Interactions between Orthodontic Treatment and Gingival Tissue

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In recent years, as the number of adults seeking orthodontic treatment has increased, so too has the number of periodontal tissue problems, particularly regarding the impact on periodontal tissue of receiving orthodontic treatment. Orthodontic treatment improves the occlusion and appearance of teeth by moving the teeth appropriately. These movements have a significant impact on the interactions between the teeth and periodontal tissues. Orthodontic treatment can also recover tooth alignment for patients with tooth displacement caused by periodontitis; however, orthodontic treatment also often has adverse effects on periodontal soft tissue, such as gingivitis, gingival enlargement and gingival recession. The purpose of this review is to summarise the current evidence and solid knowledge of periodontal soft tissue problems in orthodontic treatment and outline some prevention strategies.

Key words: *orthodontic treatment, periodontal tissue health, principle of treatment*
Chin J Dent Res 2023;26(1):11–18; doi: 10.3290/j.cjdr.b3978667

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This study was funded by the Natural Science Foundation of Xinjiang Uygur Autonomous Region (grant no. 2022D01C253); Open Project of Hubei Province Key Laboratory of Oral and Maxillofacial Development and Regeneration (grant no. 2022kqhm008).

Orthodontic treatment can help patients with malocclusion to obtain regular tooth alignment, normal oral physiological function, a coordinated face profile, healthy dentomaxillofacial development, and even improved quality of life[1,2]. The arrangement of teeth achieved is also conducive to oral hygiene and reduces the risk of caries lesions, gingivitis and periodontitis. Moreover, normal occlusion can improve the function of the stomatognathic system, increase masticatory efficiency and swallowing ability, improve pronunciation and prevent further trauma of the temporomandibular joint (TMJ)[3,4]. Since orthodontic treatment can change the facial features and appearance directly, evidence indicates that it could enhance patients' confidence and reduce psychological disorders[5,6]. Furthermore, if performed at a specific time, orthodontic treatment can promote the development of soft and hard dentomaxillofacial tissues[7,8]; however, in addition to the therapeutic benefits of orthodontic treatment, the risks it can pose to the dental pulp, TMJ, occlusion and periodontal tissue must not be overlooked, The principle of orthodontic movement is to move the teeth by applying directional external force followed by altering the periodontal tissues, which results in two requirements[9,10]: firstly, the orthodontic process should not result in destruction of an excessive amount of periodontal tissue or any irreversible damage, and secondly, the periodontal tissues should remain

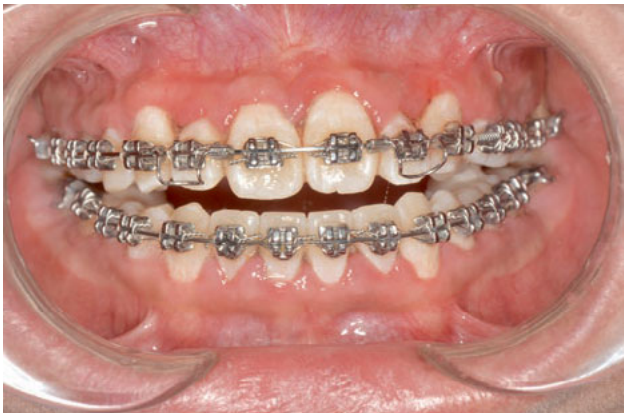

Fig 1 Gingivitis in the anterior region during orthodontic treatment.

stable after the teeth have been moved into their new position. Orthodontic movements are achieved through the reconstruction of periodontal tissue[11]; however, the conditions of the jaws and alveolar bone, the tissue structure characteristics and the response to external stimuli remain variable, and changes in the periodontal soft and hard tissues are not detected or corrected in a timely manner, thus continuing the problem[12]. Periodontal soft tissue abnormalities during orthodontic treatment mainly include gingivitis, gingival enlargement, gingival recession and gingival invagination[13,14]. These problems may affect patients' oral hygiene, cause tooth sensitivity, impact gum aesthetics and even affect the results and success of orthodontic treatment[15,16]. This article reviews the causes, prevention and treatment of periodontal soft tissue abnormalities during orthodontic treatment, aiming to draw attention and consideration to the colleagues.

