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Case Series

Soft tissue impactions associated with orthodontic appliances: clinical insights into periodontal intervention cases involving quad helix, coil spring, and temporary anchorage devices

Divya Manikantan[1]*, Ardra CK[2], Bindu R Nayar[1], Koshi Philip[3]

[1]Dept. of Periodontics, Kerala University of Health Science, Kerala, India
[2]Dept. of Periodontics, Government Dental College Thiruvananthapuram, Kerala, India
[3]Dept. of Orthodontics, Kerala University of Health Science, Kerala, India

Abstract

Orthodontic appliances such as quad helix, NiTi coil springs, and temporary anchorage devices (TADs) are indispensable tools in modern orthodontics. However, their prolonged intraoral presence, improper positioning, and lack of patient compliance can occasionally result in soft tissue complications, including mucosal overgrowth and impaction. This case series presents three distinct presentations of soft tissue impaction around orthodontic appliances; each managed with periodontal intervention. It highlights the etiopathogenesis, clinical manifestations, and management strategies while underscoring the critical role of the periodontist in early detection and minimally invasive resolution of such complications.

Keywords: Soft tissue impactions, Quad helix, NiTi coil, Temporary anchorage device.

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1. Introduction

Orthodontic appliances are indispensable in contemporary practice, but they are not without complications. Among the less frequently reported yet clinically important problems is soft tissue impaction, where components of appliances such as quad helix helices, NiTi coil springs, or temporary anchorage devices (TADs/OMIs) become embedded within the oral mucosa. This complication not only compromises patient comfort but may also lead to inflammation, infection, and failure of anchorage systems if not recognized early.

The underlying pathogenesis is multifactorial. Mechanical trauma from appliance components, particularly in areas of thin or mobile mucosa, initiates a cascade of chronic inflammation, granulation tissue formation, and eventual mucosal overgrowth. Factors such as altered anatomy from previous surgeries, inadequate oral hygiene, poor appliance design, and improper positioning further predispose to impaction. In particular, placement of TADs in non-keratinized or movable alveolar mucosa significantly increases the risk of soft tissue irritation and peri-implant complications. Despite its relevance, soft tissue impaction associated with orthodontic appliances remains underreported in the literature. Case documentation and systematic analysis are therefore essential to enhance awareness, improve preventive strategies, and guide clinical management. This report presents cases illustrating soft tissue impaction linked to different orthodontic appliances, highlighting the multifactorial etiology, clinical presentation, and the indispensable role of the periodontist in diagnosis, treatment, and prevention.

2. Case Series

2.1. Case 1

Palatal Mucosal Impaction of a Quad Helix Appliance in a Cleft Palate Patient a 14-year-old male with a repaired cleft palate reported palatal discomfort and difficulty in

*Corresponding author: Divya Manikantan
Email: mahekuttan@gmail.com

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maintaining hygiene. He had undergone quad helix therapy for maxillary expansion. Examination revealed partial mucosal overgrowth encasing the helices of the appliance, accompanied by mild inflammation. Diode laser assisted removal of the appliance was done.

Amoxycillin 500 mg three times daily for three days and paracetamol 500mg twice daily for the first day was given, the patient was instructed on hygiene maintenance. Healing was uneventful over a 10-day period **(Figure 1)**.

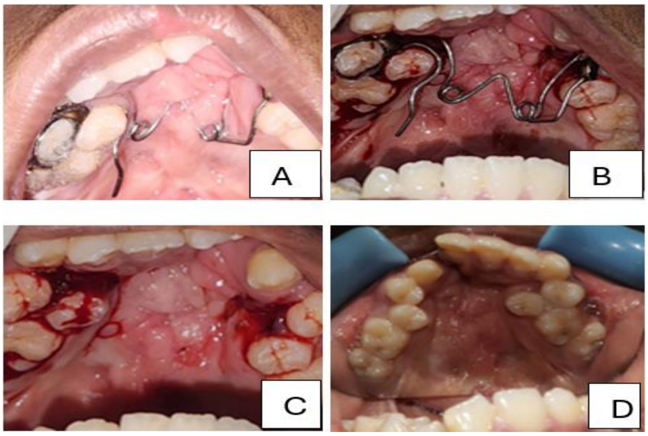

Figure 1: A: Pre-operative view; **B:** Incision placed; **C:** Post-operative view; **D:** After 10 days

2.2. Case 2

Buccal Mucosal Embedding of a NiTi Open Coil Spring a 16-year-old male undergoing fixed orthodontics presented with pain and swelling in the buccal mucosa near the upper right premolars. Examination showed partial embedding of a NiTi coil spring, along with erythematous change The spring was removed, using diode laser and the site debrided and irrigated. Post operative antibiotics amoxycillin 500 mg three times daily for five days and paracetamol 500 mg twice daily for next two days were given. The lesion healed within 10 days without recurrence (**Figure 2**).

