Concise Clinical Review

Treatment of Gingival Recession: When and How?

Jean-Claude Imber [a,b], *Adrian Kasaj* [a*]

[a] *Department of Periodontology and Operative Dentistry, University of Mainz, Mainz, Germany*
[b] *Department of Periodontology, University of Bern, Bern, Switzerland*

ARTICLE INFO

Article history:
Available online 4 September 2020

Key words:
Gingival recession
Mucogingival surgery
Soft tissue management

ABSTRACT

Gingival recession is a common finding in daily clinical practice. Several issues may be associated with the apical shift of the gingival margin such as dentine hypersensitivity, root caries, non-carious cervical lesions (NCCLs), and compromised aesthetics. The first step in an effective management and prevention program is to identify susceptibility factors and modifiable conditions associated with gingival recession. Non-surgical treatment options for gingival recession defects include establishment of optimal plaque control, removal of overhanging subgingival restorations, behaviour change interventions, and use of desensitising agents. In cases where a surgical approach is indicated, coronally advanced flap and tunnelling procedures combined with a connective tissue graft are considered the most predictable treatment options for single and multiple recession defects. If there is a contraindication for harvesting a connective tissue graft from the palate or the patient wants to avoid a donor site surgery, adjunctive use of acellular dermal matrices, collagen matrices, and/or enamel matrix derivatives can be a valuable treatment alternative. For gingival recession defects associated with NCCLs a combined restorative-surgical approach can provide favourable clinical outcomes. If a patient refuses a surgical intervention or there are other contraindications for an invasive approach, gingival conditions should be maintained with preventive measures. This paper gives a concise review on when and how to treat gingival recession defects.

Introduction

Gingival recession or soft tissue recession is defined as the displacement of the gingival margin apical to the cemento-enamel junction (CEJ) of a tooth or the platform of a dental implant.[1,2] This condition is associated with the loss of periodontal tissues, including gingiva, periodontal ligament, root cementum, and bone at dental sites as well as the loss of mucosa and bone around dental implants. It has been suggested that the term 'periodontal recession' rather than 'gingival recession' would represent a more accurate definition of this condition at teeth.[2] However, both terms are currently used synonymously.

It was demonstrated in several epidemiological studies that gingival recession is a common finding in daily clinical practice. The prevalence is ranging from 40% to 100% depending on both the population and the methods of analysis.[3–7] Gingival recession defects can either be localised or generalised and can be located at labial, lingual and/or interproximal tooth surfaces.[8]

The exposure of the root surface by gingival recession is frequently associated with dentine hypersensitivity, root caries, non-carious cervical lesions (NCCLs), compromised plaque control, and unaesthetic appearance.[9–11] Moreover, untreated gingival recession has a tendency for further apical displacement over time in spite of good patient motivation.[12,13] Nevertheless, not all patients with gingival recession are willing to undergo a surgical intervention to obtain soft tissue root coverage. Consequently, as clinicians we should select the right treatment approach considering patient- and tooth-related factors.

Therefore, the aim of this review is to provide a concise and up-to-date overview regarding how and when to treat gingival recession defects.

Classification and aetiology of gingival recession

For diagnosis and subsequent therapy, it is of importance to classify mucogingival conditions. According to the new

* *Corresponding author*. Adrian Kasaj, Department of Periodontology and Operative Dentistry, Johannes Gutenberg-University Mainz, Augustusplatz 2, 55131 Mainz, Germany.
E-mail address: kasaj@gmx.de (A. Kasaj).

https://doi.org/10.1111/idj.12617

classification scheme of periodontal and peri-implant diseases, gingival recession belongs to the category 'mucogingival deformities and conditions around teeth'.[14]

For many years and in most of the available clinical studies, gingival recession has been classified according to Miller[15]. In general, this classification proved to be very useful but also revealed some limitations over time.[16] Miller's classification system depends on the mucogingival junction (MGJ), the presence or absence of keratinised tissue (KT), and the interdental bone/soft tissue loss. For example, a gingival recession not exceeding the MGJ but with interdental bone/soft tissue loss does not fit in any of the existing Miller classes. Moreover, the MGJ is not always identifiable and usually teeth exhibit, even if not clinically visible, a minimal amount of KT. Furthermore, the Miller classification takes the position of the gingival margin of two adjacent teeth into account, and if missing, making a proper diagnosis can be difficult. Thus, this classification system does not cover all possible variations of recession defects. To make a prognosis of a root coverage procedure, it is essential to classify a recession defect properly while considering patient-, site- and technique-related factors.

Recently, a novel treatment-oriented classification has been proposed, which is based on the assessment of the gingival phenotype, the severity of the recession, and the presence of associated cervical lesions.[17] In a first step, the gingiva should be categorised into different phenotypes/biotypes. In general, three different categories can be distinguished.[18] Thin scalloped phenotypes show a higher association with a slender triangular-shaped crown, subtle cervical convexity, interproximal contacts close to the incisal edge and a narrow zone of KT, clear thin delicate gingiva, and a relatively thin alveolar bone. Thick flat phenotypes exhibit more square-shaped tooth crowns, pronounced cervical convexity, large interproximal contact points located more apically, a broad zone of KT, clear thick, fibrotic gingiva and a comparatively thick alveolar bone. Thick scalloped phenotypes show a clear thick fibrotic gingiva, slender teeth, narrow zone of KT and a high gingival scallop. Nevertheless, there are overlaps in definitions and the gingival phenotype can vary within the same individual and among different individuals.

Apart from classifying the gingival phenotype, it is also important to consider the recession depth and the gingival thickness.[17] A common way to distinguish between thick or thin tissue is to place a periodontal probe in the gingival sulcus and observe its transparency. If the probe is visible through the gingiva it is considered to be a thin phenotype, if not, it is usually a thick phenotype.[19] For treatment, these clinical measurements are of significance since the probability to obtain complete root coverage (CRC) following surgical procedures will be reduced with the depth of the recession.[20] Furthermore, in thin phenotypes (<1.1 mm) the results after root coverage procedures are less favourable[21]. Another key factor for final root coverage is the integrity of the interdental attachment. Based on interdental clinical attachment level measurements, gingival recessions can be classified into three different types[22] (Table 1).

After assessing the soft tissue, the condition of the tooth surface in the area of the gingival recession should also be evaluated. NCCLs are common findings in patients with gingival recession defects. Therefore, a classification for these defects has been introduced.[23] A tooth with an identifiable CEJ belongs to class A, while one with no identifiable CEJ belongs to class B. Each class is subdivided depending on the presence (+) or absence (−) of a cervical step over 0.5 mm, yielding four classes (A+, A−, B+, B−).

