PAPER – OPEN ACCESS

The Use of Rich Platelet Fibrin (PRF) In Root Coverage

Author : Shabirah Inas Ferrina Siregar, et al
DOI : 10.32734/tm.v2i1.2696
Electronic ISSN : 1234-1234
Print ISSN : 1234-5678

Volume 2 Issue 3 – 2025 TALENTA Conference Series: Tropical Medicine (TM)

TM Conference Series 02 (2025)
TALENTA Conference Series
Available online at https://talentaconfseries.usu.ac.id

The Use of Rich Platelet Fibrin (PRF) In Root Coverage

Shabirah Inas Ferrina Siregar[1], Armia Syahpura[2], Aini Hariyani Nasution[2]

[1]*Periodontic Residency Program, Faculty of Dentistry,Universitas Sumatera Utara, Medan, Indonesia*
[2]*Department of Periodontics, Faculty of Dentistry, Universitas Sumatera Utara, Medan, Indonesia*

Armia.syahputra@usu.ac.id

Abstract

Hello there! When the gum line pulls away from the cemento-enamel junction (CEJ), exposing the tooth root, this condition is called gingival recession. Issues include root cavities, dentin hypersensitivity, trouble controlling plaque, and cosmetic concerns arise when the tooth root is exposed. The use of desensitizing materials, interventions to modify poor behaviors, and optimum plaque management are non-surgical therapy options for gingival recession. When surgical intervention is necessary, a combination of a coronally advanced flap (CAF), tunneling, and gingival graft yields satisfactory outcomes for both isolated and systemic recessions. As an adjunctive treatment to pedicle surgery, platelet-rich fibrin (PRF) represents the cutting edge of gingival surgery. Review of Existing Literature. Autogenous platelet-rich fibrin (PRF) is a biomaterial that is rich in platelets and fibrin because it is made from blood without the addition of biochemical compounds such dextrose citrate (ACD), which are anticoagulants.As growth factors and cytokines, platelets are essential for periodontal regeneration and the development of soft tissues. Final thoughts. The One alternative for recession therapy that assists with periodontal regeneration and wound healing quicker and makes patients feel better is platelet-rich fibrin (PRF).

Keywords: Gingival recession; Platelet-rich fibrin (PRF); Coronally advanced flap; Connective tissue graft

1. Introduction

When the gum line pulls away from the cemento-enamel junction (CEJ), it exposes the tooth root, a process known as gingival recession [1]. Recession of the gingiva is a mucogingival defect that may be seen on any aspect of the teeth, including the vestibular, lingual, proximal, or all surfaces. It can affect people of any age. [2]. Gingival recession is most often found on the vestibular or proximal surfaces of teeth, especially in adult patients. Gingival recession can occur in subjects with poor or good oral hygiene. Exposed tooth roots due to gingival recession cause problems such as dentin hypersensitivity, root caries, difficulty in plaque control and aesthetic problems, if gingival recession occurs in the interdental area it will causeblack triangle between adjacent teeth that will predispose to dental plaque and food debris. Some etiologies and predisposing factors that cause gingival recession are trauma when brushing teeth, tooth malposition, frenulum and muscle attachments [3].

Non-surgical treatment options for gingival recession include optimal plaque control, interventions to change bad habits, and the use of desensitizing agents [4]. You should consider surgical treatment. When choosing a surgical technique, there are several factors to consider that relate to the defect's anatomy. These include the defect's size, the biotype of the gingiva, the level of the interdental papilla and alveolar bone, the depth of the vestibular, and the position of the labial frenulum. Tunneling with the inclusion of gingival graft and coronally advanced flap (CAF) are two surgical methods that are often used to close recessions. The most recent development in gingival surgery,

Selection and peer-review under responsibility of Seminar Periodental Update 5 2024
p-ISSN: 1234-5678, e-ISSN: 1234-1234, DOI: 10.32734/tm.v2i1.2696

platelet-rich fibrin (PRF) is an adjunctive treatment to pedicle surgery [5]. PRF is a blood-based autogenous platelet fibrin matrix that has been centrifuged without the addition of bovine thrombin or extra anticoagulant. Postoperative regenerative fibroblasts (PRFs) aid in tissue repair and alleviate pain for patients in the first few days after surgery [1]. Biologically active proteins found in platelets attach to the extracellular matrix or fibrin network as it forms. A chemotactic gradient for stem cell attraction will be created by the proteins.Cell regeneration is enhanced by stem cells as they differentiate. Consequently, periodontal regeneration with autologous platelets is an exciting new area of research. [6].

2. Literature Review

2.1. Gingival Recession

2.1.1. Definition

Recession of the Gums The process of gingival recession occurs when the apical gingival edge moves toward the cementoenamel junction (CEJ), exposing the root surface. It may manifest themselves in a specific area or be more often linked to many tooth surfaces. [3]. The etiology is multifactorial including inadequate brushing technique, tooth malposition, periodontal disease, frenulum aberrant, occlusion trauma and iatrogenic factors (orthodontic treatment, or prosthetic treatment) [7]. Included in the mucogingival complex are the alveolar mucosa, free and connected gingiva, and the mucogingival junction. Sufficient mucogingival complex, in which the mucogingival tissue is able to maintain its biomorphological integrity and adherence to the teeth and subgingival tissues. When pockets develop, it's known as a closed mucogingival condition. When gaps form in the gingiva and recession occurs, it's known as an open mucogingival disorder. [8].

