A Novel Surgical Approach to Modify the Periodontal Phenotype for the Prevention of Mucogingival Complications Related to Orthodontic Treatment

Tali Chackartchi, DMD[1]
Ruth Gleis, DMD[2]
Anton Sculean, DMD, MS, PhD[3]
Myron Nevins, DDS[4]

Certain bone morphologies and soft tissue thickness (ie, phenotype) are considered to be risk factors for the development of gingival recessions following orthodontic tooth movement. Preoperative evaluation of the periodontal phenotype, in the frame of orthodontic treatment plan, identify teeth at high risk for mucogingival complications related to orthodontic therapy. The new surgical technique is illustrated in a clinical case. A patient with a thin phenotype without visible gingival recession had bone dehiscences in the anterior mandible. Prior to orthodontic treatment, simultaneous bone and soft tissue augmentation was performed using the combination of a highly cross-linked ribose porcine type I collagen membrane and a subepithelial palatal connective tissue graft. Two years after augmentation surgery and initiation of orthodontic treatment, a thick buccal tissue with a wide band of attached gingiva was observed without any clinical signs of root prominences, indicating a substantial change in periodontal phenotype. The clinical findings were corroborated by the 3D analysis, demonstrating substantial bone apposition on the buccal aspect of all roots in the treated area. The described surgical technique offers a valuable approach for regenerating hard and soft tissues in deficient areas prior to orthodontic therapy, thus preventing the development of gingival recessions. Int J Periodontics Restorative Dent 2021;41:811–817. doi: 10.11607/prd.5774

[1]Department of Periodontology, Faculty of Dental Medicine, Hebrew University-Hadassah, Jerusalem, Israel.
[2]Department of Orthodontics, Tel Aviv University, Tel Aviv, Israel.
[3]Department of Periodontology, School of Dental Medicine, University of Bern, Bern, Switzerland.
[4]Division of Periodontology, Department of Oral Medicine, Infection, and Immunity, Harvard School of Dental Medicine, Boston, Massachusetts, USA.

Correspondence to: Dr Tali Chackartchi, Department of Periodontology, The Hebrew University-Hadassah Medical Center, P.O. Box 12272, Jerusalem 91120, Israel. Fax: +972-2-6438705. Email: Tali222@hotmail.com

The term periodontal phenotype refers to the characteristics of the bone and its overlying gingival phenotype, including keratinized tissue width and gingival and mucosal tissue thickness.[1] Alveolar bone dehiscence[2] and alveolar mucosa thickness[3,4] are considered to be factors contributing to the development of gingival recessions. Orthodontic tooth movement changes the buccal or lingual tooth position, thus potentially causing or worsening alveolar bone defects such as dehiscences or fenestrations, affecting periodontal support.[5,6] Retrospective studies analyzing the possible effect of orthodontic treatment on the development of gingival recessions indicate mandibular incisors to be more prone to developing gingival recession.[7,8] These teeth demonstrate a high prevalence of alveolar bone defects.[9]

Bone augmentation techniques were suggested to enhance buccolingual dimensions of the anterior portion of the ridge prior to orthodontic tooth movement.[10,11] Soft tissue augmentation is performed after orthodontic treatment.

In 2007, a porcine type I collagen membrane, cross-linked with ribose, was histologically proven to undergo ossification when placed over an intact bone surface in a preclinical investigation.[12] Similar findings of ongoing ossification have been observed in humans.[13]

Fig 1 *Clinical views at the start of treatment.*

This current case report presents a surgical technique designed to prevent mucogingival complications following orthodontic tooth movement. An ossifying collagen membrane, fixed directly on bone, is combined with a subepithelial connective tissue graft, thus enabling bone and soft tissue augmentation, changing the periodontal phenotype.

Case Description

A 28-year-old woman with noncontributory medical history was referred to a periodontist 2 weeks after placement of orthodontic appliances. The patient arrived with a full set of periapical radiographs indicating no sign of interproximal bone loss. The periodontal examination indicated a stable periodontal condition, with periodontal pocket depth (PPD) < 4 mm, a plaque score of 14%, and a bleeding score of 16%. The clinical appearance of the mandibular anterior area demonstrated prominent roots ("wash board" appearance), indicating possible alveolar bone defects. The overlaying gingiva had a thin phenotype (Fig 1) and a narrow band of keratinized tissue, especially on the central incisors and canines. Subsequent to the clinical evaluation, a CBCT scan was performed to evaluate bone morphology (Fig 2). The bone imaging results and the clinical examination led to a periodontal-orthodontic consultation. Considering the planned direction of tooth movements, preparatory surgery was planned to augment hard and soft tissues. In the 3-week interval between the initial examination and the day of surgery, recession had already begun on the right lateral incisor (Fig 1b).