Recently, a classification for peri-implant soft tissue dehiscence/deficiency (PSTD) was also proposed for single implants in the aesthetic zone.[24] Based on the level of the soft tissue margin compared to the gingival margin of the homologous tooth and the bucco-lingual position of the implant head, four different classes of PSTD were introduced. All classes were further divided based on the height of the anatomical papillae into two (class I) or three (class II–IV) subclasses.[24] There are several factors which can lead to mucosal recession on peri-implant sites such as lack of a certain amount of keratinised mucosa,[25] immediately placed implants,[26,27] buccal placement of the implant,[28] and surgeon experience.[29]

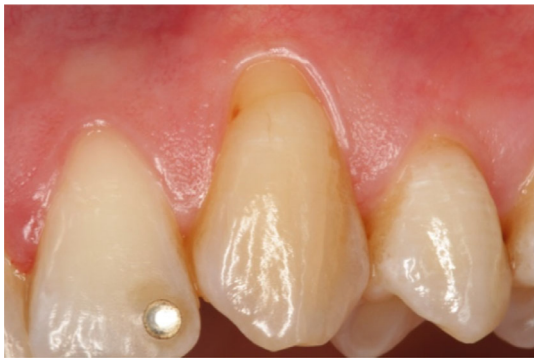
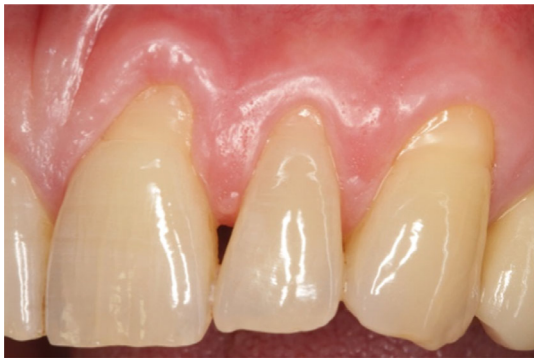
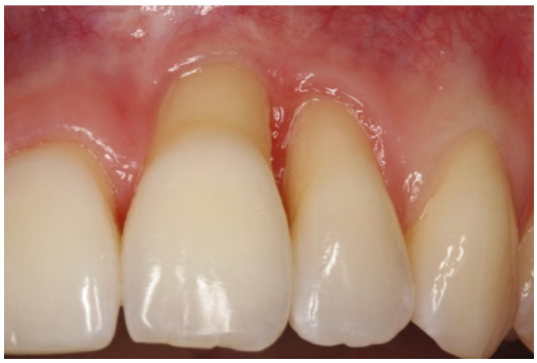
Before considering treatment of gingival recession defects, it is important to identify aetiological factors. At present, the true aetiology still remains unclear.[17] Thus, gingival recession defects frequently have multifactorial causes. Plaque-induced periodontal inflammation and mechanical trauma caused by improper tooth brushing habits can be considered as the most important aetiological factors.[30–32] Furthermore, the clinician should focus on susceptibility factors and modifiable conditions associated with gingival recession.[33] The major susceptibility factors are thin gingival tissue, absence or a narrow band (<2 mm) of KT, probing depths extending beyond the MGJ and a positive history of progressive gingival recession and/or inflammatory periodontal disease. Modifiable conditions are plaque accumulation, inflammatory periodontal disease, aberrant frenum position, traumatic oral hygiene habits, subgingival restoration margins, smoking, and systemic conditions like diabetes.[33] Therefore, the clinician should identify these factors and, if possible, modify them accordingly. In addition, an effective management and prevention program should help to improve patient awareness about gingival recession and to adopt adequate oral hygiene measures.

Non-surgical management of gingival recession

Gingival recession is related to several undesirable conditions. The most obvious is of aesthetic nature, but dental hypersensitivity, a reduced resistance against pathogenic stimuli such as cariogenic bacteria, and compromised plaque control are further important factors leading to patient discomfort.[34] Although gingival augmentation is a well-studied, secure and efficient method resulting in pain relief and prevention of further progression,[35,36] patients are not always willing to undergo invasive surgical treatment. If this is the case, a cascade of subsequent steps should be followed as a guideline.

First, as already mentioned above, the underlying aetiology of the recession defect should be identified. Thus, traumatic oral hygiene habits can be identified when associated

Table 1 – Recession types.[22]

Recession Type 1 (RT1): 	Gingival recession with no loss of interproximal attachment. Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth.
Recession Type 2 (RT2): 	Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal sulcus/pocket) is less than or equal to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).
Recession Type 3 (RT3): 	Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is greater than the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).

CEJ, cemento-enamel junction.

with NCCLs. These defects have a round-shaped 'washed out' look, whereas sharp, chipped defects are usually associated with bruxism and occlusal stress.[37] Consequently, such clinical findings should lead to a focus on oral hygiene instruction and motivation. Furthermore, oral care products including toothpaste and toothbrush should be inspected. A soft or ultra-soft brush is less damaging to the soft tissues.[38] Moreover, an electric toothbrush with a force-detector could act as a simple and educational tool for patients when applying heavy forces.[39] In addition, toothpastes with a high abrasive factor (relative dental abrasity, RDA) should be exchanged for toothpastes with low RDA.[40] A supportive oral hygiene regime can help to prevent further recession.

In contrast to patient-related factors, dental restorations (e.g. composite, ceramic, metal) may further influence gingival health. The position of a direct or indirect restorative margin can interfere with the supracrestal connective tissue attachment (biological width). Therefore, subgingival restoration margins may lead to inflammation, and as a result, gingival recession can develop.[41] In addition, unpolished or overhanging surfaces tend to accumulate more plaque than smooth ones. Thus, careful preparation and finishing of restorations is essential to prevent the further development of gingival recession defects.[42]

If alveolar bone is lost due to periodontal disease, the gingival envelope or crevice moves in an apical direction. The vestibular bone lamella is very thin and can be resorbed in an

early stage of the disease, which could lead to smaller amounts of KT and/or attached tissue. Some studies have demonstrated that an insufficient band of KT along with missing attached gingiva is associated with poor plaque resistance and impaired access for oral hygiene.[43,44] Nevertheless, the majority of evidence supports the view that gingival health can be maintained independent of its dimensions.[45–47] Thus, gingiva with a narrow band of KT is considered to have the same ability to resist further attachment loss as gingiva with a wide zone of KT.[35]

Dentinal hypersensitivity is another common finding in patients with gingival recession defects. Exposure of dentinal tubules results in hydrodynamic liquid flow that activates pain sensation. The hydrodynamic theory was proposed by Brännström and Åström and is the most rational theory to date.[48] If a patient is suffering from dentinal hypersensitivity, the first step should include the use of a desensitising toothpaste. Calcium sodium phosphosilicate, arginine and calcium carbonate, and strontium are ingredients that tend to occlude the dentinal tubules and prevent fluid movement. As a consequence, a decrease in activation of nociceptors will result.[49–51] Moreover, other available desensitisers contain glutaraldehyde, which is a biological fixative leading to coagulation of plasma proteins and tubular blockage.[52] Potassium nitrate is another desensitising agent with a different mode of action. This additive depolarises the nerve surrounding the odontoblast, thus reducing the action potential and therefore the dental nociception.[53,54] Furthermore, a high fluoride application over time results in apposition of secondary dentin possibly leading to dentinal occlusion.[55] It is proven that all these dentifrices require some time before the patient notices any pain relief.[53] If interventions with different desensitisers do not achieve satisfactory pain relief, bonding agents can be carefully used to seal the dentinal tubules.