Orthodontic treatment and gingivitis

The main cause of periodontal inflammation in orthodontic patients is chronic gingivitis (Fig 1), which is caused by the accumulation of dental plaque near the gingival margin. Wearing orthodontic devices affects patients' self-cleaning and increases the difficulty of local plaque control[17], which affects patients' personal oral hygiene maintenance and oral flora composition[18]. A previous study found that the detection rate of *Actinobacillus actinomycetemcomitans* in children with orthodontic devices is 85%, which is significantly higher than that in children without such devices (15%)[19]. It was reported that after 3 months, the detection rate of *Porphyromonas gingivalis* and *Fusobacterium nucleatum* increased significantly, with the Plaque Index (PI), Gingival Index (GI) and bleeding on probing (BOP) being significantly higher in individuals having received orthodontic treatment[20]. A systematic evaluation conducted by Verrusio et al[21] noted an increase in periodontal parameters after orthodontic treatment, indicating that it influenced the accumulation and composition of the subgingival microbiota and subsequently induced more inflammation and higher BOP; however, orthodontic treatment is not the direct cause of gingivitis, and good self-administered plaque control can reduce the probability of gingivitis during orthodontic treatment significantly. Thus, oral hygiene should be administered before orthodontic treatment and repeated during every visit. In addition, regular tooth brushing is the primary way of controlling dental plaque.

Prevention and treatment of gingivitis

The most effective measure for preventing gingivitis is to offer chairside oral health education and manage existing periodontal disease before orthodontic treatment[22]. At present, it is generally believed that it is not sufficient for patients with periodontal disease to only carry out self-administered plaque control during the maintenance period. Instead, professional mechanical plaque control should be performed regularly during the orthodontic process[23], and interventions should be carried out in areas of the dentition that are often neglected and difficult to clean. In addition, the design and manufacture of orthodontic devices should follow the principles of facilitating oral plaque control and avoiding gingival irritation[24].

If orthodontic patients have gingivitis, they should be transferred immediately to the periodontal department for systematic periodontal examination and treatment. The treatment should remove plaque and calculus thoroughly and eliminate factors that may cause local plaque retention and gingival irritation[25]. For patients with severe gingivitis, local medication can be used appropriately. During this period, orthodontic force should be stopped until the condition of the gingiva has been recovered entirely.

For orthodontic patients with excessive plaque deposition and severe gingival inflammation, it is necessary to remove the orthodontic device completely for periodontal treatment until the inflammation is under control[26]. This process may lead to a relapse of the orthodontic effect and longer treatment time. Thus, some patients and orthodontists are reluctant to remove the orthodontic device for periodontal treatment. Sometimes, airborne-particle abrasion under the guidance of a stain can remove plaque more effectively, improve the efficiency of inflammation control, and

reduce the removal rate of fixed orthodontic devices to a certain extent[27].

Orthodontic treatment and gingival hypertrophy

Gingival enlargement is another problem that is common in orthodontic patients (Fig 2), especially adolescents[28]. In adolescents with poor oral hygiene, local stimulation of orthodontic devices and changes in sexual hormone levels often lead to inflammatory gingival swelling, accompanied by compensatory proliferation of cells and collagen fibres. The manifestations are globular or nodular enlargement of the gingival papilla, pink colour of the gingival margin, thickening and difficulty in bleeding on probing. Histologically, the basal layer and mesothelium showed excessive growth and a lack of chronic inflammatory exudation. In severe cases, gingival inflammatory hyperplasia may cover the tooth surface or orthodontic appliances, which can affect the progress of orthodontic treatment[29].

Analysis of related elements between orthodontic treatment and gingival hypertrophy

Gingival hypertrophy usually occurs 1 to 2 months after orthodontic treatment[30]. Many factors can aggravate gingival inflammation and cause gingival fibrosis and hypertrophy, such as reduced plaque control, chemical and physical stimulation of adhesives, mechanical band stimulation and food impaction.