Surgical Procedure

A full-thickness mucoperiosteal buccal flap was elevated with two vertical releasing incisions to gain an adequate exposure to the surgical field and avoid any tension. The elevated flap was split at its base, separating the periosteum from the overlying mucosa (Fig 3). Bone dehiscence over the involved roots was confirmed. Subsequently, a 1.5-mm–thick palatal subepithelial connective tissue graft (SCTG) was harvested by means of the single incision technique, and closure of the donor site was performed using modified mattress sutures. After mechanical debridement of the exposed roots, the roots were rinsed with sterile saline. A $1 \times 4 \times 2$–mm crossed-linked membrane (OSSIX Volumax, Datum Dental) was sutured to the exposed root surfaces using internal cross-mattress sutures, with a 5/0 polyglactin resorbable suture (Coated Vicryl, Ethicon, Johnson & Johnson) extending from the inner periosteal flap to the lingual papillae (Fig 4). The SCTG was adapted on the cross-linked membrane at the level of the cementoenamel junction using a 6/0 polyglycolic acid resorbable sling suture (Stoma PGA, Stoma; Fig 5). Finally, the buccal flap was coronally repositioned over the graft and membrane, then sutured with 6/0 nylon sling sutures (Nylon, Ethicon, Johnson & Johnson) and single interrupted 5/0 polytetrafluoroethylene sutures (OsseoGuard, Zimmer Biomet) to accomplish tension-free complete coverage of the graft and denuded roots. The vertical incisions were also sutured using 6/0 nylon sutures by applying simple interrupted sutures (Fig 6).

Fig 2 (a) CBCT scans taken of the mandibular arch. (b) Anterior teeth sections taken at the beginning of therapy to evaluate bone morphology in 3D.
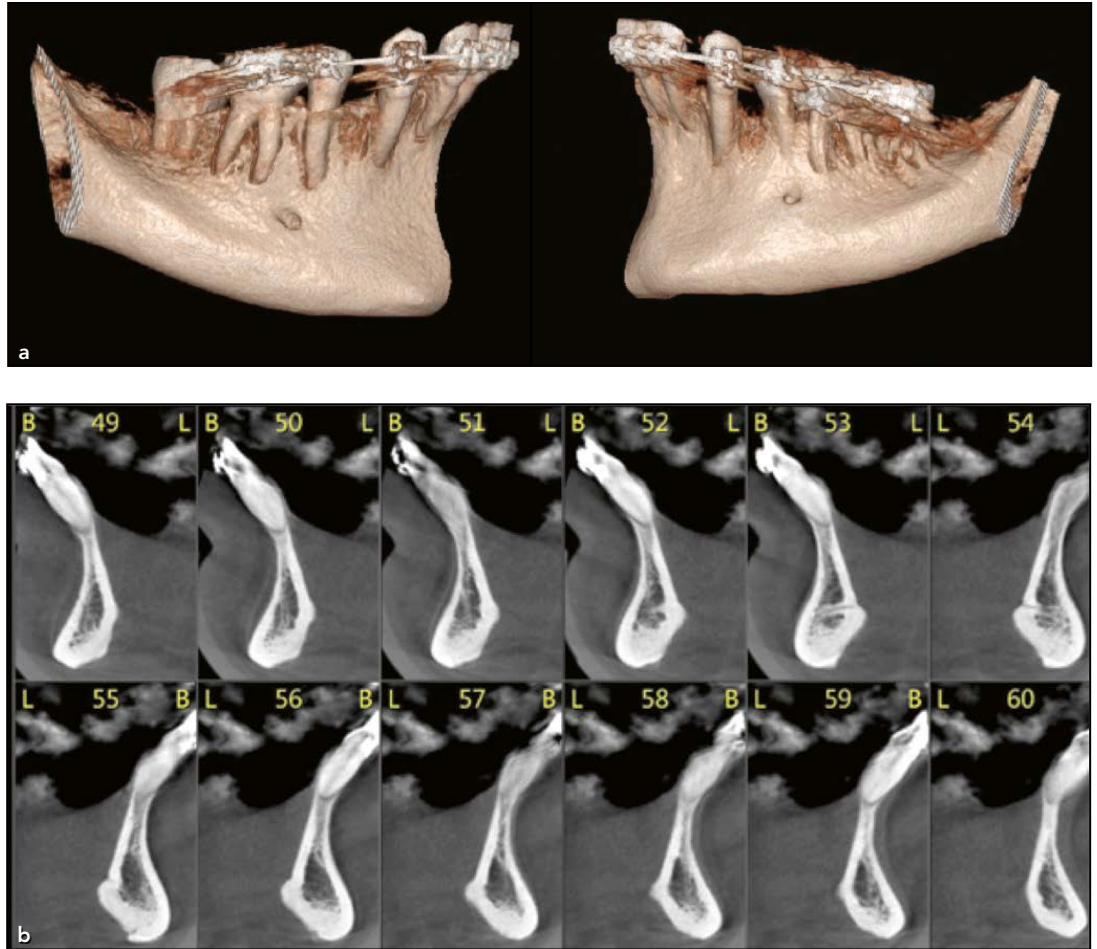