A different approach to treat dentinal hypersensitivity is based on laser therapy. Low intensity laser treatment aims to depolarise the cells in order to avoid exceeding the neurobiological threshold of the membrane's potential. On the other hand, high intensity laser treatment occludes dentinal tubules.[56,57] Based on clinical findings, lasers are showing results as good as other desensitising strategies.[58] Nevertheless, if these therapy options are not successful, the last step is endodontic treatment.

Current surgical techniques for treating gingival recession

The primary goal of any surgical approach for treating gingival recession defects will be CRC. Nevertheless, CRC is not always achievable and therefore, patients need to be informed about the chances to reach optimal clinical outcomes.

The most widely used techniques to treat gingival recession defects are the so-called pedicle flap surgical techniques (coronally advanced or rotational flaps). Norberg was the inventor of the coronally advanced flap (CAF) technique.[59] Thereafter, the CAF was modified several times. Currently, for single recession defects, a flap with a trapezoid design is the treatment of choice.[60] This surgical procedure includes two vertical releasing incisions and a split-full-split approach to overcome the problem with blood supply and the formation of scar tissue. Moreover, for multiple recession defects a modified CAF procedure was proposed by Zucchelli and De Sanctis.[61] This technique requires no vertical releasing incisions, and therefore, a good blood supply of the flap will be maintained (Figures 1–5).

Other options for treating gingival recession defects are the so-called tunnel techniques.[62,63] The tunnel can be prepared in

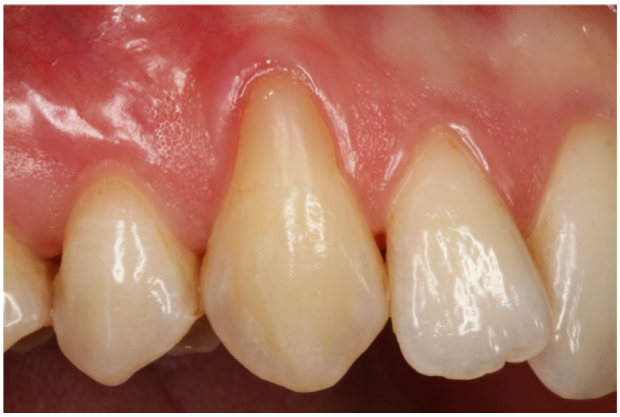


Fig. 1 – Baseline tooth 13 (Recession Type 2).

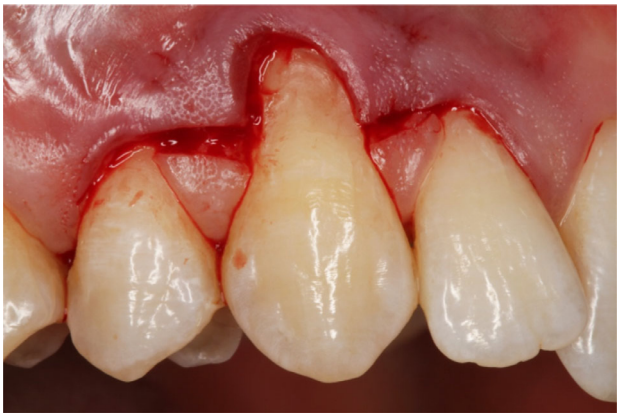


Fig. 2 – After incision and flap elevation.

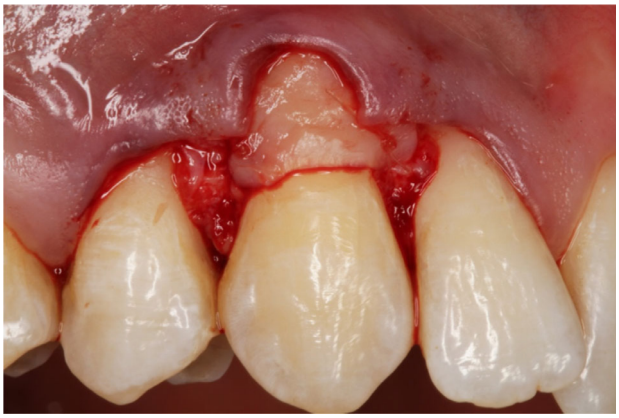


Fig. 3 – Connective tissue graft.

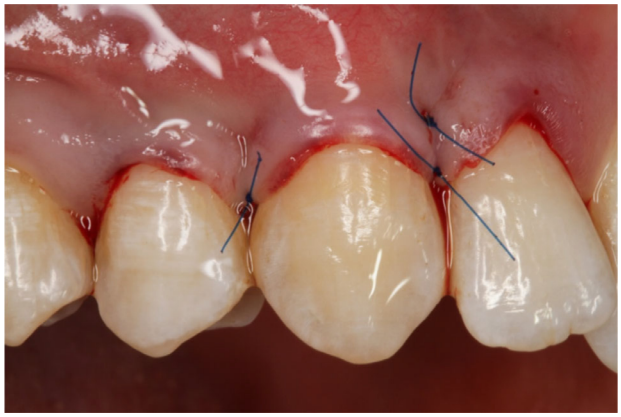


Fig. 4 – Flap closure with sling sutures.

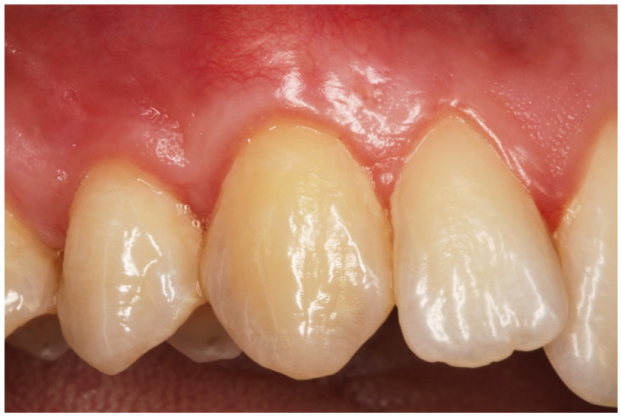


Fig. 5 – Outcome after 3 months.

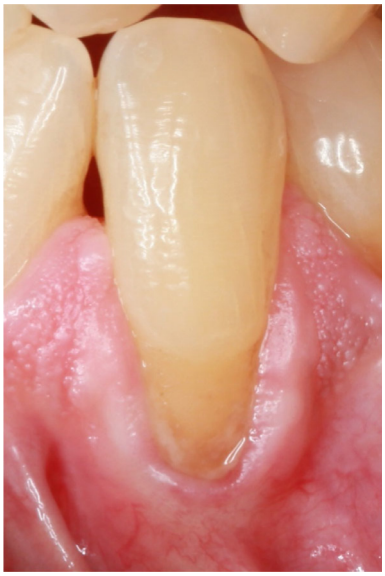


Fig. 6 – Baseline tooth 33 (Recession Type 2).