Although plaque is often believed to be the leading cause of gingival inflammation and hypertrophy, it has been reported that gingival hypertrophy also occurs in patients with good oral hygiene, suggesting that orthodontic force and periodontal remodelling may also be associated with gingival hypertrophy[31]. For example, Surlin et al[32] found that out of 22 fixed orthodontic patients, 15 developed gingival hypertrophy. The level of matrix metalloproteinase (MMP)-8 in these patients was significantly higher than that in the standard orthodontic treatment group (no periodontal lesions in the latter group)[32]. The authors also found a positive correlation between the degree of gingival hypertrophy and the expression of MMP-9/IV collagen in gingival tissue in orthodontic patients without inflammation[29]. Based on these results, they believe that the increase in MMP level caused by orthodontic force may be one of the causes of gingival hypertrophy[29,32], but whether pure orthodontic power is a direct factor in gingival hypertrophy still needs to be explored further.

In addition, it was shown that a continuous low concentration of nickel ion stimulation in some orthodontic devices is an essential cause of gingival hypertrophy in orthodontic treatment[33]. Nickel ions may stimulate the growth of epithelial cells and the proliferation of keratinocytes by inducing T-lymphocytes to produce interferon and interleukin (IL)-2, IL-5, and IL-10, which may lead to gingival hypertrophy. Nickel ion release may be a time-dependent type IV allergic reaction[33]. Thus, it is necessary to know whether the patient has a history of nickel allergy to avoid the occurrence of gingival hypertrophy during orthodontic treatment.

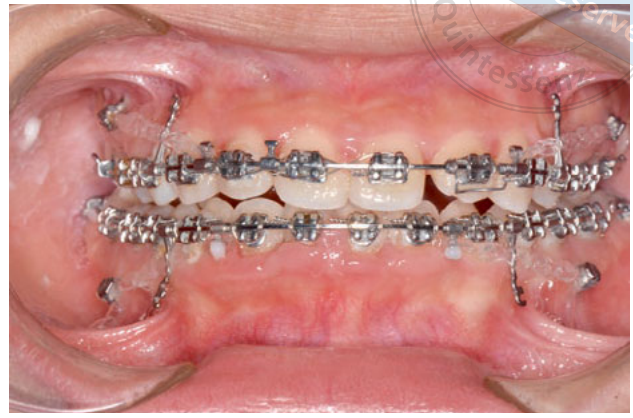

Fig 2 Gingival hypertrophy in the site of the mandibular canines during orthodontic treatment.

Prevention and treatment of gingival hypertrophy

To prevent gingival hypertrophy, besides reasonable plaque control, careful diagnosis and regular periodontal maintenance during orthodontic treatment are also needed[22]. Prior to orthodontic treatment, detailed inquiries about patients' allergies, chairside oral hygiene guidance, rational selection of orthodontic instruments and careful removal of bonding materials can effectively avoid gingival hypertrophy. Orthodontic patients with gingival hypertrophy should undergo standard periodontal treatment[10]. In some patients, the problem can be eliminated partially or entirely. If the gingival hypertrophy has not completely subsided, gingivoplasty is required to restore the gingival shape to normal. Some patients who have gingivoplasty with periodontitis will require periodontal flap surgery[26].

Traditional scalpels, electric knives or lasers can remove hypertrophic gingival tissue. Traditional plastic surgery is the classical option, but bleeding is obvious with this approach, and a periodontal plug is needed to protect the wound[34]. Patients' pain and discomfort are obvious after the operation. Use of an electrotome is simple and can stop bleeding at the same time, but it may cause thermal damage to adjacent tissues,