Fig 3 A full-thickness buccal flap was reflected. The elevated flap was split at its base, separating the periosteum and muscle insertions from the overlying mucosa.
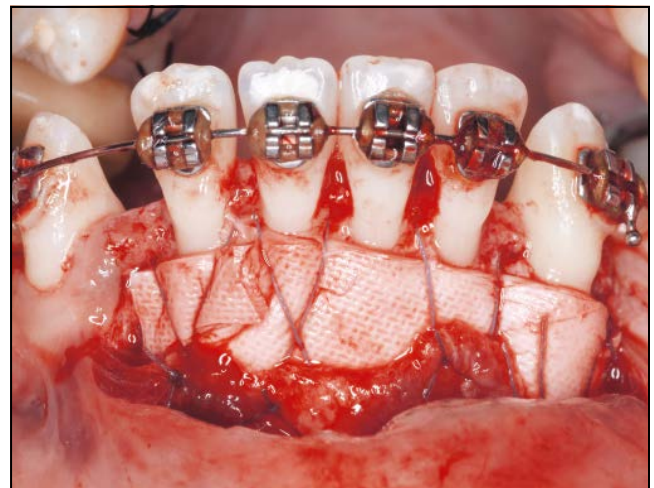
Fig 4 A ribose-crossed linked membrane adapted to the exposed root surfaces using internal cross-mattress sutures.
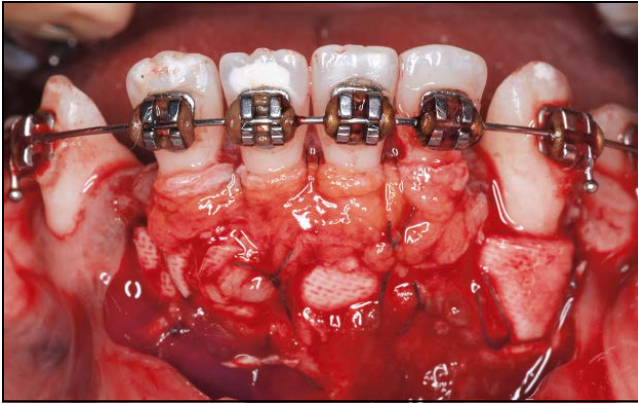
Fig 5 *The subepithelial connective tissue graft was adapted over the cross-linked membrane at the level of the cementoenamel junction using a 6/0 resorbable sling suture.*
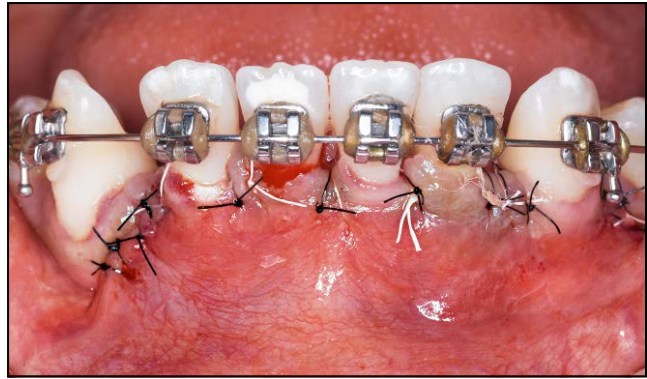