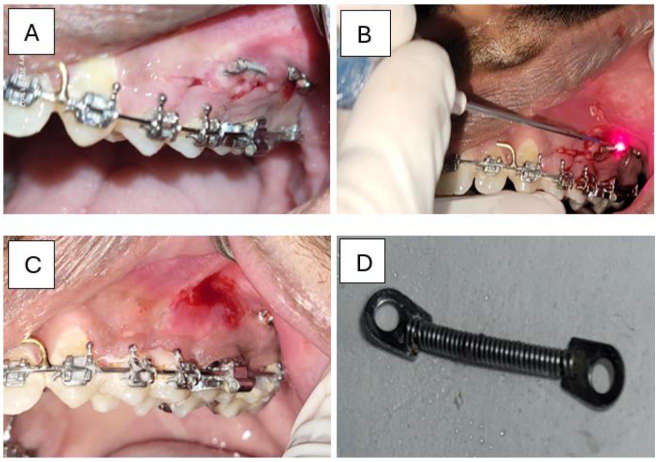

Figure 2: A: Pre-operative view; **B:** Laser excision; **C:** Post-operative view; **D:** Removed appliance

2.3. Case 3

Soft Tissue Overgrowth around a TAD in the mandible a 14-year-old female reported localized discomfort near a buccally placed TAD used as Mini Anchorage and Movement Platform (MAMP), for maxillary protraction Clinical findings included soft tissue proliferation over the TAD head and mild peri-implant inflammation. The TAD was exposed, using diode laser and local irrigation and corticosteroid gel were administered along with antibiotics. As depicts in **(Figure 3)** satisfactory healing was noted after ten days,

Figure 3: A: Pre-operative view; **B:** Post-operative view; **C:** After 10 days

3. Discussion

Soft tissue impaction associated with orthodontic appliances is an underreported complication with multifactorial ethology. The contributing pathogenesis includes: The pathogenesis of soft tissue impaction typically begins with chronic low-grade mechanical irritation of the mucosa by an orthodontic appliance—be it the helices of a quad helix, a NiTi coil spring, or the head of a TAD. This persistent trauma stimulates a localized inflammatory response, marked by vasodilation, recruitment of neutrophils and macrophages, and increased release of inflammatory cytokines such as IL-1β and TNF-α. Over time, the repeated injury prompts epithelial hyperplasia, proliferation of fibroblasts, and angiogenesis, leading to granulation tissue formation and fibrous mucosal overgrowth. If the mechanical stimulus is not eliminated, this can culminate in partial or complete embedding of the appliance into soft tissue.[1]

In Case 1, a unique contributing factor was the patient's prior cleft palate repair. The altered palatal architecture due to surgical scarring likely impaired tissue resilience and vascularity. Scarred or tethered mucosa tends to respond atypically to mechanical stimuli—often with exaggerated fibrous overgrowth or granulation. The helices of the quad helix, in close contact with the palatal mucosa, acted as a chronic irritant, especially in the context of suboptimal oral hygiene and irregular follow-up as the patient failed to report back for one and half year after appliance placement.

Case 2 involved a NiTi coil spring that had migrated into the buccal mucosa. The open-coil design, while effective for space closure, carries the risk of soft tissue impaction if not secured adequately. The pathogenesis here involved displacement-induced trauma, which disrupted the epithelial barrier, allowing bacterial ingress and leading to localized ulceration and purulence. The inflammatory exudate and tissue breakdown were likely potentiated by poor hygiene and biofilm accumulation, further perpetuating tissue injury.

In Case 3, the placement of the TAD too close to the movable buccal mucosa created a site of constant friction. The micro-motion of the screw during mastication and tongue movement, coupled with inadequate clearance from soft tissue, initiated chronic irritation. This triggered peri-implant mucositis, characterized by mucosal edema, epithelial thickening, and eventual soft tissue impingement over the TAD head. Without timely removal, such lesions could progress to soft tissue hyperplasia or even peri-implantitis.