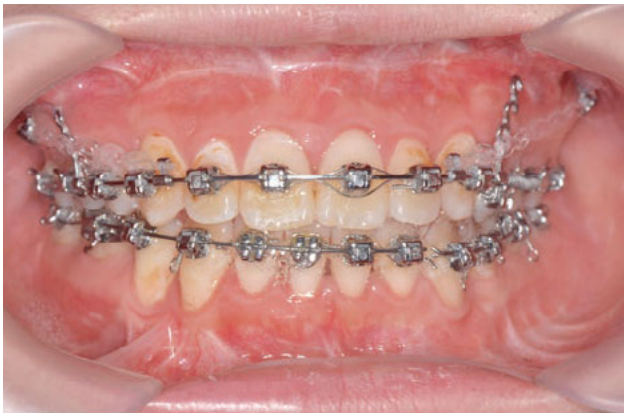

Fig 3 Gingival recession in the site of the mandibular canines and right first premolar during orthodontic treatment.

leading to delayed wound healing. Use of lasers has many advantages, such as analgesia, rapid haemostasis, antibacterial and anti-inflammatory effects, and promotion of tissue healing, which greatly improves patient comfort during and after the operation. A previous study used Nd:YAG and CO_2 lasers to remove hypertrophic gingiva in orthodontic patients, and the length of the clinical crown and depth of the gingival sulcus improved significantly afterwards[34]. There was no pain, and the operative site healed quickly[35]. Thus, the use of oral soft-tissue laser gingivectomy and gingivoplasty is a possible choice.

Orthodontic treatment and gingival recession

Gingival recession refers to root exposure caused by the movement of the gingival margin to the root of the enamel–cementum boundary. Gingival recession may lead to poor aesthetic effects, increased root sensitivity and susceptibility to caries lesions[10,36]. In addition to anatomical factors, gingival recession is related to age, periodontal disease, improper brushing, occlusal trauma and invasion of biological width[37]. Whether orthodontic treatment causes gingival recession remains controversial (Fig 3).

Studies on the factors of gingival recession

An epidemiological investigation showed that the incidence of gingival recession during orthodontic treatment was around 1.3% to 12.0%[38]. The mandibular central incisors are most prone to gingival recession, which is linked to the thinner buccal bone plate[39] and insufficient keratinised gingiva[37]. It is now generally accepted that gingival recession during orthodontic treatment is mainly due to the movement of teeth, which is beyond the physiological range of alveolar processes, such as excessive arch expansion and immoderate inclination of teeth[40]. Orthodontic force causes the root to squeeze the thin bone plate, resulting in rapid absorption and secondary gingival recession[41]; however, other studies have not found such associations between tooth movement and gingival recession[16,18,42]. Ruf et al[43] investigated 392 mandibular incisors of adolescents treated with a Herbst appliance. The appliance resulted in a mean incisor movement of 8.9 degrees (0.5 to 19.5 degrees)[43]. Only 12 patients had gingival recession or aggravation, and there was no correlation between the anterior edge of the mandibular incisor and gingival degeneration[43]. A systematic review of labial movement and gingival recession of mandibular incisors also found no association[44]. These studies suggested that insufficient free gingival thickness, a narrow mandibular-chin junction, poor plaque control and excessive brushing are potential risk factors for gingival recession[41-43]; however, Renkema et al[45] compared the incidence of gingival recession between 100 orthodontic patients after orthodontic treatment and 120 non-orthodontic patients in a retrospective study and found that the incidence of gingival recession in the orthodontic group was always higher than that in the non-orthodontic group, and there was more gingival recession in the orthodontic group. The odds ratio (OR) of gingival recession in the orthodontic group was 4.48, indicating that orthodontic treatment was a risk factor for gingival recession[45]. A systematic review found that orthodontic movement is one of the factors affecting gingival recession, and tooth movement beyond the physiological range of the alveolar process may lead to more gingival recession[46]. However, due to an absence of an optimal evaluation mechanism and a limited research sample size, strong evidence is lacking for both sides and further investigation is required.

Prevention and treatment of gingival recession

Before orthodontic treatment, the width and thickness of the compressed lateral keratinised gingiva should be evaluated and the range of alveolar process and bone plate thickness should be analysed using CBCT, whether there is bone dehiscence or fenestration, and the potential risks should be discussed; this is necessary to prevent orthodontic-related gingival recession[37,47,48]. If the periodontal soft tissue or bone is insufficient, early intervention, such as soft tissue transplantation or incremental bone surgery, is needed to ensure that the teeth being moved always move within the physiological range of the alveolar process[49]. To prevent gingival

recession during orthodontic treatment, dental practitioners or orthodontists should avoid exerting force on single teeth and moving teeth back and forth, and recommend use of segmented arch technology. In addition, it is always essential for orthodontic patients to maintain good oral hygiene.

