A 32-year-old female patient complaining of discomfort and pain from her mandibular anterior region was referred for a specialist periodontal opinion. She was experiencing sensitivity of the teeth, tenderness and intermittent gingival bleeding. She felt that there had been progressive deterioration over the last two years (Fig. 1).

The patient regularly saw her general dental practitioner, who was undertaking supportive care that included scaling, polishing and desensitisation with the use of fluoride varnishes. Having been given oral hygiene advice, she was using a soft-bristled manual toothbrush on a twice daily basis in order to maintain her plaque levels. Medically, she was a fit and healthy non-smoker, working as a primary school teacher. As far as she was aware, she did not have any parafunctional habits such as clenching or grinding her teeth. There was no history of previous orthodontic treatment.

Clinical examination using the ZEISS EyeMag Pro F loupes (Carl Zeiss) established that all permanent teeth were present, excluding her third molars, and she had a caries-free dentition. She showed a good level of oral hygiene, although she did not have any parafunotional habits such as clenching or grinding her teeth. There was no history of previous orthodontic treatment.

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Clinical examination using the ZEISS EyeMag Pro F loupes (Carl Zeiss) established that all permanent teeth were present, excluding her third molars, and she had a caries-free dentition. She showed a good level of oral hygiene, although there were some small plaque and calculus deposits present throughout the dentition. Assessment of the area of main concern found there was a reduced vestibular sulcus with a relatively broad mandibular labial frenum insertion. The superior insertion of the frenum was at the mucogingival junction of teeth #41 and #31. There were 3 mm of labial recession associated with tooth #31 and #41 and #31 was erythematous and swollen, this was tender and bled easily upon probing. The interdental papillae were intact, albeit the midline papilla particularly inflamed. There was very little in the way of attached keratinised tissue apical to the recessive defects.

Teeth #41 and #31 were labially displaced owing to mild overcrowding and there was mild attritive tooth surface loss affecting both the maxillary and mandibular central incisors. Protrusive and lateral guidance involved these teeth, but there was no significant mobility (Fig. 2). Radiographically, there was no apical pathology and there was minimal interdental crestal bone loss. The interdental bone between teeth #41 and #31 was, however, limited owing to the mild overcrowding (Fig. 3).

A diagnosis of Class IIb Miller’s defects affecting teeth #41 and #31 was made. In such a case, the lateral and posterior movement of the tooth was limited owing to the mild overcrowding. This was also observed radiographically where there was minimal interdental bone loss. The interdental bone between teeth #41 and #31 was, however, limited owing to the mild overcrowding (Fig. 3).

After the diagnosis, the patient was advised on additional preventive measures with appropriate toothbrushing techniques. She was subsequently reviewed after further simple scaling and polishing procedures. She then consented for mucogingival surgery to the mandibular anterior region. The proposed treatment was an internal frenectomy and pre-existing labial bone deficiency (dehiscence or fenestration) as a result of the mild overcrowding. The condition may have been exacerbated by some occlusal overload and attrition (Figs. 4 & 5).

Surgical treatment

First, the creation of a partial-thickness supra-periosteal pouch...
in the region of teeth #42 to #32 was achieved with the use of tunnelling instruments. There were partial papilla separation and internal frenotomy (Fig. 6). After this, an autogenous connective tissue graft was harvested from the left anterior lateral aspect of the palate. This was subsequently guided through the tunnel to rest over the exposed root surfaces of teeth #41 and #31. In addition to this, the graft would provide supplemental support for the overlying soft tissue in the region (Fig. 7). The gingival soft tissue lay passively over the connective tissue graft prior to suturing and wound closure (Fig. 8).

Coronal advancement of the overlying pouch/ap was achieved with a continuous suture technique. Tension-free closure of the wound was possible; however, specific caution was required particularly in the region of tooth #31 owing to the previous separated frenal insertions. Were there to be excessive coronal advancement of the pouch/ap, this could have led to potential wound breakdown due to increased tension in the region. The connective tissue graft was intentionally left exposed to allow for an increase in the zone of keratinised tissue after healing (Figs. 9 & 10).

At the two-year review, the patient reported no sensitivity or tenderness in the region and was delighted with the outcome. She was able to fully clean the teeth and excellent gingival health was observed (Fig. 11). At the review stage, there were no signs of inflammation, no bleeding on probing, and no swelling or oedema present. Although there was still minor recession (1 to 2 mm) present affecting teeth #41 and #31, it was not possible to achieve full root coverage owing to the general positioning of the teeth, the attritive wear present, and the limited support and width for the interdental papillae, especially in between teeth #41 and #31. The persistent mild recession was no cause of concern for the patient.

The thickness of the gingiva and the zone of attached keratinised tissue had been increased, in addition to the vestibular sulcus being deepened. All of these features enabled the patient to fully maintain the area. The crucial aspects for a successful outcome for the case were to ensure careful soft-tissue handling, good adaptation and stability of the connective tissue graft at the recipient site, and tension-free wound closure.

At three months post-treatment, the hard palate donor site was fully healed with no signs of scarring (Fig. 12).

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