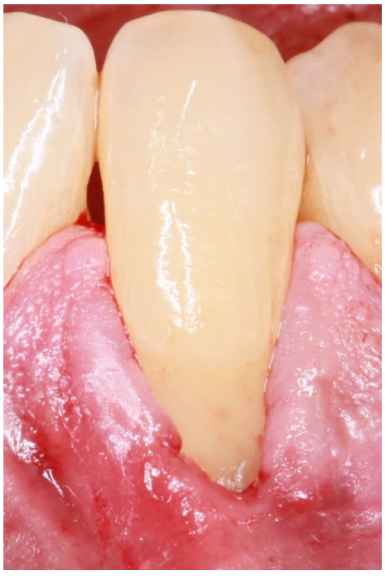


Fig. 7 – After full-thickness tunnel preparation.

a full-thickness or split-thickness manner depending on the soft tissue dimensions. In most cases, the tissues are thin, and therefore, a full-thickness flap design is the safer approach to avoid perforations and ruptures. Good results can be obtained with the use of a modified coronally advanced tunnel (MCAT) technique in single and multiple gingival recession defects[64–68] (Figures 6–10). More recently, the technique was modified by closing the tunnel laterally using simple sling or double sling sutures.[69] This modification is mainly indicated in deep and narrow recession defects (Figures 11–15).

The CAF has also been proposed in combination with a sub-epithelial connective tissue graft (CTG). The indication to perform a CAF without a CTG is given if the KT height apical to the root exposure is more than 2 mm[70] and the soft tissue thickness is more than 0.84 mm[71]. Furthermore, there are anatomical factors that influence the indication and the efficacy of a CAF without a CTG.[72] It is not advisable to use a CAF without a CTG if interdental clinical attachment loss is diagnosed and/or only a minimal amount of KT apical to the gingival recession is present. A high frenulum insertion at the soft tissue margin or a very shallow vestibulum could be additional limitations. Furthermore, if the root is dislocated buccally or the root has a deep cervical defect, a CTG should be used in conjunction with the CAF.[72] As arguments against harvesting palatal connective tissue, however, there may be limited availability, in addition to the prolonged surgical time involved and an associated increase in patient morbidity.[73] Recently, a site-specific application of a CTG for the treatment of multiple gingival recession defects was proposed[74]. Thus, only teeth with the aforementioned limitations in KT height and gingival thickness receive a CTG. This approach allows reducing the amount of soft tissue graft and donor site morbidity. The CTG can also be used in combination with tunnel techniques providing good clinical results in terms of CRC and mean root coverage.[64,68,69,75,76] Two recent studies comparing the tunnel technique + CTG with CAF + CTG showed no significant differences in terms of CRC or mean root coverage.[77,78] In contrast, Santamaria et al. showed significantly

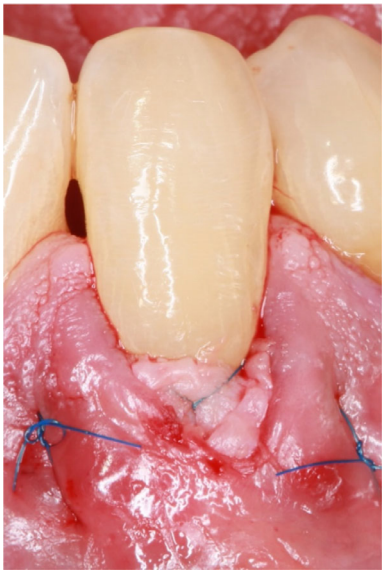


Fig. 8 – Connective tissue graft.

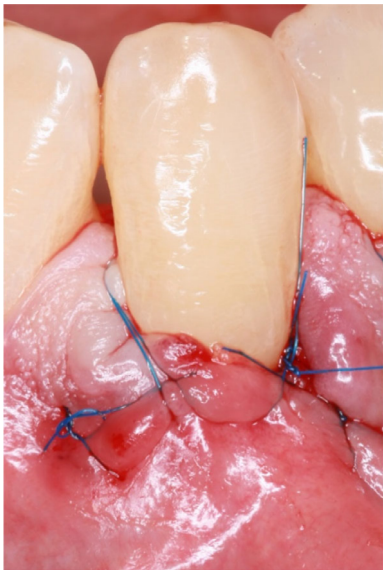


Fig. 9 – Flap closure with sling sutures.

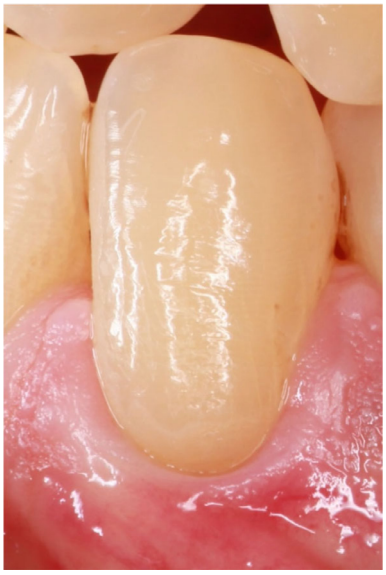


Fig. 10 – Outcome after 2 years.

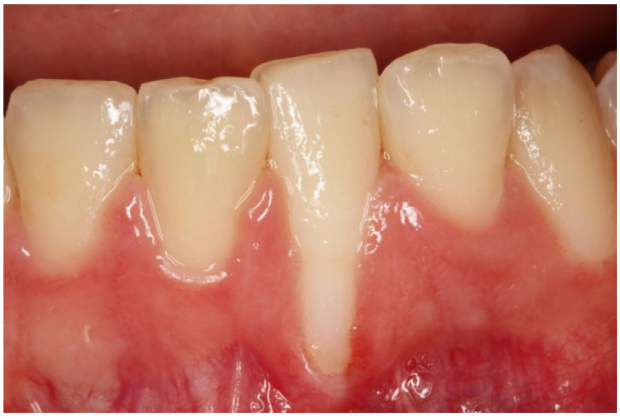


Fig. 11 – Baseline tooth 31 (Recession Type 1).

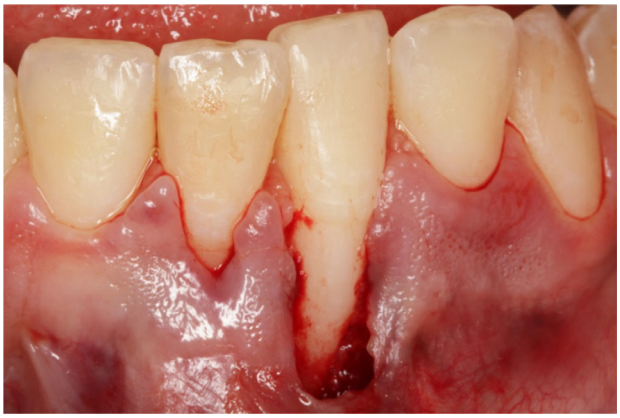


Fig. 12 – After full-thickness tunnel preparation.

superior clinical outcomes for CAF + CTG compared to tunnel technique + CTG in terms of CRC and mean root coverage, but more patients reported pain in the CAF group.[79]

