't Hof M, Kuijpers-Jagtman AM. Apical root resorption six and 12 months after initiation of fixed orthodontic appliance therapy. Angle Orthod. 2005 Nov;75(6):919-26. 9. Al-Qawasmi RA, Hartsfield JK Jr, Everett ET, Flury L, Liu L, Foroud TM, et al. Genetic predisposition to external apical root resorption in orthodontic patients: linkage of chromosome-18 marker. J Dent Res. 2003 May;82(5):356-60.

10. Hughes AE, Ralston SH, Marken J, Bell C, MacPherson H, Wallace RG, et al. Mutations in TNFRSF11A, affecting the signal peptide of RANK, cause familial expansile osteolysis. Nat Genet. 2000 Jan;24(1):45-8.

11. Nakagawa N, Kinosaki M, Yamaguchi K, Shima N, Yasuda H, Yano K, et al. RANK is the essential signaling receptor for osteoclast differentiation factor in osteoclastogenesis. Biochem Biophys Res Commun. 1998 Dec 18:253(2):395-400.

12. Beertsen W, VandenBos T, Everts V. Root development in mice lacking functional tissue non-specific alkaline phosphatase gene: inhibition of acellular cementum formation. J Dent Res. 1999 Jun;78(6):1221-9.

13. Le J, Vilcek J. Tumor necrosis factor and interleukin 1: cytokines with multiple overlapping biological activities. Lab Invest. 1987 Mar;56(3):234-48.

14. Lowney JJ, Norton LA, Shafer DM, Rossomando EF. Orthodontic forces increase tumor necrosis factor alpha in the human gingival sulcus. Am J Orthod Dentofacial Orthop. 1995 Nov;108(5):519-24.

15. Sameshima GT, Sinclair PM. Predicting and preventing root resorption: Part II. Treatment factors. Am J Orthod Dentofacial Orthop. 2001 May;119(5):511-5.

16. Ngan DC, Kharbanda OP, Byloff FK, Darendeliler MA. The genetic contribution to orthodontic root resorption: a retrospective twin study. Aust Orthod J. 2004 May;20(1):1-9.

17. Nishioka M, Ioi H, Nakata S, Nakasima A, Counts A. Root resorption and immune system factors in the Japanese. Angle Orthod. 2006 Jan;76(1):103-8.

18. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. J Clin Pediatr Dent. 1999 Spring;23(3):259-64.

19. Ong CK, Walsh LJ, Harbrow D, Taverne AA, Symons AL. Orthodontic tooth movement in the prednisolone-treated rat. Angle Orthod. 2000 Apr;70(2):118-25.

20. Mavragani M, Brudvik P, Selvig KA. Orthodontically induced root and alveolar bone resorption: inhibitory effect of systemic doxycycline administration in rats. Eur J Orthod. 2005 Jun;27(3):215-25.

21. Verna C, Dalstra M, Melsen B. Bone turnover rate in rats does not influence root resorption induced by orthodontic treatment. Eur J Orthod. 2003 Aug;25(4):359-63.

22. Owman-Moll P, Kurol J. Root resorption after orthodontic treatment in high- and low-risk patients: analysis of allergy as a possible predisposing factor. Eur J Orthod. 2000 Dec;22(6):657-63.

23. Han G, Huang S, Von den Hoff JW, Zeng X, Kuijpers-Jagtman AM. Root resorption after orthodontic intrusion and extrusion: an intraindividual study. Angle Orthod. 2005 Nov;75(6):912-8.

24. Weiland F. Constant versus dissipating forces in orthodontics: the effect on initial tooth movement and root resorption. Eur J Orthod. 2003 Aug;25(4):335-42.

25. Smale I, Artun J, Behbehani F, Doppel D, Van't Hof M, Kuijpers-Jagtman AM. Apical root resorption 6 months after initiation of fixed orthodontic appliance therapy. Am J Orthod Dentofacial Orthop. 2005 Jul;128(1):57-67.

26. Sringkarnboriboon S, Matsumoto Y, Soma K. Root resorption related to hypofunctional periodontium in experimental tooth movement. J Dent Res. 2003 Jun;82(6):486-90.

27. Levander E, Bajka R, Malmgren O. Early radiographic diagnosis of apical root resorption during orthodontic treatment: a study of maxillary incisors. Eur J Orthod. 1998 Feb;20(1):57-63.

28. Paredes V, Gandia JL, Cibrián R. Digital diagnosis records in orthodontics. An overview. Med Oral Patol Oral Cir Bucal. 2006 Jan 1;11(1): E88-93.

29. Mavragani M, Bøe OE, Wisth PJ, Selvig KA. Changes in root length during orthodontic treatment: advantages for immature teeth. Eur J Orthod. 2002 Feb;24(1):91-7.

30. Llena-Puy MC, Amengual-Lorenzo J, Forner-Navarro L. Idiopathic external root resorption associated to hypercalciuria. Med Oral. 2002 May-Jun;7(3):192-9.