**REVIEW ARTICLE**

# Deleterious Effects of Orthodontic Force to the Periodontal Tissue

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**Abstract**

The possibility of saving and rehabilitating a deteriorating dentition depends not only on position of teeth within the alveolar process but also on number of remaining teeth and their periodontal status. An unstable condition arises when the balance between forces acting on the teeth and the constraints of periodontal tissues gets disturbed either by extraction of teeth or reduction of the periodontium. Orthodontic forces are unlikely to convert gingivitis into a destructive periodontitis, but poorly executed orthodontic therapy in patients with periodontitis can easily lead to further periodontal breakdown. This article provides an overview of the potential adverse effects of orthodontic tooth movement on gingival and periodontal tissues during fixed appliance therapy.

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**Key words**: Orthodontic forces; Fixed appliances; Microbial plaque; Inflammation; Periodontal diseases.

## Introduction

The aim of orthodontic therapy is not only to improve facial esthetics and function, but also to address to the health of supporting tissues. A magnificent orthodontic correction can be destroyed, no matter how talented the orthodontist is, by failure to identify the susceptibility to periodontal disease. The pathogenesis of periodontal disease is multifactorial and the orthodontist must recognize the clinical forms of inflammatory periodontal disease. Orthodontic treatment involves application of forces on the dental structures and supporting tissues of the tooth which poses a risk to teeth and periodontal tissues and if, conditions of oral ecosystem are not favourable, then an excessive growth of microorganisms occurs that initiates caries and periodontal disease. Fixed appliance therapy produces adverse effects on the periodontium ranging from gingivitis to bone loss.

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### 1. Gingival Inflammation

Plaque retention is one of the primary etiologic factors in the development of gingivitis.[1] Orthodontic treatment leads to changes in oral hygiene habits which result in plaque retention and gingival inflammation and it is attributed to specific bacterial types which proliferate during orthodontic therapy.[2,3,4] The retention of plaque around orthodontic appliances leads to organic acids production by bacteria present in the dental plaque, which causes enamel demineralization.[5,6] Fixed orthodontic treatment create new retention areas, which are suitable for bacteria to colonize and lead to an increase in the absolute number and percentage of *Streptococcus mutans* and lactobacilli.[7,8,9]

The bacterial plaque of patients undergoing orthodontic treatment is mainly composed of spirochetes and motile rods and an increased levels of bacteroids and streptococcus species seen after orthodontic banding.[9,10] Fixed appliances encroach on the gingival sulcus, inhibits effective maintenance of oral hygiene.[11]

The gingiva is the only dental tissue which is visually accessible for direct clinical evaluation of the classic signs of inflammation and redness and swelling are commonly observed.

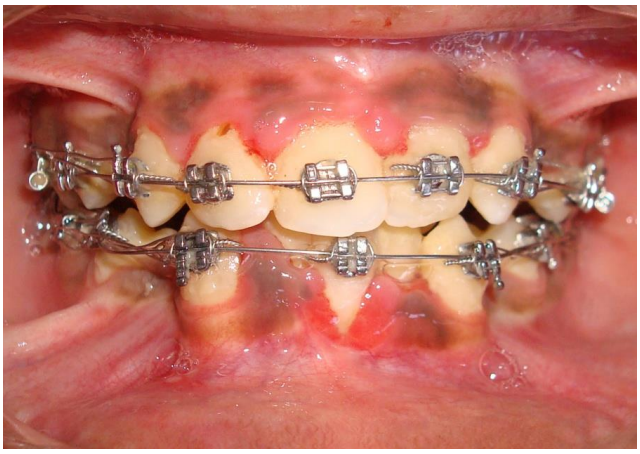

**Fig 1.** Intraoral view in a patient in both dental arches with gingivitis

Presence of bands, brackets, wires and other orthodontic attachments poses a high susceptibility to plaque accumulation on the teeth. Plaque deposition around gingival margin is the most important etiological factor of periodontal disease.[12,13,14] In healthy, reduced periodontal tissue support regions, orthodontic forces kept within the biological limits do not cause gingival inflammation.[15] On fixed appliance placement, gingival inflammation is usually transient and does not lead to attachment loss.[16] Both fixed and removable appliance impede the maintenance of oral hygiene which results in plaque accumulation.[17,18]

Fixed orthodontic treatment encourages the growth of periodontopathic bacteria species such as *Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus, Actinobacillus actinomycetemcomitans, Fusobacterium nucleatum and Treponema denticola*.[19]

Patients usually experience mild to moderate gingivitis within 1 to 2 months of appliance placement, even after maintaining seemingly excellent oral hygiene. It is usually quiescent, with no permanent damage to tissues.[20,21]

## 2. Gingival Recession

Another long-term complication of orthodontic treatment is gingival recession. The incidence ranges from 1.3% to 10% and at least 2 mm width of keratinized gingiva is necessary for gingival health.[22]