Overall, the use of a CTG provides more long-term gingival stability and better root coverage outcomes compared to treatment with a CAF alone.[80]

In contrast to the CTG, free gingival grafts (FGG) are rarely recommended for root coverage procedures because of the low predictability and poor aesthetic outcome.[81] The portion of a FGG placed on the denuded root surface does not have a good blood supply, and therefore, graft shrinkage or early necrosis may occur.[70] To overcome the low predictability of root coverage with FGG, a two-step procedure was introduced.[82–84] In the first step, the FGG is used for gingival augmentation apical to the recession defect, followed by a CAF procedure to cover the root surface. More recently, this treatment approach was modified by minimising the

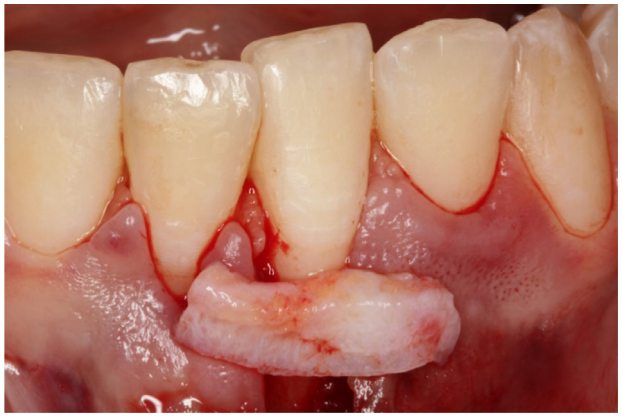


Fig. 13 – Connective tissue graft.

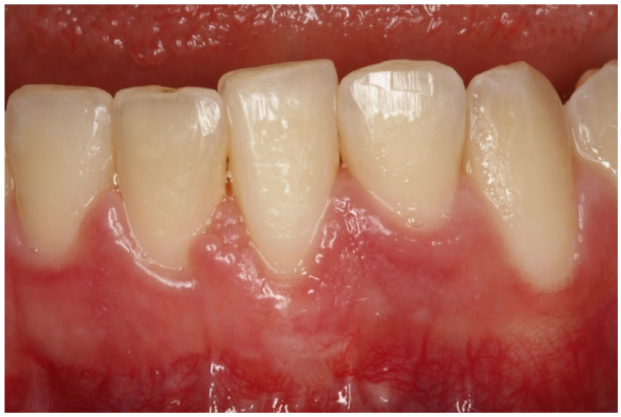


Fig. 15 – Outcome after 3 months.

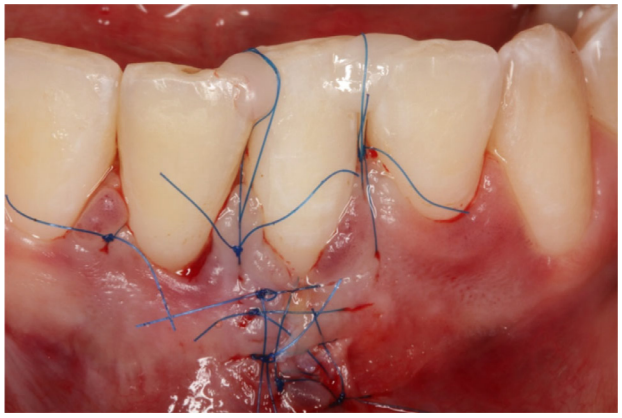


Fig. 14 – Flap closure with single interrupted sutures and sling sutures.

dimensions of the graft (same height of keratinisation as the adjacent teeth) and by standardising the surgical procedures.[85] This technique is a possible treatment option in cases with a high frenulum insertion and insufficient KT apical to the recession defect. It is mainly used in the mandible because of the aforementioned aesthetic issues.

To overcome the disadvantages associated with harvesting the CTG from the palate, various biomaterials have been proposed. Acellular dermal matrices (ACDM) from human and porcine origin and collagen matrices (CM) of porcine origin are possible substitutes for the CTG. They act as scaffolds for fibroblasts and endothelial cells to build up new connective tissue and promote epithelial cell migration from the borders of the adjacent tissue over the matrix.[86,87] Furthermore, collagen matrices can increase tissue thickness but are not as effective at increasing KT width.[88] Based on the available evidence, ACDM may provide similar clinical outcomes to those achieved by CTG in conjunction with a CAF.[89,90] In a recently published multicentre study,[91] CM with CAF reduced the patient recovery time, treatment time and decreased morbidity compared to CAF with CTG. Nevertheless, the clinical results in terms of root coverage and CRC were inferior in the CM treated group. Similar findings were demonstrated for MCAT+ CM versus MCAT +CTG.[65] Moreover, a recent meta-analysis showed that a flap alone, ACDM and CM were not effective in maintaining the stability of the gingival margin over time in comparison to CTG procedures.[92]

Other treatment modalities including the use of non-resorbable and resorbable membranes (Guided Tissue Regeneration) have been proposed for treating gingival recession defects.[84] However, considering the high incidence of complications (e.g. membrane exposure, wound dehiscence), the routine use of this technique cannot be recommended at present.[70,93,94]

The use of enamel matrix derivative (EMD) in conjunction with root coverage procedures is a further treatment option. CAF in conjunction with EMD provides more favourable clinical results in terms of CRC, gain of KT, and recession reduction when compared to CAF alone.[36,95] There is a lack of evidence to support the use of EMD in conjunction with tunnelling procedures.[64,96]

Since NCCLs are a common finding in conjunction with gingival recession,[23] various treatment options have been proposed. Based on the type of recession and the associated tooth defect, Zucchelli et al.[97] suggested a combined restorative and surgical approach for treating recession defects associated with NCCLs. The treatments included CAF with or without CTG in combination with complete[98–101] or partial[71,97,102,103] defect restoration of the NCCLs. After 1 year, the combined approaches resulted in high patient satisfaction and an optimal aesthetic outcome.

Santamaria et al.[98–101] demonstrated comparable clinical outcomes when gingival recession defects associated with NCCLs were treated by a CAF alone or CAF + CTG, irrespective of whether the NCCLs were restored or not. Recently, a partial restoration of class B+ cervical defects with the apical border of the restoration placed 1 mm beyond the CEJ estimation was proposed.[103] To reconstruct the missing CEJ, interdental papilla[104] and CEJ levels at contralateral teeth[102] have been used as reference points for the partial defect restoration. The treatment with CAF + CTG showed comparable clinical outcomes between partially restored and non-restored cervical lesions.[97,102,103] However, the use of partial restoration resulted in greater reduction of dentin hypersensitivity and a

better gingival margin contour.[103] Cairo et al.[71] suggested that adding a CTG under a CAF in the treatment of gingival recession with a previously restored CEJ should only be considered in a thin gingival phenotype (≤0.84 mm).

When it comes to peri-implant soft tissue dehiscence/deficiencies, different treatment protocols, depending on the position of the implant-supported crown, bucco-lingual position of the implant head, and height of the anatomical papillae, were proposed.[24] If a surgical intervention is applied, a CAF in conjunction with a CTG is the most studied treatment option in such cases.

