Original Article

Pulp Vitality in Teeth Suffering Trauma during Orthodontic Therapy

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ABSTRACT

Objective: To examine pulpal vitality in teeth suffering dental trauma during orthodontic therapy with fixed appliances.

Materials and Methods: Pulpal condition was evaluated in 59 teeth that had suffered dental trauma during orthodontic treatment (TO-group), in 800 orthodontically treated teeth without previous dental trauma (O-group), and in 193 orthodontically untreated teeth with previous dental trauma (T-group). Pulpal vitality was examined clinically and with radiographs. Degree of pulp obliteration was rated as absent, partial, or total. All teeth in the TO-group showed a positive sensibility test prior to resumption of orthodontic therapy.

Results: Teeth in the TO-group revealed a significantly higher frequency of pulp necrosis than teeth in the O-group or teeth in the T-group ($P < .001$, respectively). In the TO-group, teeth with extrusive or lateral luxation ($P = .031$) and teeth with intrusive luxation ($P = .015$) injuries showed a significantly higher rate of pulp necrosis than teeth with fracture of enamel. In addition, teeth with total pulp obliteration showed a significantly higher frequency of pulp necrosis than teeth without pulp obliteration ($P = .013$).

Conclusion: Teeth with severe periodontal injury during orthodontic therapy and subsequent total pulp obliteration have an increased risk of pulp necrosis during additional orthodontic treatment stages. (*Angle Orthod.* 2009;79:166–171.)

KEY WORDS: Dental trauma; Orthodontic treatment; Pulp necrosis; Pulp obliteration

INTRODUCTION

Epidemiologic studies indicate that traumatic dental injuries are a serious dental health problem.[1–5] The vast majority of dental injuries involve the upper incisors, and Class II division 1 malocclusion with increased overjet and inadequate lip coverage have been identified as possible predisposing factors.[6–13] Because these predisposing factors are frequent findings in patients with an orthodontic treatment need, a high prevalence of traumatized permanent incisors is found in candidates for orthodontic therapy.[12,13] Therefore, orthodontic movement of previously traumatized teeth represents a major problem in routine orthodontic treatment.

Only a few studies have analyzed the influence of orthodontic treatment on pulpal vitality of previously traumatized teeth.[14–16] However, to our knowledge, no previous study has examined pulpal vitality in teeth suffering dental trauma during orthodontic treatment. The aim of the present study was, therefore, to examine pulpal vitality in permanent upper incisors suffering dental trauma during orthodontic therapy with fixed appliances. A further objective was to assess the influence of different parameters, such as type of trauma, type of incisor, orthodontic treatment time before and after dental trauma, archwire exchanges per month during orthodontic treatment after trauma, and degree of pulp obliteration after trauma.

MATERIALS AND METHODS

In this retrospective study, patients who had suffered dental trauma to their permanent upper incisors during orthodontic treatment were compared with orthodontically treated patients without dental trauma, and with patients with previous dental trauma to the maxillary incisors and no subsequent orthodontic treat-

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Table 1. Distribution of Traumatized Teeth in the TO-Group According to Type of Trauma and Type of Incisor

Type of Trauma	Number of Patients	Number of Traumatized Teeth	Type of Incisor	
			Central	Lateral
Enamel fracture (n = 13)	8	13	8	5
Enamel-dentin fracture (n = 15)	11	15	10	5
Subluxation (n = 8)	5	8	5	3
Extrusive or lateral luxation (n = 11)	10	11	9	2
Intrusive luxation (n = 12)	12	12	11	1
Total (N = 59)	46	59	43	16

ment. All patients had been treated in three private practices between the years 1990 and 2007. Dental traumas were classified into categories according to hard tissue or periodontal tissue injuries.[4] In cases of more than one type of injury to the same tooth, only the most serious one was used to categorize the tooth.

Patients Suffering Dental Trauma During Orthodontic Treatment

This group (the TO-group) consisted of 46 patients (18 female and 28 male) with 59 traumatized permanent maxillary incisors, 43 central incisors, and 16 lateral incisors. All patients had sustained dental trauma to their permanent upper incisors during orthodontic treatment with fixed appliances. Thirty-nine patients had one tooth affected. Three patients had two, and two had three injured teeth. In two patients, all four permanent upper incisors had been traumatized. Traumatized teeth belonged to the following categories: fracture of enamel (n = 13), fracture of enamel-dentin (n = 15), subluxation (n = 8), extrusive (n = 2) or lateral luxation (n = 9), and intrusive luxation (n = 12) (Table 1). For further statistical analysis, teeth with extrusive or lateral luxations were treated as one group.