Gingival recession is defined as “the exposure of the root surface by an apical shift in the position of gingiva.” [23]

Mandibular incisors mostly express gingival recession in response to orthodontic mechanotherapy and shows apical displacement of the gingival margin with labial bodily movement. It is due to thin or nonexistent labial plate of bone and inadequate or absent keratinized gingiva that covers labially prominent teeth. This condition is risky and harmful because it leads to poor esthetics, root sensitivity, loss of periodontal support, difficulty in maintaining oral hygiene, difficulty in successful periodontal repair, and increased caries susceptibility.[24]

Appropriately applied orthodontic forces do not damage the periodontium. However, insufficient width of attached gingiva is believed to be a predisposing factor for recession.[25,26] Another factor is chronic marginal gingivitis, or chronic necrotizing ulcerative gingivitis, which

rapidly destroy the marginal alveolar bone and gingival attachment.[22] Alveolar bone dehiscence is a predisposing factor for the development of gingival recession.

Orthodontic tooth movement (OTM) will not result in recession, as long as a tooth is housed within the alveolar bone.[27,28] Tension in the marginal tissue created by the orthodontic forces is an important factor in causing gingival recession.[27] as thin, delicate tissues are more susceptible to gingival recession during orthodontic treatment than in normal or thick tissue.[27,29] If the patient exhibits a minimal zone of attached gingiva or thin tissue, a free gingival graft can be performed to enhance the type of tissue around the tooth. This controls the inflammation and it should be done before initiation of orthodontic movement.[27]

### 3. Attachment Loss

In many orthodontic patients, mechanical irritation caused by the band or cement, in addition to trapped plaque is the the principal reason for the associated gingival and periodontal inflammation.[20,30]

Use of optimum forces during orthodontic treatment in patients with excellent oral hygiene and in the absence of pre-existing periodontal disorders, does not pose any significant periodontal risk to the patient.[31] However, in the presence of poor oral hygiene and pre-existing untreated periodontal disorders, fixed orthodontic appliances and tooth movement can contribute to significant and permanent periodontal damage and in absence of plaque and with orthodontic forces at physiological levels, bone loss do not occur.[32]

The risk of attachment loss can be anticipated when such iatrogenic irritations are present.[33]

When loss of attachment in orthodontic patients compared with controls was examined, the data showed that 10% had significant attachment loss, but 50% had no attachment loss.[34]

Patient age also influences the risk of alveolar bone loss. Adults may experience greater bone loss than adolescents, since the periodontal disease is higher in this group.[35,36]

Patients with pre-existing periodontal problems and bone loss, must be referred to and treated by the periodontist before initiating the orthodontic treatment.[37]

### 4. Direct Gingival Traumatic Impingement

In patients with a Class II, Division 2 malocclusion, functional trauma arises from impingement of incisors on the mandibular soft tissue can lead in marginal recession of facial gingiva of mandibular incisors.[38]

Similarly, in extreme cases of deep bite, direct trauma to the gingiva from the incisal edges of mandibular incisors contribute to gingival recession palatal to maxillary incisors and it result in the complete ablation of the gingival unit which provides a portal of entry for infection spread to the subjacent periodontal attachment apparatus.[39]

When such process occurs, the periodontal status of the patient is compromised, because the depth of the pocket beyond 3 mm inhibits complete removal of bacterial biofilm subgingivally.[10,40]

### 5. Gingival Enlargement

The gingival overgrowth is the most common problem associated with gingivitis in orthodontic treatment. The affected tissue is generally edematous, and bleed on gentle probing.[41]

The average incidence of gingival enlargement is 4 times greater around posterior teeth compared with incisors and canines.

They listed the following causes:

1. Mechanical irritation caused by bands, more on posterior than on anterior teeth,
2. Chemical irritation produced by cements used for banding,
3. Food impaction, because of the proximity of the arch wires to the soft tissues, and
4. Less efficient oral hygiene maintenance.

Interdental region has greater incidence compared with the facial aspect of the gingival margin and as long as a band is in place, it is prone to produce gingival irritation, leading to enlargement and can be prevented only by

properly fitting each band and making it self-cleansing.[42]

### 6. Black Triangles

Gingival embrasures are defined as the embrasure existing cervical to the interproximal contact.[43]

When the embrasure space is not completely filled by the gingival tissue, open gingival embrasures exist which contribute to food debris accumulation. This adversely affects the health of the periodontium and are more common in adult patients with bone loss.[44]

Black triangle or open gingival embrasure can occur as potential complication in about than 1/3 of all adult orthodontic patients.[43,45] Patients with triangular crown morphology are more susceptible to open gingival embrasures as the crowns of the central incisors are much wider incisally than cervically, resulting in a high contact point.