Fig 6 *The buccal flap was coronally repositioned over the graft and the membrane, then sutured with 6/0 nylon sling sutures and simple 5/0 polytetrafluoroethylene sutures to accomplish tension-free complete coverage of the graft and the denuded roots. The vertical incisions were sutured using 6/0 nylon sutures with a simple interrupted technique.*
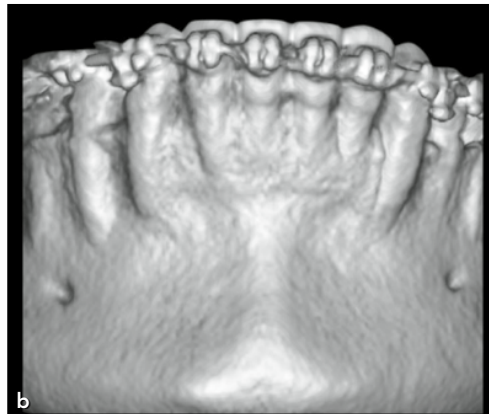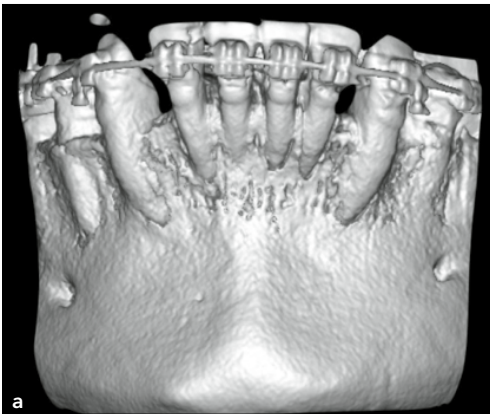

Fig 7 *3D reproduction of bone (a) at the start of treatment and (b) 2 years postsurgery, demonstrating bone apposition on the buccal aspect of roots of the anterior teeth.*

Postoperative Care

Postsurgically, the patient was prescribed analgesics (400 mg ibuprofen [Ibufen, Dexcel] twice a day for 2 to 3 days) and antibiotics (500 mg amoxicillin [Moxypen Forte, Teva] for 7 days) to prevent infection. The patient was instructed to avoid toothbrushing of the surgical sites for 21 days postoperatively and was advised to use a 0.2% chlorhexidine-digluconate mouth rinse (Corsodyl, GlaxoSmithKline) twice a day for 1 minute for the first 21 days postsurgery. The patient resumed toothbrushing 21 days after surgery. The palatal sutures were removed 7 days after surgery, while those from the treated teeth were removed 21 days postoperatively. At that time, the patient was instructed to perform mechanical cleaning of the surgical site using an ultra-soft electric toothbrush (Smart 4000S, Oral-B). Periodontal recall appointments included professional supragingival tooth debridement every 2 months.

Two years postsurgery, the CBCT indicated substantial buccal bone apposition (Fig 7). The clinical evaluation revealed a stable thick buccal tissue with a wide band of attached gingiva without any signs of root prominences (Fig 8).

Orthodontic Treatment

Two mandibular first premolars were extracted prior to orthodontic treatment to enable upright positioning

Fig 8 *Facial view 2 years after the initiation of orthodontic tooth movement, demonstrating thick buccal tissue, a wide band of attached gingiva, and no clinical signs of root prominence.*
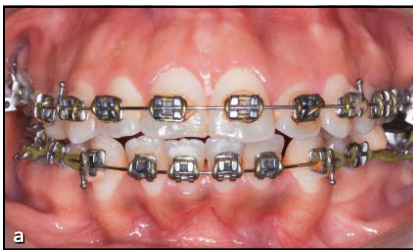

Fig 9 *Clinical views (a) at patient admission, (b) prior to orthognathic surgery (2 years after the initiation of orthodontic tooth movement), and (c) after orthognathic surgery.*

of the proclined mandibular anterior teeth. Orthodontic tooth movement was resumed 2 weeks after surgery at an accelerated pace for 4 months, following the protocol suggested by Wilcko et al,[11] taking advantage of the transient osteopenia induced by the surgical procedure.[14] After 24 months of orthodontic tooth movement, orthognathic surgery was performed, including maxillary Lefort I osteotomy with maxillary impaction and 4-mm mandibular advancements using a bilateral sagittal split osteotomy technique.[15]

Treatment Results

The orthodontic treatment enabled coordination of the two arches and established an overjet in accordance with the orthosurgical plan (maxillary impaction and advancement of the mandible). Despite the limited buccolingual bone volume at the start of orthodontic tooth movement, the clinical parameters monitored throughout the course of the treatment indicated periodontal stability. PPD was unchanged (< 4 mm), and the plaque and bleeding scores did

not exceed 20% throughout the entire treatment period. The change in gingival phenotype obtained by means of the surgical augmentation was stable during the entire duration of orthodontic tooth movement, without any signs of gingival recession (Fig 9).