All patients were treated with a preadjusted appliance with 0.018 inch slot brackets. The archwire sequence was usually 0.012 inch, 0.016 inch, 0.016 × 0.022 inch, and 0.017 × 0.025 inch nickel-titanium, followed by a 0.016 × 0.022 inch and a 0.017 × 0.025 inch stainless steel wire. In cases of hard tissue (fracture of enamel and fracture of enamel-dentin) or slight periodontal injury (subluxation), orthodontic movement of the traumatized teeth was interrupted for 2 to 3 months. In cases with extrusive or lateral luxations, the traumatized teeth and associated fractures of the socket wall were repositioned carefully, and fixation was performed with a 0.016 × 0.022 inch stainless steel wire. This connected the traumatized tooth with both adjacent teeth and was left for 3 weeks. In cases with intrusive luxation, spontaneous re-eruption was awaited for 3 to 4 weeks. If no evidence of re-eruption was noted at that time, orthodontic extrusion was carried out by elastic traction. Afterward, fixation was carried out with a 0.016 × 0.022 inch stainless steel wire for 3 to 4 weeks. In all cases with severe periodontal injury, orthodontic movement was interrupted for 5 to 6 months.[17] The mean age at the beginning of orthodontic treatment was 11.2 years (range, 9.5–16.7 years). Orthodontic treatment time before dental trauma averaged 13.9 months (range, 7–24 months), and treatment time after trauma averaged 12.7 months (range, 6–24 months). The mean follow-up period after termination of orthodontic treatment was 9 months (range, 6–12 months). Additional inclusion criteria were as follows:

- Positive sensibility testing of the traumatized teeth before resumption of orthodontic treatment
- No clinical or radiologic signs and no history of dental trauma before onset of orthodontic treatment
- A minimum treatment period of 6 months after trauma
- Exchange of at least two archwires prior to termination of orthodontic treatment
- No extreme intrusive, extrusive, or lateral tooth movements
- A follow-up period of at least 6 months after termination of active orthodontic treatment

Orthodontically Treated Patients Without Dental Trauma

This group (the O-group) consisted of 200 randomly selected patients (60 female and 140 male) with 800 permanent upper incisors. All patients in this group had also been treated with a preadjusted appliance with 0.018 inch slot brackets and the same archwire sequence as patients in the TO-group. The mean age before onset of orthodontic treatment was 12.7 years (range, 9.7–17.5 years). The total treatment time with fixed appliances averaged 23.7 months (range, 11.6–31.7 months). The mean follow-up time after termination of orthodontic treatment was 2.1 years (range, 0.5–4.2 years). Additional inclusion criteria for this group consisted of (1) no clinical or radiologic signs and no history of dental trauma before, during, or after orthodontic treatment, (2) positive sensibility testing before onset of orthodontic treatment, and (3) a follow-

up period of at least 6 months after termination of orthodontic treatment.

Orthodontically Untreated Patients With Dental Trauma

This group (the T-group) consisted of 173 patients and was recruited from a Private Practice in Oral and Maxillofacial Surgery. The patients (61 female and 112 male) showed a total of 193 traumatized permanent upper incisors, 146 central incisors, and 47 lateral incisors. One tooth was affected in 160 patients, and eight patients had two teeth affected. Three patients had three injured teeth, and in two patients, all four permanent upper incisors had been traumatized. Enamel fracture had occurred in 36 teeth, enamel-dentin fracture in 32, subluxation in 31, extrusive luxation in 30, lateral luxation in 33, and intrusive luxation in 31 teeth. The mean age of patients at the time of trauma was 9.3 years (range, 6.6–16.4 years). The mean follow-up time after trauma was 5.4 years (range, 3.0–10.9 years). Additional inclusion criteria for this group consisted of (1) positive sensibility testing during the first 6 months after dental trauma, (2) minimum follow-up period of 3 years after dental trauma, (3) no subsequent grinding or filling therapy after trauma, and (4) no history of multiple dental trauma.

Evaluation of Pulpal Condition

Pulpal vitality was examined clinically and with radiographs in all three groups during the final follow-up and in the TO- and O-groups during orthodontic treatment in case of an indication of pulp necrosis. The clinical examination included rating of crown color (normal or grayish) and sensibility testing with a cryogenic spray. The radiologic examination consisted of standardized periapical radiographs and contributed to the assessment of periapical radiolucencies. The following clinical and radiologic criteria were used to define pulp necrosis: (1) gray color changes in the crown, (2) loss of pulpal sensibility, and (3) periapical radiolucency. All three diagnoses or loss of pulpal sensibility in combination with gray color changes in the crown or periapical radiolucency were considered necessary before the diagnosis was made.[18]

Pulp obliteration was evaluated on the final periapical radiographs in the TO-group. Degree of pulp obliteration was rated according to Jacobsen and Kerekes[19] as total obliteration (pulp chamber and root canal hardly or not discernible) or partial obliteration (pulp chamber not discernible and root canal markedly narrowed but clearly visible). According to the presence and degree of pulp obliteration, the teeth were divided into three different categories: (1) traumatized teeth without pulp obliteration, (2) traumatized teeth with partial obliteration, and (3) traumatized teeth with total pulp obliteration.

