

Randy R Fitzgerald, Swati Y Rawal, Ann W Walters, John D Walters

## Pre-orthodontic gingival augmentation with an acellular dermal matrix allograft: a case report



**Randy R Fitzgerald**

Private practice of periodontics, Phoenix, AZ, USA

**Swati Y Rawal**

Department of Periodontology, College of Dentistry, The University of Tennessee Health Sciences Center, Memphis, TN, USA

**Ann W Walters**

Private practice of orthodontics, Marion, OH, USA

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Human and animal studies suggest that the thickness of the marginal tissue is a determinant of susceptibility to gingival recession during facial tooth movement. When thin facial marginal tissues are inflamed, there is even greater concern about the risk of attachment loss during orthodontic treatment. This report describes a case where a subepithelial acellular dermal matrix allograft was used to augment the thickness of gingival tissue and the zone of attached gingiva overlying prominent mandibular incisor roots prior to orthodontic treatment of a young patient. Use of an allograft shortened the procedure time and eliminated the need for palatal donor sites. The graft enhanced the tissue thickness facial to the mandibular anterior teeth and produced relatively little post-operative discomfort. Although plaque-induced inflammation developed around the mandibular anterior teeth during orthodontic treatment, there was no loss of attachment.

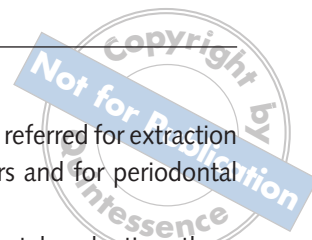
**Correspondence to:**

John D Walters  
College of Dentistry  
The Ohio State University  
305 West 12th Avenue,  
P.O. Box 182357  
Columbus,  
OH 43218-2357, USA  
Email: walters.2@osu.edu  
Tel: (614) 292-1169  
Fax: (614) 292-2438

### ■ Introduction

Although gingival recession is most common in adults, it also occurs in children. In 12% to 19% of children, there is a site at which the zone of keratinised gingiva measures < 1 mm in apicocoronal width or has minimal tissue thickness when measured in the buccolingual direction<sup>1</sup>. In most instances, sites with a minimal width of keratinised gingiva do not lose attachment during orthodontic treatment<sup>2</sup>. However, under some

circumstances, facial tooth movement at these sites may induce attachment loss and gingival recession<sup>1-3</sup>. Recession may occur during orthodontic movement of facially prominent teeth and may also occur during facial tipping or translation of an incisor that has thin facial alveolar bone and gingiva<sup>4</sup>. Permanent teeth that erupt facial to a primary tooth may also be susceptible to recession<sup>5</sup>. Several investigators have suggested that gingival augmentation prior to orthodontic treatment may be beneficial<sup>1,4-6</sup>.



Studies in monkeys have suggested that the buccolingual thickness of the marginal tissue, rather than the apicocoronal width of keratinised gingiva, is an important determinant of susceptibility to gingival recession during facial orthodontic movement<sup>7</sup>. Furthermore, sites that undergo attachment loss during facial tooth movement in monkeys typically exhibit clinical signs of inflammation<sup>7,8</sup>. In humans, thin marginal gingiva appears to be more prone to inflammatory changes and gingivitis<sup>9,10</sup>. Thin marginal tissue is also associated with an increased risk of recession when the mandibular incisors are inclined beyond 95 degrees relative to the mandibular plane<sup>11</sup>. In rats with plaque-induced gingivitis, sites with thin marginal tissue appear to be more susceptible to attachment loss compared with sites with thick marginal tissue<sup>12</sup>. Thus, it is reasonable to consider augmentation of thin marginal tissue at sites that are at risk for attachment loss during facial tooth movement. This report describes a case where an acellular dermal matrix allograft (ADMA; Alloderm®, BioHorizons USA) was used to augment the thickness of gingival tissue overlying prominent mandibular incisor roots prior to orthodontic treatment.

### ■ Case description and results

The patient, a Caucasian female with no medical risk factors, was referred at age eight for an orthodontic evaluation, and orthodontic records were obtained. Maxillary and mandibular space maintainers were placed to control a vertical growth pattern and preserve space for eruption of the premolars due to early loss of the mandibular left second primary molar. When the patient's orthodontic records were updated at age ten, she presented with a class II malocclusion, severe maxillary crowding, mild mandibular crowding, an overjet of 7 mm, an overbite of 30%, lip incompetence and an unerupted maxillary right canine. The lower incisor to mandibular plane angle measured 94 degrees. In addition, she had a slight midline discrepancy and a tooth size discrepancy related to her relatively large maxillary teeth. Cephalometric analysis revealed a hyperdivergent pattern and protrusion of the maxillary teeth. There were concerns about inadequate oral hygiene and the relatively thin gingiva over her prominent man-

dibular anterior teeth. She was referred for extraction of her maxillary first premolars and for periodontal evaluation.