Discussion

Periodontal evaluation of patients prior to orthodontic tooth movement usually relates to the presence

or absence of periodontal disease and inflammation and should also include anatomical considerations. Despite the well-established anatomical risk factors for the development of recessions related to orthodontic tooth movement (such as bone morphology, gingival thickness, and muscle pull),[16] there are no established protocols to evaluate the risk of recessions following orthodontic tooth movements. Final tooth inclination is often overlooked in the preorthodontic risk assessment.[16] There is a clear tendency to treat gingival recessions after their development and not as a preventive treatment.

Alveolar bone morphology plays an important role in proper planning of orthodontic treatment, as it might limit tooth movement.[17,18] Lack of bone support on the buccal aspect of the roots might lead to mucogingival deformities.[19–22] Alveolar bone defects may develop or worsen after orthodontic tooth movement.[5,6] Two-dimensional radiographs do not have the ability to reflect buccal bone morphology. Therefore, 3D radiographs, such as CBCT scans, are recommended prior to orthodontic tooth movement.[23] A majority of the techniques suggested for the prevention of mucogingival complications following orthodontic tooth movement are related to hard tissue augmentation. Different bone augmentation techniques were suggested to enhance buccolingual dimensions of the anterior ridge prior to orthodontic tooth movement.[10] The most established technique is the periodontally accelerated osteogenic orthodontics (PAOO) in-troduced by Wilcko et al,[11] which combines selective alveolar bone decortication and bone augmentation with particulate grafting material. Interestingly, it was reported that bone augmentation following PAOO enhanced keratinized tissue width,[24] demonstrating the essential reciprocal relations between bone and soft tissue dimensions. Alveolar bone morphology is not frequently discussed as a factor potentially influencing the treatment plan and outcomes in mucogingival cases, but it might contribute to short-term complications and long-term stability of treatment. These teeth might be at further risk if covered by thin soft tissue, typical in the mandibular anterior area.

The 3D architecture of the tissues was evaluated in the present study, considering the planned direction of tooth movement to formulate a combined periodontal-orthodontic treatment plan. This offered a surgical approach for simultaneous enhancement of bone and gingival tissues using a combination of an ossifying ribose cross-linked collagen membrane and a connective tissue graft. A highly cross-linked ribose porcine type I collagen membrane was introduced in 2007 for guided bone regeneration procedures.[12] Histologic analysis of a preclinical investigation demonstrated that the membrane underwent total ossification within 6 months. Despite the fact that the ossification mechanisms have not been explored yet, analysis of the histologic sections over time (ie, at 2, 4, and 6 months) revealed direct apposition of the calcified mate-rial on the original glycated, cross-linked collagen matrix.[12] This direct mineral apposition on the glycated collagen was further confirmed in humans.[25]

The postoperative CBCT scan in the present case supports the bone formation enhanced by the use of this ossifying membrane. Combining bone augmentation with soft tissue augmentation in the same procedure enabled a change in the periodontal phenotype. The induction of the transient osteopenia and bone remodeling derived from the surgical procedure[26] are probably responsible for the formation of a wide bony envelope for tooth movement, simultaneously preserving periodontal stability.

Conclusions

The present case treatment highlights the advantages of a combined periodontic-orthodontic consultation, and of thorough clinical and radiographic analyses prior to orthodontic tooth movement, to design the optimal treatment strategy that avoids possible mucogingival complications. The described surgical technique appears to offer a valuable approach for regenerating hard and soft tissues in deficient areas prior to orthodontic therapy, thus preventing the development of gingival recessions. Long-term follow-up studies and comparative studies are needed to further establish the advantages of the presented technique.

Acknowledgments

References

1. Jepsen S, Caton JG, Albandar JM, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Periodontol 2018;89(suppl):s237–s248.