Interproximal reduction (IPR) of enamel between the triangular crowns will broaden the contact area which will reduce open gingival embrasures. Typically, 0.5-0.75 mm of enamel is removed with IPR for correction of black triangles.[46]

### 7. Gingival Hyperplasia

A large number of drugs have been implicated as cause for gingival hypertrophy / hyperplasia. Gingival enlargement in these patients is not due to increase in the number of periodontal cells but due to an increase in the extracellular volume.[47] This increase in the extracellular volume is caused by hyperplasia involving fibroblasts.

Three types of drug categories have been implicated as causative factors of gingival enlargement.[48] They include:

1. Anti epileptic drugs – Phenytoin, Phenobarbitone, Valproic acid, Primidone, Vigabatrin and carbamazepine.
2. Calcium channel blockers – Nifidepine, Verapamil, Diltiazem and Amlodepin
3. Immunosuppresive drugs – Cyclosporine.

Drug induced gingival overgrowth usually occurs within first three months of starting drug therapy with the offending drug. This usually begins as an enlargement involving interdental papillae.

#### Etiology

- Poor oral hygiene: Presence of dental plaque can provide a reservoir for accumulation of drugs like phenytoin/ cyclosporin.[49]
- In patients who have undergone orthodontic procedures the presence of nickel could predispose to formation of gingival over growth.
- Susceptibility of some subpopulation of fibroblasts and keratinocytes to phenytoin, cyclosporin and other drugs which could cause gingival overgrowth.
- Number of langerhans cells present in the oral epithelium is another risk factor. More the number worse the risk. These drugs have a tendency to accumulate inside these cells causing prolonged effect on the gums.[5]

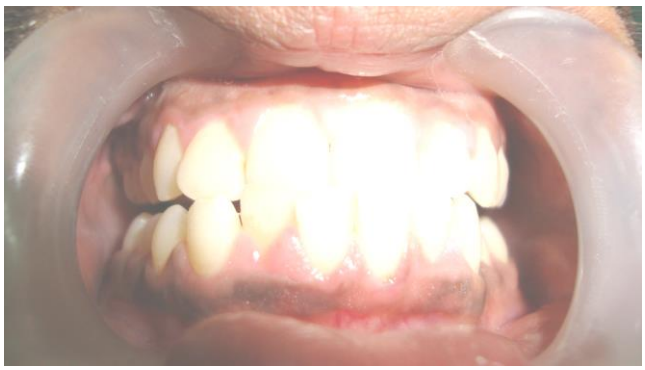

**Fig 2.** Clinical picture of gingival enlargement in a male epileptic patient (Grade 1) after 2 months of initiation of phenytoin therapy.

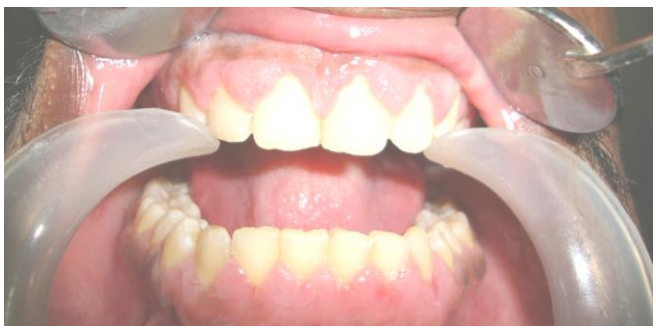

**Fig 3.** Clinical picture of gingival enlargement in a male epileptic patient (Grade 2) after 6 months of initiation of phenytoin therapy.

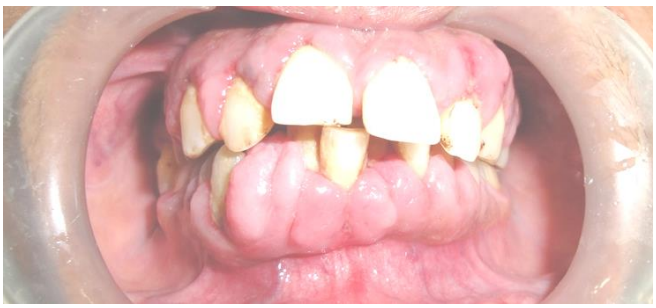

**Fig 4.** Clinical picture of gingival enlargement in a female epileptic patient with more prominent involvement of interdental papillae (Grade 3) after 6 months of initiation of phenytoin therapy.

### Management

1. Stopping / substituting the offending medicine.
2. Gingival overgrowth reverts back to normal within 3 months.
3. Maintenance of strict oral hygiene.
4. Regular mouth wash using cholorhexidine.
5. Oral metronidazole for 21 days.
6. Surgical removal of the gingival overgrowth (gingivectomy).

It has been shown that an average 7 mm reduction in occlusal vertical dimension occurs in as many years[10].

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