At the time of the periodontal evaluation, there was no attachment loss facial to the patient's lower anterior teeth and the probing depths on the direct facial aspect of these teeth did not exceed 2 mm. However, facial concavities were visible between prominent lower anterior roots, suggesting that the bone and soft tissue overlying the roots was thin. The average width of the keratinised gingiva facial to the central incisors, lateral incisors and canines was approximately 2.3 mm, 2.9 mm and 1.8 mm, respectively (Fig 1). The gingiva in this region exhibited slight to moderate marginal inflammation, with a Gingival Index (GI<sup>13</sup>) of 2 between teeth 31, 41 and 42, a GI of 1 elsewhere, and a Plaque Index (PI<sup>13</sup>) of 1. To address concerns about incisor inclination, inflammation and the thin facial tissue and bone, gingival augmentation facial to the lower incisors and canines was recommended to reduce the risk of recession during orthodontic treatment. The palate was evaluated as a potential connective tissue autograft donor site, but the connective tissue in this area appeared to be less than 3 mm thick. Based on the patient's age, the relatively broad scope of the proposed surgery and difficulty in obtaining sufficient autograft material, a subepithelial ADMA was recommended.

The soft tissue graft was performed under local anaesthesia with 2% lidocaine containing epinephrine 1:100,000. As the facial tissue was thin, it was not feasible to prepare the graft bed with a partial thickness dissection. A full thickness flap was dissected from the distofacial of tooth 35 to the distofacial of tooth 45, taking care to preserve the interproximal papillae (Fig 2). The ADMA was hydrated in Hanks Balanced Salts Solution with three changes of solution. The allograft was trimmed and positioned with its connective tissue surface facing bone, using 5-0 chromic gut interrupted sutures (Fig 3). The mucosal flap was advanced to cover the allograft and closed with a 5-0 coated Vicryl (Ethicon) sling suture (Fig 4). Chlorhexidine gluconate rinse (0.12%) was prescribed for post-operative plaque reduction, and liquid ibuprofen (400 mg q.i.d.) for analgesia.

The patient was careful to avoid trauma to the surgical site, so the post-operative course was



**Fig 1** Facial anterior view of the patient's dentition at the orthodontic consultation, showing thin facial gingiva overlying prominent roots of mandibular anterior teeth. Markers indicate the position of the mucogingival junction.



**Fig 2** Facial view of the graft bed. The interproximal papillae were dissected in partial thickness, whereas the remaining bed was full thickness.



**Fig 3** Facial view of the hydrated acellular dermal matrix allograft positioned with interproximal resorbable sutures.



**Fig 4** Facial view of the full thickness flap coronally advanced over the allograft and positioned with sling sutures.

uneventful. At the time of suture removal (9 days), the site exhibited moderate redness and swelling (Fig 5), but the patient was reasonably comfortable. During the next 3 weeks, the patient continued with the use of chlorhexidine rinse and clinical signs of inflammation noticeably resolved (Fig 6). Healing appeared to be complete after 2 months and the concavities between the mandibular incisor roots appeared less pronounced, suggesting that the thickness of the gingiva overlying the roots had increased.

Orthodontic treatment was initiated 3 months after grafting and continued for 25 months. Plaque removal during this period was difficult for the patient and plaque was visible at most orthodontic appointments (PI = 2). This produced inflammatory

changes in the marginal and interproximal tissues (GI = 2; Fig 7). However, there was no evidence of clinical attachment loss at the conclusion of orthodontic treatment (Fig 8) and probing depths did not change significantly. The width of the keratinised gingiva facial to the central incisors, lateral incisors and canines averaged 5.0 mm, 4.2 mm and 3.4 mm, respectively. Compared with pretreatment dimensions, the keratinised gingiva increased by 2.7 mm facial to the centrals, 1.3 mm facial to the laterals and 1.6 mm facial to the canines. Oral hygiene improved after removal of the orthodontic appliances, leading to resolution of clinical signs of gingivitis (GI = 0; Fig 8). A comparison of models taken before and after grafting revealed that the thickness of the facial soft



Fig 5 Facial view of the surgical site after 9 days of healing.



Fig 6 Facial view of the site taken 4 weeks after surgical treatment, but prior to the beginning of orthodontic treatment.



Fig 7 Facial view of the site taken 16 months after surgical treatment and 13 months after initiation of orthodontic treatment. Plaque-induced inflammatory changes are evident, but there is no evidence of gingival recession.