The periodontist plays an indispensable role in the diagnosis, management, and prevention of such iatrogenic complications. Interventions such as laser excision, soft tissue conditioning, repositioning of appliances, and reinforcement of oral hygiene protocols form the cornerstone of treatment.

The acceptance of quad helix device by cleft palate patients who had been traumatized by multiple surgeries suggests that it would be effective in early treatment of non-cleft children requiring transverse expansion of the maxilla. Temporary Mucosal Protector thus helps to protect the impingement of Palatal expander on palatal soft tissues and provides relief to the patient.[2]

Orthodontic mini-implants (OMIs) placed in movable alveolar mucosa carry a high risk of tissue irritation. The insertion zone of opportunity is described as extending 2 mm coronol from the mucogingival junction (MGJ), an area where the mucosa is virtually immobile. However, root proximity in this zone may necessitate placing mini-screws more apical to the MGJ—where the mucosa is thinner and more mobile—predisposing to soft tissue overgrowth, inflammation, micro-tears, and ulcerations, resulting in patient discomfort or pain [3]

A thin gingival phenotype and inadequate width of keratinized gingiva (< 2 mm) are significant risk indicators for peri-implant mucositis, peri-implantitis, and brushing discomfort. Palatal gingival grafts, first described by Björn in 1963, remain a reliable method for augmenting keratinized tissue, Free gingival grafts (FGG) and connective tissue grafts (CTG) have demonstrated high success rates in increasing the width of keratinized gingiva and thickening the gingival phenotype.[4] From a biological perspective, biotype represents a genetically determined trait, whereas phenotype reflects the combined influence of genetic factors and environmental conditions. Gingival phenotype is site-specific and can change over time. Phenotype modification therapy aims to create a more favourable environment for disease prevention and periodontal stability. Phenotype modification in orthodontic patients offers multiple advantages, including improved periodontal health, enhanced stability of orthodontic outcomes, a reduction in periodontal complications, and shorter treatment times. It also broadens treatment possibilities such as arch expansion and minimizes the need for extractions or camouflage approaches. From a biomechanical perspective, it facilitates force application closer to the center of resistance, while simultaneously improving patient comfort and decreasing soft tissue-related problems.[5]

In specific situations, combining orthodontic mini-implant (TAD/OMI) placement with free gingival grafting can transform thin, non-keratinized peri-implant mucosa into a thick, keratinized zone. This modification enlarges the available insertion area, increases patient comfort, prevents mucosal complications, and reduces the likelihood of root contact. Such an approach is particularly useful when TADs are placed in non-keratinized vestibular interradicular mucosa, extra-radicular supra-apical or subapical sites, the mandibular retromolar region, or the posterior buccal shelf.[6]

Several studies support the role of keratinized mucosa in the success of TADs. Heng et al., in a prospective study of 140 mini-implants, reported that lack of keratinized mucosa significantly increased infection and failure risk.[7] Lai and Chen, in a retrospective analysis of 266 TADs, observed a survival rate of 96.2% for implants placed in keratinized mucosa compared with only 66.7% in oral lining mucosa.[8] Similarly, Manni et al., in a study of 300 mini-screws, found higher success when inserted in attached gingiva or along the mucogingival line.[9] Chen et al., in a retrospective study of 492 TADs, identified peri-implant soft tissue inflammation and premature loading within three weeks as the most significant predictors of failure. Earlier, Lang and Löe (1972) highlighted the importance of keratinized gingiva for gingival health, and later, Lang et al. (1995), in an experimental monkey model, demonstrated that absence of keratinized mucosa around dental implants increased susceptibility to plaque-induced tissue breakdown. A minimum of 2 mm of keratinized tissue was shown to exert a protective effect, improving peri-implant health, plaque control, and soft tissue indices while reducing inflammation and bone loss. Overall, phenotype modification around orthodontic appliances helps establish a more favorable peri-implant environment, preventing soft tissue complications and supporting long-term stability. This approach is especially valuable in non-compliant orthodontic patients where plaque control is often inadequate.[10]

4. Conclusion

Soft tissue impaction around orthodontic appliances is a preventable complication. A comprehensive approach—encompassing careful appliance design and positioning, phenotype modification, mucosal protection, and strict hygiene reinforcement—can minimize risks. For non-compliant patients, phenotype modification provides a particularly valuable biological safeguard, ensuring long-term orthodontic stability.

5. Source of Finding

None.

6. Conflict of Interest

None.

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