Statistical Analysis

The Chi-square test was used to detect significant intergroup differences regarding the frequency of pulp necrosis. The Fisher exact test was carried out to determine significant relationships between pulp necrosis and type of trauma, type of incisor, and degree of pulp obliteration. In the TO-group, the number of archwire exchanges per month was calculated for the treatment period after dental trauma by dividing the number of archwire exchanges through the number of treatment months after trauma. Afterward, a two-sample *t*-test was used to determine significant differences with respect to treatment time before trauma, treatment time after trauma, and archwire exchanges per month. The significance level was set at $\alpha = .05$. Statistical analysis was performed with the Statistical Package for the Social Sciences (SPSS), version 11.0 (SPSS Inc, Chicago, Ill).

Error of the Method

Reproducibility of radiographic assessment of pulp obliteration and periapical radiolucencies was determined through double ratings of 100 randomly selected periapical radiographs taken during the final examination. Both ratings were performed within a 4-week interval by two examiners. Afterward, interexaminer agreement was calculated with the use of weighted kappa statistics. A very good interexaminer agreement was determined for the assessment of pulp obliteration ($\kappa = 0.85$) and periapical radiolucencies ($\kappa = 0.89$).

RESULTS

Pulp necrosis was identified in 18.6% of teeth in the TO-group (n = 11), in 0.3% of teeth in the O-group (n = 2), and in 1.6% of teeth in the T-group (n = 3). No patient with more than one tooth with pulp necrosis was included in any of the investigated groups. Teeth in the TO-group revealed a significantly higher frequency of pulp necrosis than teeth in the O-group or teeth in the T-group ($P < .001$, respectively). No significant differences were detected between the O-group and the T-group.

In the TO-group, pulp necrosis was observed in 6.7% of teeth with fracture of enamel-dentin (n = 1), in 12.5% of teeth with subluxation (n = 1), in 36.4% of teeth with extrusive or lateral luxation (n = 4), and in 41.7% of teeth with intrusive luxation (n = 5). No cases of pulp necrosis were detected in teeth with fracture of enamel. Teeth with extrusive or lateral lux-

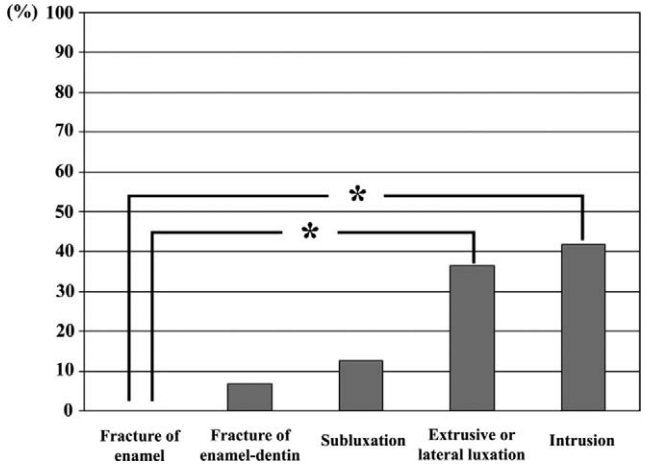

Figure 1. Frequency of pulp necrosis in the TO-group according to type of trauma. *$P < .05$.

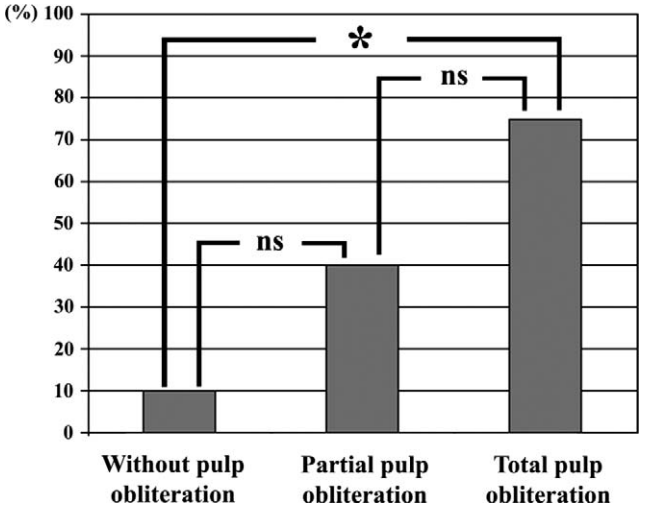

Figure 2. Frequency of pulp necrosis according to degree of pulp obliteration in teeth with serious periodontal injuries in the TO-group. *$P < .05$; ns indicates not significant.

ation ($P = .031$) and teeth with intrusive luxation ($P = .015$) showed a significantly higher rate of pulp necrosis than teeth with fracture of enamel. No significant differences were noted between the remaining types of trauma (Figure 1).