Fig 8 Facial view of the mandibular anterior site following completion of orthodontic treatment (30 months after soft tissue grafting), showing a reduction in the root prominences and an increased zone of attached gingiva with more apical positioning of the mucogingival junction.

tissue increased by approximately 0.4 mm. Interestingly, post-treatment analysis of models and cephalometric radiographs demonstrated that the mandibular incisors were not advanced during orthodontic treatment, but the inter-canine distance increased by 0.5 mm and the mandibular canines and molars erupted 3 mm. The mandibular incisor to mandibular plane angle did not increase during treatment. Unanticipated mandibular growth made it possible to correct the overjet without proclining the incisors and lateral expansion in the premolar region created sufficient space to correct the mandibular crowding.

## Discussion

Gingival augmentation is frequently accomplished with autografts harvested from the palate or an edentulous ridge. In patients with palatal exostoses, a shallow palate or thin palatal tissue, it is difficult to harvest a sufficient amount of autograft material<sup>14</sup>. Moreover, many patients are reluctant to have a second wound at the donor site. Under these circumstances, a soft tissue allograft is a reasonable alternative approach. Alloderm<sup>®</sup> is a bioactive soft tissue allograft material that is processed to remove the





epidermis and all antigenic cells in the dermis, yielding an acellular matrix comprising proteoglycans, collagen, elastin and blood vessel channels that facilitate revascularisation, cell ingrowth and repopulation. There are no documented cases in which disease was transmitted from a donor to a recipient using this ADMA product.

The rationale for grafting in this case was to increase the thickness of soft tissue to reduce the risk of gingival recession associated with facial tooth movement in the presence of thin facial bone and thin, inflamed soft tissue. This risk appears to be higher in individuals where the incisors are proclined to yield a final mandibular incisor to mandibular plane angle that exceeds 95 degrees<sup>11</sup>. In this case, the incisor to mandibular plane angle measured 94 degrees before treatment and was expected to increase slightly during orthodontic treatment. However, differentially greater mandibular growth made it possible to correct the overjet without proclining the mandibular incisors. Consistent with previous clinical trials with sub-epithelial grafting of ADMA<sup>15,16</sup>, there was an increase in facial soft tissue thickness of approximately 0.4 mm in this case. The increase in gingival thickness obtained with an ADMA is reportedly similar to that obtained with palatal connective tissue autografts<sup>17</sup>. There are previous reports of cases where ADMA was used for gingival augmentation in children prior to orthodontic treatment<sup>18,19</sup>. In these cases the ADMA was grafted onto a split-thickness bed, using an approach similar to that used in a free gingival autograft. As this approach is associated with healing by secondary intention, post-operative inflammation is usually more pronounced and more persistent than that observed with a subepithelial ADMA.

Although root coverage was not required in the present case, the stability of root coverage obtained with ADMA at recession defects appears to be reasonably good. In short-term (6 to 12 months) clinical studies that compared use of subepithelial ADMA with subepithelial connective tissue autografts in the treatment of Miller class I or II recession defects, ADMA and autografts both produced significant root coverage. There were no significant differences between ADMA and autografts with respect to root

coverage and gains in clinical attachment level, but sites treated with autografts developed a significantly larger zone of keratinised gingiva<sup>20,21</sup>. Longer-term (24 to 48 months) studies suggest that subepithelial placement of ADMA and autografts both produce significant gains in root coverage, but coverage by autografts appears to be superior to that provided by ADMA<sup>22,23</sup>.

In this case, use of an ADMA shortened the procedure time and eliminated the need for palatal donor sites for this relatively large soft tissue graft. Both of these factors were critical for acceptance of the surgical procedure by the patient and her parents. As this ADMA product has an excellent record of safety with respect to disease transmission, the risks associated with its use were minimal. The graft enhanced tissue thickness gingiva facial to the mandibular anterior teeth while producing relatively little post-operative discomfort. During the 28-month course of periodontal and orthodontic treatment, the mean width of the keratinised gingiva in the mandibular anterior region increased by approximately 1.9 mm. In the absence of soft tissue grafting, increases in keratinised gingiva may occur on some teeth during the course of orthodontic treatment, but this is rare in the mandibular anterior region<sup>2</sup>. However, the width of keratinised gingiva can increase with age, in parallel with tooth eruption<sup>24</sup>. Thus, it is possible that a portion of the observed increase in keratinised gingiva may be attributable to growth and not the grafting procedure. Plaque-induced inflammation developed around the mandibular anterior teeth during orthodontic treatment, but there was no loss of attachment. Whereas the pretreatment inclination of the mandibular incisors was very close to that associated with an increased risk of recession, it should be noted that differentially greater mandibular growth and lateral expansion in the mandibular premolar region made it unnecessary to permanently increase the inclination of the incisors during orthodontic treatment. Without this unanticipated mandibular growth and the gingival augmentation provided by the graft, there could have been an increased risk of recession during correction of the mandibular crowding. The tissue graft helped manage this risk, and use of an ADMA made the option to graft more acceptable to the patient.