For mild and asymptomatic gingival recession, there is only a need to maintain good local plaque control without surgical periodontal therapy. If gingival recession continues to progress, aetiological treatment is required, such as removing local risk factors, correcting improper brushing or flossing, and adjusting the orthodontic or occlusal force[50]. Once the gingival recession exceeds 2 mm, it is necessary to stop the movement of the teeth to the loading side immediately and to consider shortening the orthodontic treatment time[51]. If necessary, periodontal surgery should be performed after routine scaling and root planing[51].

Periodontal surgery includes mucogingival surgery and guided bone regeneration[52]. Mucogingival surgery involves free gingival transplantation, pedicled gingival flap transplantation and subepithelial connective tissue transplantation. It is used for a single tooth or a few teeth with gingival recession, with a mean root coverage rate of around 65% to 98%[53]. For patients with a thin alveolar bone plate or bone dehiscence, the mean root coverage rate of guided bone regeneration is around 48% to 92%[54]. Systematic reviews suggest that autogenous connective tissue transplantation or enamel matrix derivatives combined with use of a coronally advanced flap increase the possibility of root coverage in Miller type I and II single gingival recession[55]. A meta-analysis also showed that laser-assisted gingival surgery has clinical advantages in terms of improving keratotic tissue width, depth of the probe and attachment level[56]. The complete root coverage rate was 70% to 90%, which was significantly different to that of the non-laser group[56].

At present, type I and II gingival recession can be covered completely as a result of surgical treatment, while type III recession can be partially covered. Patients with type IV gingival recession are advised to undergo prosthetic treatment for coverage.

Orthodontic treatment and gingival invagination

Gingival invagination is a type of gingival cleft that occurs during orthodontic space closure with at least 1 mm depth or a vertical and horizontal probe depth of at least 2 mm in patients undergoing extraction[57]. The incidence of gingival invagination ranges from 30% to 100%[58]. It can affect plaque control, increase the risk of periodontal tissue injury, prolong closure time and disturb the orthodontic effect and stability[57,58]. Thus, sufficient attention should be paid to gingival invagination (Fig 4).

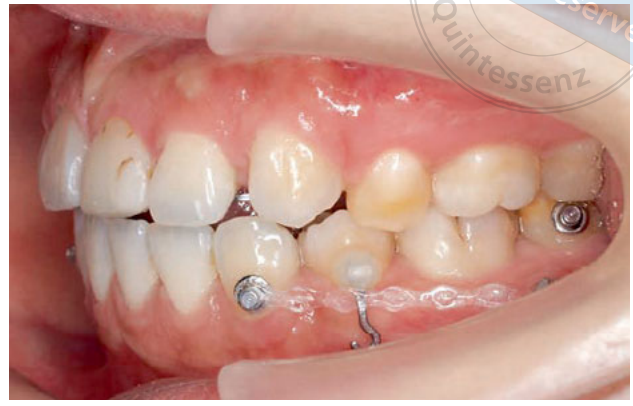

Fig 4 Gingival invagination in the extraction site of the maxillary left first premolar during orthodontic treatment.

Factors relating to gingival invagination

The relapse factor after closure of the orthodontic space may be the cause of gingival recession[59]. When the extraction space is closed, the gingival epithelium on the tension side becomes loose and 'red triangular plaque' forms[60]. At the same time, because the teeth do not move along with the gingiva, they squeeze the gingival tissue and alveolar bone on the pressure side, making the gingiva roll in, thus causing gingival invagination. Changes in bone tissue may also be a cause of gingival invagination[60]. The results showed that the immediate closure group had higher bone mineral density, less alveolar bone resorption and lower incidence of gingival invagination than the 12-week closure group. In addition to the interval time of closing the gap, the speed also has an impact on alveolar bone absorption, thus affecting the occurrence of gingival invagination[59,60]. Some studies have found that the severity of gingival invagination increased significantly in patients whose closure speed was less than 6.0 mm per month compared with those for whom it was > 1.2 mm per month[59,60]. The difference in gingival invagination between the maxilla and mandible also indirectly explains the effect of the closure space. A retrospective study showed that the incidence and severity of maxillary gingival invagination was lower than that of the mandible (maxilla 30%, mandible 70%)[58]. The closure speed was faster and bone resorption was reduced because the maxilla was less dense than the mandible[58]. These studies indicate that gingival