For further analysis of the investigated parameters, teeth with severe periodontal injury (extrusive, lateral, and intrusive luxation) were treated as one group (n = 23). Pulp necrosis was found in 40.0% of central (n = 8) and in 33.3% of lateral incisors (n = 1). However, this difference was not statistically significant. With respect to the treatment period before dental trauma, no significant differences were found between teeth that developed pulp necrosis and those with regular pulpal condition. In contrast, teeth with pulp necrosis revealed a significantly lower treatment time after trauma (7.4 months; range, 6–9 months) ($P = .002$) and a significantly higher average number of archwire exchanges per month (0.47; range, 0.33–0.57) ($P < .001$) than teeth without pulp necrosis (13.9 months; range, 6–21 months; and 0.26; range, 0.11–0.44). Absence of pulp obliteration at the final follow-up was noted in 43.5% of the teeth with severe periodontal injury (n = 10). Partial obliteration was seen in 21.7% (n = 5), and total obliteration in 34.8% of the sample (n = 8). Teeth with total pulp obliteration showed a significantly higher frequency of pulp necrosis than teeth without pulp obliteration ($P = .013$) (Figure 2).

DISCUSSION

Teeth that suffered a severe periodontal injury during orthodontic treatment revealed a significantly higher rate of pulp necrosis during later orthodontic treatment stages than teeth with only slight periodontal or hard tissue injury. All teeth in the TO-group showed a positive reaction to sensibility testing prior to resumption of orthodontic treatment, implying an adequate vascular supply to the pulp. However, previous investigations showed that orthodontic tooth movement can affect the blood supply to the dental pulp, and that an initial decrease in pulpal blood flow is usually followed by a reactive hyperemia that compensates for the lack of tissue perfusion.[20–22] Therefore, it might be concluded that in teeth with severe periodontal injury, the capacity of the blood vessels supplying the pulp was insufficient for maintenance of an adequate pulpal blood flow during further orthodontic treatment. A possible explanation could be that severe periodontal injury might cause permanent damage to and reduction of apical vessels, which might render these teeth more prone to pulp necrosis during orthodontic tooth movement.

The results of the present study also suggest that teeth with total pulp obliteration after trauma are more susceptible to pulp necrosis than traumatized teeth without obliteration. Pulp obliteration is caused by progressive hard tissue apposition along the pulp chamber that gradually diminishes the pulpal lumen.[23] Therefore, it is conceivable that this hard tissue formation also leads to progressive compression and finally to constriction of the existing pulpal vessels, resulting in an impaired blood supply to traumatized teeth with total pulp obliteration. In addition, progressive hard tissue apposition might lead to constriction of the apical foramen, and thus to compression of the neurovascular bundle. This may cause strangulation or even rupture of the apical vessels during orthodontic tooth movement. In contrast, partial pulp obliteration revealed no negative effect on pulpal vitality. Compared with total pulp obliteration, partial obliteration involves primarily the pulp chamber, whereas the root

canal and probably also the apical foramen show only a certain narrowing. Thus, only limited constriction of the apical vessels might be concluded in teeth with partial obliteration, and this might allow the pulpal circulatory system of these teeth to function adequately and to maintain sufficient pulpal perfusion during orthodontic treatment.

An interesting finding was the significant correlation between pulp necrosis and average number of archwire exchanges per month. A possible explanation might be that frequent archwire exchanges within a short period caused additional compression of the apical vessels. Because pulp canal obliteration represents the response of a vital pulp to severe injury to its neurovascular supply,[18] it is also conceivable that frequent archwire exchanges have caused progression of pulp obliteration, finally resulting in pulp necrosis during additional orthodontic treatment. Several investigations have already identified pulp obliteration as a potential side effect of orthodontic treatment.[24–27] In addition, a previous study reported an increased incidence of pulp obliteration after orthodontic band fixation of traumatized teeth, and it was assumed that band application may have caused displacement of the root with compression of the apical vessels.[28]

CONCLUSIONS

- Teeth with severe periodontal injury during orthodontic therapy and subsequent total pulp obliteration have an increased risk of pulp necrosis during later orthodontic treatment stages.
- The pulpal condition should be monitored frequently by intraoral radiographs after resumption of orthodontic treatment, and in cases of progressive pulp obliteration, orthodontic movement of these teeth should be terminated, or at least limited to a minimum.

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