Prior to a possible surgical intervention, the quality and quantity of the interproximal soft tissues, the papillae heights, and the position of the crown and/or implant could lead to removal of the implant-supported crown. Thereafter, special abutments can be used for maturation and increase of the interproximal soft tissues.

Conclusions

When developing a treatment strategy, clinicians should first focus on susceptibility factors and modifiable conditions while increasing the patient's awareness about gingival recession. In cases where a surgical approach is indicated, CAF and tunnelling procedures combined with a CTG are considered the most predictable treatment options for single and multiple recession defects. If there is a contraindication for harvesting a CTG from the palate or the patient wants to avoid a donor site surgery, adjunctive use of ACDM, CM and/or EMD can be a valuable treatment alternative. For gingival recession defects associated with NCCLs a combined restorative-surgical approach can provide favourable clinical outcomes. If a patient refuses surgical intervention or there are other contraindications for an invasive approach, gingival conditions should be maintained with preventive measures.

REFERENCES

1. Pini Prato G. Mucogingival deformities. Ann Periodontol 1999;4:98–101.
2. AAP's. Glossary of Periodontal Terms. American Academy of Periodontology. Glossary of Periodontal Terms. Available from: https://members.perio.org/libraries/glossary?ssopc=1. Accessed 6 December 2019.
3. Vignoletti F, Di Martino M, Clementini M, et al. Prevalence and risk indicators of gingival recessions in an Italian school of dentistry and dental hygiene: a cross-sectional study. Clin Oral Investig 2020;24:991–1000.
4. Thomson WM, Hashim R, Pack AR. The prevalence and intraoral distribution of periodontal attachment loss in a birth cohort of 26-year-olds. J Periodontol 2000;71:1840–5.
5. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. J Am Dent Assoc 2003;134:220–5.
6. Albandar JM, Kingman A. Gingival recession, gingival bleeding, and dental calculus in adults 30 years of age and older in the United States, 1988–1994. J Periodontol 1999;70:30–43.
7. Seong J, Bartlett D, Newcombe RG, et al. Prevalence of gingival recession and study of associated related factors in young UK adults. J Dent 2018;76:58–67.
8. Wennstrom JL. Mucogingival therapy. Ann Periodontol 1996;1:671–701.
9. Serino G, Wennstrom JL, Lindhe J, et al. The prevalence and distribution of gingival recession in subjects with a high standard of oral hygiene. J Clin Periodontol 1994;21:57–63.
10. Sangnes G, Gjermo P. Prevalence of oral soft and hard tissue lesions related to mechanical toothcleansing procedures. Community Dent Oral Epidemiol 1976;4:77–83.
11. Daprile G, Gatto MR, Checchi L. The evolution of buccal gingival recessions in a student population: a 5-year follow-up. J Periodontol 2007;78:611–4.
12. Agudio G, Nieri M, Rotundo R, et al. Periodontal conditions of sites treated with gingival-augmentation surgery compared to untreated contralateral homologous sites: a 10- to 27-year long-term study. J Periodontol 2009;80:1399–405.
13. Chambrone L, Tatakis DN. Long-term outcomes of untreated buccal gingival recessions: a systematic review and meta-analysis. J Periodontol 2016;87:796–808.
14. Caton JG, Armitage G, Berglundh T, et al. A new classification scheme for periodontal and peri-implant diseases and conditions - Introduction and key changes from the 1999 classification. J Clin Periodontol 2018;45(Suppl 20):S1–8.
15. Miller Jr. PD. A classification of marginal tissue recession. Int J Periodontics Restorative Dent 1985;5:8–13.
16. Pini-Prato G. The Miller classification of gingival recession: limits and drawbacks. J Clin Periodontol 2011;38:243–5.
17. Cortellini P, Bissada NF. Mucogingival conditions in the natural dentition: narrative review, case definitions, and diagnostic considerations. J Clin Periodontol 2018;45(Suppl 20):S190–8.
18. Zweers J, Thomas RZ, Slot DE, et al. Characteristics of periodontal biotype, its dimensions, associations and prevalence: a systematic review. J Clin Periodontol 2014;41:958–71.
19. De Rouck T, Eghbali R, Collys K, et al. The gingival biotype revisited: transparency of the periodontal probe through the gingival margin as a method to discriminate thin from thick gingiva. J Clin Periodontol 2009;36:428–33.
20. Chambrone L, Pannuti CM, Tu YK, et al. Evidence-based periodontal plastic surgery. II. An individual data meta-analysis for evaluating factors in achieving complete root coverage. J Periodontol 2012;83:477–90.
21. Hwang D, Wang HL. Flap thickness as a predictor of root coverage: a systematic review. J Periodontol 2006;77:1625–34.
22. Cairo F, Nieri M, Cincinelli S, et al. The interproximal clinical attachment level to classify gingival recessions and predict root coverage outcomes: an explorative and reliability study. J Clin Periodontol 2011;38:661–6.
23. Pini-Prato G, Franceschi D, Cairo F, et al. Classification of dental surface defects in areas of gingival recession. J Periodontol 2010;81:885–90.
24. Zucchelli G, Tavelli L, Stefanini M, et al. Classification of facial peri-implant soft tissue dehiscence/deficiencies at single implant sites in the esthetic zone. J Periodontol 2019;90:1116–24.
25. Lin GH, Chan HL, Wang HL. The significance of keratinized mucosa on implant health: a systematic review. J Periodontol 2013;84:1755–67.
26. Chen ST, Buser D. Esthetic outcomes following immediate and early implant placement in the anterior maxilla–a systematic review. Int J Oral Maxillofac Implants 2014;29 (Suppl):186–215.
27. Cosyn J, Eghbali A, Hermans A, et al. A 5-year prospective study on single immediate implants in the aesthetic zone. J Clin Periodontol 2016;43:702–9.
28. Cosyn J, Sabzevar MM, De Bruyn H. Predictors of inter-proximal and midfacial recession following single implant treatment in the anterior maxilla: a multivariate analysis. J Clin Periodontol 2012;39:895–903.
29. Evans CD, Chen ST. Esthetic outcomes of immediate implant placements. Clin Oral Implants Res 2008;19:73–80.