tissue lacks the ability to rapidly remodel, and changes occur to its morphology along with bone resorption[58-60]. There is no doubt that the more serious the bone resorption, the more obvious the gingival invagination[61].

Prevention and treatment of gingival invagination

Animal experiments and retrospective studies have found that a long interval between orthodontic closure and extraction is a potential risk factor for gingival invagination[58,61]. One of the most effective ways of preventing gingival invagination is to move teeth 2 to 4 weeks after extraction[58]. Reichert et al[62] found that early closure of the gap can only shorten the treatment time and fails to prevent or reduce the incidence and severity of gingival invagination; however, there are fewer samples in relevant studies[58,59,61], so the effect of time factors on gingival invagination requires a further design of randomized controlled trials with a larger sample size for in-depth analysis.

Multiple studies have reported that the application of different bone substitute materials at the tooth extraction site for site preservation can prevent gingival recession effectively[60,62,63]. The results indicated that the incidence of gingival invagination was 83.3% in the group in which the extraction socket underwent natural healing after complete closure of the extraction space, whereas no gingival invagination occurred in the site preservation group[61,62]. In addition, there was no gingival invagination in the guided tissue regeneration (GTR) for the group that received Gore-Tex film (Gore, Newark, DE, USA), whereas gingival invagination occurred at extraction sites in the control group[64]. Moreover, compared with the Gore-Tex group, the control group showed significant horizontal absorption of the alveolar crest, indicating that GTR combined with a barrier membrane could reduce the occurrence of gingival invagination[61]. These results suggest that guaranteeing adequate local bone mass for space maintenance may play a role in preventing gingival invagination[61,63].

Patients with gingival invagination should be treated actively to avoid it impacting their periodontal health and the outcome of orthodontic treatment at a later stage. Simple gingival resection and gingivoplasty, GTR and bone grafting can be selected based on the severity of gingival invagination. In patients with poor oral hygiene maintenance, gingival invagination is limited to the soft tissue, and patients with poor gingival morphology can undergo gingivectomy and gingivoplasty[65]. Guided periodontal tissue regeneration and bone grafting should be performed if gingival invagination penetrates the interdental papilla or accompanies alveolar bone defects[60].

Conclusion

Orthodontic treatment is closely related to gingival tissue changes and the two interact with each other. While paying attention to patients' orthodontic situation, orthodontists should cooperate closely with periodontal doctors to monitor any periodontal changes. Prior to orthodontic treatment, periodontal risk factors should be assessed and prognostic judgements should be made to predict possible risks and provide effective prevention in addition to effective chairside oral health instruction. Periodontal examination and maintenance should be performed regularly during orthodontic treatment, and periodontal problems should be addressed actively. There is still a need to increase patients' awareness of oral hygiene maintenance after treatment to ensure they maintain their periodontal health. Orthodontists should strengthen interdisciplinary cooperation with periodontal practitioners to achieve the best therapeutic effect.

Conflicts of interest

The authors declare no conflicts of interest related to this study.

Author contribution

Dr Chen Xi LI drafted the manuscript, conceptualised and designed the study, and reviewed the manuscript; Dr Yuan LIU summarised the literature and drafted the manuscript; Prof Cong Bo MI conceptualised and designed the study, and critically revised the manuscript; Drs Juan NIE and Yi ming LI took charge of bibliography retrieval and collected and integrated the literature material. All authors approved the final manuscript.

(Received May 10, 2022; accepted Nov 07, 2022)

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