## ■ References

1. Maynard JG Jr, Ochsenein C. Mucogingival problems, prevalence and therapy in children. *J Periodontol* 1975; 46:543-552.
2. Coatoam GS, Behrents RG, Bissada NF. The width of keratinized gingiva during orthodontic treatment: its significance and impact on periodontal status. *J Periodontol* 1981;52:307-313.
3. Pearson LE. Gingival height of lower central incisors orthodontically treated and untreated. *Angle Orthodontist* 1968; 38:337-339.
4. Maynard JG. The rationale for mucogingival therapy in the child and adolescent. *Int J Periodontics Restorative Dent* 1987;7:36-51.
5. Pini Prato G, Baccetti T, Giorgetti R, Agudio G, Cortellini P. Mucogingival interceptive surgery of buccally erupted premolars in patients scheduled for orthodontic treatment. II. Surgically treated versus non-surgically treated cases. *J Periodontol* 2000;71:182-187.
6. Hall WB. The current status of mucogingival problems and their therapy. *J Periodontol* 1981;52:569-575.
7. Wennstrom JL, Lindhe J, Sinclair F, Thilander B. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol* 1987;14:121-129.
8. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol* 1981;52:314-320.
9. Lang NP, Loe H. The relationship between the width of keratinized gingiva and gingival health. *J Periodontol* 1972; 43:623-627.
10. Ngan PW, Burch JG, Wei SH. Grafted and ungrafted labial gingival recession in pediatric orthodontic patients: effects of retraction and inflammation. *Quintessence Int* 1991;22: 103-111.
11. Yared KFG, Zenobio EG, Pacheco W. Periodontal status of mandibular central incisors after orthodontic proclination in adults. *Am J Orthod Dentofacial Orthop* 2006;130:6.e1-6.e8.
12. Baker DL, Seymour GJ. The possible pathogenesis of gingival recession. A histological study of induced recession in the rat. *J Clin Periodontol* 1976;3:208-219.
13. Loe H. The gingival index, the plaque index and the retention index systems. *J Periodontol* 1967;38(suppl):610-616.
14. Reiser GM, Bruno JF, Mahan PE, Larkin LH. The subepithelial connective tissue graft palatal donor site: anatomic considerations for surgeons. *Int J Periodontics Restorative Dent* 1996;16:130-137.
15. Cortes A, Martins AG, Nociti FH Jr, Sallum AW, Casati MZ, Sallum EA. Coronally positioned flap with or without acellular dermal matrix graft in the treatment of Class I gingival recessions: a randomized controlled clinical study. *J Periodontol* 2004;75:1137-1144.
16. Woodyard JG, Greenwell H, Hill M, Drisko C, Isabella JM, Scheetz J. The clinical effect of acellular dermal matrix on gingival thickness and root coverage compared to coronally positioned flap alone. *J Periodontol* 2004;75:44-56.
17. Aichelmann-Reidy ME, Yukna RA, Evans GH, Nasr HF, Mayer ET. Clinical evaluation of acellular allograft dermis for the treatment of human gingival recession. *J Periodontol* 2001;72:998-1005.
18. Wagshall E, Lewis Z, Babich SB, Sinensky MC, Hochberg M. Acellular dermal matrix allograft in the treatment of mucogingival defects in children: Illustrative case report. *J Dent Child* 2002;69:39-43.
19. Fowler EB, Francis PO, Goho C. Use of acellular dermal matrix allograft for management of inadequate attached gingiva in a young patient. *Military Med* 2003;168:261-265.
20. Novaes AB Jr, Grisi DC, Molina GO, Souza SL, Taba M Jr, Grisi MF. Comparative 6-month clinical study of a subepithelial connective tissue graft and acellular dermal matrix graft for the treatment of gingival recession. *J Periodontol* 2001;72:1477-1484.
21. Paolantonio M, Dolci M, Esposito P, D'Archivio D, Lisanti L, Di Luccio A, Perinetti G. Subpedicle acellular dermal matrix graft and autogenous connective tissue graft in the treatment of gingival recessions: a comparative 1-year clinical study. *J Periodontol* 2002;73:1299-1307.
22. Harris RJ. A short-term and long-term comparison of root coverage with an acellular dermal matrix and a subepithelial graft. *J Periodontol* 2004;75:734-743.
23. Hirsch A, Goldstein J, Goultchin J, Boyan BD, Schwartz Z. A 2-year follow-up of root coverage with subpedicle acellular dermal matrix allografts and subepithelial connective tissue autografts. *J Periodontol* 2005;76:1323-1328.
24. Ainamo J, Talari A. The increase with age of the width of the attached gingiva. *J Periodontal Res* 1976;11:182-188.