30. Joshipura KJ, Kent RL, DePaola PF. Gingival recession: intraoral distribution and associated factors. J Periodontol 1994;65:864–71.
31. Sarfati A, Bourgeois D, Katsahian S, et al. Risk assessment for buccal gingival recession defects in an adult population. J Periodontol 2010;81:1419–25.
32. Khocht A, Simon G, Person P, et al. Gingival recession in relation to history of hard toothbrush use. J Periodontol 1993;64:900–5.
33. Merijohn GK. Management and prevention of gingival recession. Periodontol 2016;71:228–42 2000.
34. Miglani S, Aggarwal V, Ahuja B. Dentin hypersensitivity: recent trends in management. J Conserv Dent 2010;13:218–24.
35. Kennedy JE, Bird WC, Palcanis KG, et al. A longitudinal evaluation of varying widths of attached gingiva. J Clin Periodontol 1985;12:667–75.
36. Chambrone L, Ortega MAS, Sukekava F, et al. Root coverage procedures for treating single and multiple recession-type defects: an updated Cochrane systematic review. J Periodontol 2019;90:1399–422.
37. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Res 2000;34:182–7.
38. Vijaya V, Sanjay V, Varghese RK, et al. Association of dentine hypersensitivity with different risk factors - a cross sectional study. J Int Oral Health 2013;5:88–92.
39. Ramsay DS. Patient compliance with oral hygiene regimens: a behavioural self-regulation analysis with implications for technology. Int Dent J 2000;50(S6_Part1):304–11.
40. Addy M, Hunter ML. Can tooth brushing damage your health? Effects on oral and dental tissues. Int Dent J 2003;53 (Suppl 3):177–86.
41. Nevins M. Attached gingiva–mucogingival therapy and restorative dentistry. Int J Periodontics Restorative Dent 1986;6:9–27.
42. Weitman RT, Eames WB. Plaque accumulation on composite surfaces after various finising procedures. J Am Dent Assoc 1975;91:101–6.
43. Ericsson I, Lindhe J. Recession in sites with inadequate width of the keratinized gingiva. An experimental study in the dog. J Clin Periodontol 1984;11:95–103.
44. Lang NP, Loe H. The relationship between the width of keratinized gingiva and gingival health. J Periodontol 1972;43:623–7.
45. Freedman AL, Green K, Salkin LM, et al. An 18-year longitudinal study of untreated mucogingival defects. J Periodontol 1999;70:1174–6.
46. Hangorsky U, Bissada NF. Clinical assessment of free gingival graft effectiveness on the maintenance of periodontal health. J Periodontol 1980;51:274–8.
47. Wennstrom JL. Lack of association between width of attached gingiva and development of soft tissue recession. A 5-year longitudinal study. J Clin Periodontol 1987;14:181–4.
48. Brännström M, Åström A. A study on the mechanism of pain elicited from the dentin. J Dent Res 1964;43:619–25.
49. Chen CL, Parolia A, Pau A, et al. Comparative evaluation of the effectiveness of desensitizing agents in dentine tubule occlusion using scanning electron microscopy. Aust Dent J 2015;60:65–72.
50. Pradeep AR, Sharma A. Comparison of clinical efficacy of a dentifrice containing calcium sodium phosphosilicate to a dentifrice containing potassium nitrate and to a placebo on dentinal hypersensitivity: a randomized clinical trial. J Periodontol 2010;81:1167–73.
51. Rajesh KS, Hedge S, Arun Kumar MS, et al. Evaluation of the efficacy of a 5% calcium sodium phosphosilicate (Novamin) containing dentifrice for the relief of dentinal hypersensitivity: a clinical study. Indian J Dent Res 2012;23:363–7.
52. Bergenholtz G, Jontell M, Tuttle A, et al. Inhibition of serum albumin flux across exposed dentine following conditioning with GLUMA primer, glutaraldehyde or potassium oxalates. J Dent 1993;21:220–7.
53. Leonard Jr RH, Smith LR, Garland GE, et al. Desensitizing agent efficacy during whitening in an at-risk population. J Esthet Restor Dent 2004;16:49–55 discussion 6.
54. Matis BA, Cochran MA, Eckert GJ, et al. In vivo study of two carbamide peroxide gels with different desensitizing agents. Oper Dent 2007;32:549–55.
55. Porto IC, Andrade AK, Montes MA. Diagnosis and treatment of dentinal hypersensitivity. J Oral Sci 2009;51:323–32.
56. Rezazadeh F, Dehghanian P, Jafarpour D. Laser effects on the prevention and treatment of dentinal hypersensitivity: a systematic review. J Lasers Med Sci 2019;10:1–11.
57. Sgolastra F, Petrucci A, Gatto R, et al. Effectiveness of laser in dentinal hypersensitivity treatment: a systematic review. J Endod 2011;37:297–303.
58. Lopes AO, de Paula Eduardo C, Aranha ACC. Evaluation of different treatment protocols for dentin hypersensitivity: an 18-month randomized clinical trial. Lasers Med Sci 2017;32:1023–30.
59. Norberg O. Ar en utlaknig utan vov-nadsfortus otankbar vid kirugisk behandling av S. K. Alveolarpyorrohoe? Sven Tandlak Tidskr 1926;19:171–2.
60. de Sanctis M, Zucchelli G. Coronally advanced flap: a modified surgical approach for isolated recession-type defects: three-year results. J Clin Periodontol 2007;34:262–8.
61. Zucchelli G, De Sanctis M. Treatment of multiple recession-type defects in patients with esthetic demands. J Periodontol 2000;71:1506–14.
62. Azzi R, Etienne D. Recouvrement radiculaire et reconstruction papillaire par greffon conjonctif enfoui sous un lambeau vestibulaire tunnélisé et tracte coronairement. J Parodontol d'Implantol Oral 1998;17:71–7.
63. Allen AL. Use of the supraperiosteal envelope in soft tissue grafting for root coverage. I. Rationale and technique. Int J Periodontics Restorative Dent 1994;14:216–27.
64. Aroca S, Keglevich T, Nikolidakis D, et al. Treatment of class III multiple gingival recessions: a randomized-clinical trial. J Clin Periodontol 2010;37:88–97.
65. Aroca S, Molnar B, Windisch P, et al. Treatment of multiple adjacent Miller class I and II gingival recessions with a Modified Coronally Advanced Tunnel (MCAT) technique and a collagen matrix or palatal connective tissue graft: a randomized, controlled clinical trial. J Clin Periodontol 2013;40:713–20.
66. Hofmanner P, Alessandri R, Laugisch O, et al. Predictability of surgical techniques used for coverage of multiple adjacent gingival recessions–A systematic review. Quintessence Int 2012;43:545–54.
67. Molnar B, Aroca S, Keglevich T, et al. Treatment of multiple adjacent Miller Class I and II gingival recessions with collagen matrix and the modified coronally advanced tunnel technique. Quintessence Int 2013;44:17–24.
68. Sculean A, Cosgarea R, Stahli A, et al. The modified coronally advanced tunnel combined with an enamel matrix derivative and subepithelial connective tissue graft for the treatment of isolated mandibular Miller Class I and II gingival recessions: a report of 16 cases. Quintessence Int 2014;45:829–35.
69. Sculean A, Allen EP. The laterally closed tunnel for the treatment of deep isolated mandibular recessions: surgical technique and a report of 24 cases. Int J Periodont Restorative Dent 2018;38:479–87.