2. Wennström JL. Mucogingival therapy. Ann Periodontol 1996;1:671–701.

3. Susin C, Haas AN, Oppermann RV, Haugejorden O, Albandar JM. Gingival recession: Epidemiology and risk indicators in a representative urban Brazilian population. J Periodontol 2004;75:1377–1386.

4. Lovegrove J, Leichter J. Exposed root surface: A review of aetiology, management and evidence-based outcomes of treatment. N Z Dent J 2004;100:72–81.

5. Löst C. Depth of alveolar bone dehiscences in relation to gingival recessions. J Clin Periodontol 1984;11:583–589.

6. Wennström JL, Lindhe J, Sinclair F, Thilander B. Some periodontal tissue reactions to orthodontic tooth movement in monkeys. J Clin Periodontol 1987;14:121–129.

7. Renkema AM, Fudalej PS, Renkema A, Kiekens R, Katsaros C. Development of labial gingival recessions in orthodontically treated patients. Am J Orthod Dentofac Orthop 2013;143:206–212.

8. Renkema AM, Fudalej PS, Renkema AA, Abbas F, Bronkhorst E, Katsaros C. Gingival labial recessions in orthodontically treated and untreated individuals: A case - control study. J Clin Periodontol 2013;40:631–637.

9. Rupprecht RD, Horning GM, Nicoll BK, Cohen ME. Prevalence of dehiscences and fenestrations in modern American skulls. J Periodontol 2001;72:722–729.

10. Ma Z, Zheng J, Yang C, Xie Q, Liu X, Abdelrehem A. A new modified bone grafting technique for periodontally accelerated osteogenic orthodontics. Medicine (Baltimore) 2018;97:e12047.

11. Wilcko WM, Wilcko T, Bouquot JE, Ferguson DJ. Rapid orthodontics with alveolar reshaping: Two case reports of decrowding. Int J Periodontics Restorative Dent 2001;21:9–19.

12. Zubery Y, Goldlust A, Alves A, Nir E. Ossification of a novel cross-linked porcine collagen barrier in guided bone regeneration in dogs. J Periodontol 2007;78:112–121.

13. Levin BP, Zubery Y. Use of a sugar cross-linked collagen membrane offers cell exclusion and ossification. Compend Contin Educ Dent 2018;39:44–48.

14. Frost HM. The biology of fracture healing. An overview for clinicians. Part II. Clin Orthop Relat Res 1989:294–309.

15. Trauner R, Obwegeser H. The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Part I. Surgical procedures to correct mandibular prognathism and reshaping of the chin. Oral Surg Oral Med Oral Pathol 1957;10:677–689.

16. Yared KF, Zenobio EG, Pacheco W. Periodontal status of mandibular central incisors after orthodontic proclination in adults. Am J Orthod Dentofac Orthop 2006;130:6.e1–6.e8.

17. Ghafari JG, Ammoury MJ. Overcoming compact bone resistance to tooth movement. Am J Orthod Dentofac Orthop 2020;158:343–348.

18. Proffit W, Fields H. Contemporary Orthodontics, ed 5. Elsevier: Amsterdam, 2014.

19. Wainwright WM. Faciolingual tooth movement: Its influence on the root and cortical plate. Am J Orthod 1973;64:278–302.

20. 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.

21. Dorfman HS. Mucogingival changes resulting from mandibular incisor tooth movement. Am J Orthod 1978;74:286–297.

22. Årtun J, Krogstad O. Periodontal status of mandibular incisors following excessive proclination. A study in adults with surgically treated mandibular prognathism. Am J Orthod Dentofac Orthop 1987;91:225–232.

23. Kapila SD, Nervina JM. CBCT in orthodontics: Assessment of treatment outcomes and indications for its use. Dentomaxillofacial Radiol 2015;44:20140282.

24. Wilcko MT, Ferguson DJ, Makki L, Wilcko WM. Keratinized gingiva height increases after alveolar corticotomy and augmentation bone grafting. J Periodontol 2015;86:1107–1115.

25. Zubery Y, Nir E, Goldlust A. Ossification of a collagen membrane cross-linked by sugar: A human case series. J Periodontol 2008;79:1101–1107.

26. Yaffe A, Fine N, Binderman I. Regional accelerated phenomenon in the mandible following mucoperiosteal flap surgery. J Periodontol 1994;65:79–83.