70. Zucchelli G, Mounssif I. Periodontal plastic surgery. Periodontol 2015 2000;68:333–68.
71. Cairo F, Cortellini P, Nieri M, et al. Coronally advanced flap and composite restoration of the enamel with or without connective tissue graft for the treatment of single maxillary gingival recession with non-carious cervical lesion. A randomized controlled clinical trial. J Clin Periodontol 2020;47:362–71.
72. Stefanini M, Marzadori M, Aroca S, et al. Decision making in root-coverage procedures for the esthetic outcome. Periodontol 2018;77:54–64 2000.
73. Griffin TJ, Cheung WS, Zavras AI, et al. Postoperative complications following gingival augmentation procedures. J Periodontol 2006;77:2070–9.
74. Stefanini M, Zucchelli G, Marzadori M, et al. Coronally advanced flap with site-specific application of connective tissue graft for the treatment of multiple adjacent gingival recessions: a 3-year follow-up case series. Int J Periodontics Restorative Dent 2018;38:25–33.
75. Sculean A, Cosgarea R, Stahli A, et al. Treatment of multiple adjacent maxillary Miller Class I, II, and III gingival recessions with the modified coronally advanced tunnel, enamel matrix derivative, and subepithelial connective tissue graft: a report of 12 cases. Quintessence Int 2016;47:653–9.
76. Pietruska M, Skurska A, Podlewski L, et al. Clinical evaluation of Miller class I and II recessions treatment with the use of modified coronally advanced tunnel technique with either collagen matrix or subepithelial connective tissue graft: a randomized clinical study. J Clin Periodontol 2019;46:86–95.
77. Azaripour A, Kissinger M, Farina VS, et al. Root coverage with connective tissue graft associated with coronally advanced flap or tunnel technique: a randomized, doubleblind, monocentre clinical trial. J Clin Periodontol 2016;43:1142–50.
78. Salhi L, Lecloux G, Seidel L, et al. Coronally advanced flap versus the pouch technique combined with a connective tissue graft to treat Miller's class I gingival recession: a randomized controlled trial. J Clin Periodontol 2014;41:387–95.
79. Santamaria MP, Neves F, Silveira CA, et al. Connective tissue graft and tunnel or trapezoidal flap for the treatment of single maxillary gingival recessions: a randomized clinical trial. J Clin Periodontol 2017;44:540–7.
80. Dai A, Huang JP, Ding PH, et al. Long-term stability of root coverage procedures for single gingival recessions: a systematic review and meta-analysis. J Clin Periodontol 2019;46:572–85.
81. Zucchelli G, Amore C, Sforza NM, et al. Bilaminar techniques for the treatment of recession-type defects. A comparative clinical study. J Clin Periodontol 2003;30:862–70.
82. Harvey PM. Surgical reconstruction of the gingiva. II. Procedures. N Z Dent J 1970;66:42–52.
83. Maynard Jr. JG. Coronal positioning of a previously placed autogenous gingival graft. J Periodontol 1977;48:151–5.
84. Pini Prato G, Tinti C, Vincenzi G, et al. Guided tissue regeneration versus mucogingival surgery in the treatment of human buccal gingival recession. J Periodontol 1992;63:919–28.
85. Zucchelli G, De Sanctis M. Modified two-stage procedures for the treatment of gingival recession. Eur J Esthet Dent 2013;8:24–42.
86. Tavelli L, McGuire MK, Zucchelli G, et al. Extracellular matrix-based scaffolding technologies for periodontal and peri-implant soft tissue regeneration. J Periodontol 2020;91:17–25.
87. Bohac M, Danisovic L, Koller J, et al. What happens to an acellular dermal matrix after implantation in the human body? A histological and electron microscopic study. Eur J Histochem 2018;62:2873.
88. Yu SH, Tseng SC, Wang HL. Classification of soft tissue grafting materials based on biologic principles. Int J Periodontics Restorative Dent 2018;38:849–54.
89. Chambrone L, Salinas Ortega MA, Sukekava F, et al. Root coverage procedures for treating localised and multiple recession-type defects. Cochrane Database Syst Rev 2018;10:Cd007161.
90. Tatakis DN, Chambrone L, Allen EP, et al. Periodontal soft tissue root coverage procedures: a consensus report from the AAP Regeneration Workshop. J Periodontol 2015;86(2 Suppl): S52–5.
91. Tonetti MS, Cortellini P, Pellegrini G, et al. Xenogenic collagen matrix or autologous connective tissue graft as adjunct to coronally advanced flaps for coverage of multiple adjacent gingival recession: randomized trial assessing non-inferiority in root coverage and superiority in oral health-related quality of life. J Clin Periodontol 2018;45:78–88.
92. Tavelli L, Barootchi S, Cairo F, et al. The effect of time on root coverage outcomes: a network meta-analysis. J Dent Res 2019;98:1195–203.
93. Jepsen K, Heinz B, Halben JH, et al. Treatment of gingival recession with titanium reinforced barrier membranes versus connective tissue grafts. J Periodontol 1998;69:383–91.
94. Trombelli L, Scabbia A, Tatakis DN, et al. Subpedicle connective tissue graft versus guided tissue regeneration with bioabsorbable membrane in the treatment of human gingival recession defects. J Periodontol 1998;69:1271–7.
95. Cairo F, Nieri M, Pagliaro U. Efficacy of periodontal plastic surgery procedures in the treatment of localized facial gingival recessions. A systematic review. J Clin Periodontol 2014;41(Suppl 15):S44–62.
96. Stahli A, Imber JC, Raptis E, et al. Effect of enamel matrix derivative on wound healing following gingival recession coverage using the modified coronally advanced tunnel and subepithelial connective tissue graft: a randomised, controlled, clinical study. Clin Oral Investig 2020;24:1043–51.
97. Zucchelli G, Gori G, Mele M, et al. Non-carious cervical lesions associated with gingival recessions: a decision-making process. J Periodontol 2011;82:1713–24.
98. Santamaria MP, Ambrosano GM, Casati MZ, et al. Connective tissue graft plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesion: a randomized-controlled clinical trial. J Clin Periodontol 2009;36:791–8.
99. Santamaria MP, da Silva Feitosa D, Nociti Jr FH, et al. Cervical restoration and the amount of soft tissue coverage achieved by coronally advanced flap: a 2-year follow-up randomized-controlled clinical trial. J Clin Periodontol 2009;36:434–41.
100. Santamaria MP, Suaid FF, Casati MZ, et al. Coronally positioned flap plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesions: a randomized controlled clinical trial. J Periodontol 2008;79:621–8.
101. Santamaria MP, Queiroz LA, Mathias IF, et al. Resin composite plus connective tissue graft to treat single maxillary gingival recession associated with non-carious cervical lesion: randomized clinicaltrial. J Clin Periodontol 2016;43:461–8.
102. Cairo F, Pini-Prato GP. A technique to identify and reconstruct the cementoenamel junction level using combined periodontal and restorative treatment of gingival recession. A prospective clinical study. Int J Periodontics Restorative Dent 2010;30:573–81.
103. Santamaria MP, Silveira CA, Mathias IF, et al. Treatment of single maxillary gingival recession associated with non-carious cervical lesion: randomized clinical trial comparing connective tissue graft alone to graft plus partial restoration. J Clin Periodont 2018;45:968–76.
104. Zucchelli G, Testori T, De Sanctis M. Clinical and anatomical factors limiting treatment outcomes of gingival recession: a new method to predetermine the line of root coverage. J Periodontol 2006;77:714–21.