2.1.2. Etiology of Gingival Recession

- Gingival recession is a consequence of inflammation in the neighboring connective tissue junctional epithelium, which is caused by plaque and calculus. [9].
- Bacterial interactions between plaque and the human immune system cause periodontal disease, which in turn causes deterioration of the matrix and resorption of bone, which in turn causes periodontal pockets, recession of the gingiva, or both. [10].
- Gingival recession may be caused by mechanical pressure or by cleaning teeth incorrectly. Although there doesn't seem to be any inflammation in the gingiva, the tooth root is showing since the gingival edge is more apically exposed. [10].
- Iatrogenic factors, tooth movement with orthodontic appliances can change the margin and gingival papilla. The formation ofdehiscenceduring orthodontic treatment is often accompanied by gingival recession. Prosthodontic and restorative procedures such as subgingival cervical preparation, impressions with gingival retraction, restorations and artificial crowns placed subgingivally, restorations that overhang. Poor denture design and clasp placement can result in plaque accumulation on the abutment teeth and lead to gingival recession if left untreated [11].
- Alveolar bone dehiscence, frenulum attachment, tooth malposition, and tooth shape are some of the anatomical elements that might contribute to the development of gingival recession. Biotype and morphology Thin and scalloped periodontium and thick and flat periodontium are the two varieties. Thin biotypes are more prone to gingiva recession, which affects the space between teeth and the face. [12].

2.1.3. Classification of Gingival Recession

Miller in 1985 proposes the most widely used classification until these days. Miller's classification is as follows:

Class I—Gingival margin recession that does not extend into the mucogingival junction (MGJ), with no bone loss in the interdental area.
Class II—Gingival margin recession extending into the mucogingival junction (MGJ), there was no bone loss in the interdental area.
Class III—Gingival margin recession extending into themucogingival junction(MGJ); loss of bone or soft tissue in the interdental area
Class IV—Gingival margin recession extending into or beyond the mucogingival junction (MGJ). Severe bone or soft tissue loss in the interdental area [10].

Nordland and Tarnow 1998 proposed a classification system for interproximal papilla height based on three landmark anatomy: Interdental contact points, apical level of the facial CEJ and coronal level of the proximal CEJ.
Normal: interdental papillae fill the space from the apical level to the interdental contact area.
Class I: the tip of the interdental papilla is located between the interdental contact points and extends coronal to the interdental CEJ.
Class II: the tip of the interdental papilla is more apical to the interproximal papilla but still coronal to the facial CEJ.
Class III: the tip of the interdental papilla is located parallel to or more apically than the facial CEJ [10].

Cairo 2011 proposed a newer classification using the interdental clinical attachment level (ICAL) as the criterion. The following are the recession types according to Cairo: Type 1 recession (RT1) is associated with no loss of interdental attachment level.
Type 2 Recession (RT2) ICAL loss is equal to or less than buccal attachment loss.
Type 3 Recession (RT3) ICAL loss is higher than the amount of buccal attachment loss [10].

2.1.4. Treatments

Non-surgical treatment

There should be no root caries observation if the recession defect is small, not in a region where it would be seen as unsightly, and there is no dentin hypersensitivity. Finding and controlling the root cause of the recession is crucial in order to stop it from happening again. Additionally, it is critical to keep up with dental hygiene in order to avoid plaque-induced gingival irritation, which may worsen recession, particularly in those with thin gingival biotypes. When root cavities, dentin hypersensitivity, recession, or an uneven gingival zenith cause a patient to have a high smile line, intervention and care may be necessary. Sensitivity therapy with over-the-counter desensitizing medications may be adequate if the patient mainly expresses discomfort from sensitivity and not from aesthetic issues [4]. When patients have recession as a result of overcrowding, it is important to delay any planned surgical intervention until after orthodontic therapy has been completed. Research indicates that when teeth are moved to the lingual side of the mouth via orthodontics, the buccal portion of the jawbone grows, thickening the gingival tissue. Then, when the teeth are shifted back into place, the recessed gingival border is corrected. [11].

Surgical Treatment

Carlo et al. provide a schematic illustration of the several recession closure techniques for grades I, II, and II. It is crucial to first measure the socket's depth. The GTR surgery can be carried out with CAF if there is enough keratinized tissue and the socket depth is 5 mm [11]. If there is insufficient keratinized tissue, treatment is required without the use of a donor organ.

The connective tissue graft (CTG) technique is one of the most well-liked and effective ways to cosmetically address gum recession. Although this procedure necessitates a donor site at the recession site, numerous studies have demonstrated that it produces exceptional cosmetic results, with success rates ranging from 69% to 97%. When attempting to cure recessions, problems often develop, such as the scarcity of grafts, the necessity of two treatments, and the discomfort that patients endure following surgery. [14].

Histological analysis after traditional mucogingival surgery should reveal the formation of a lengthy junctional epithelium following healing following recession closure. Root conditioners, enamel matrix proteins, and platelet-rich fibrin (PRF) are a few of the supplementary substances utilized to speed healing and enhance clinical results since traditional surgical procedures alone are not enough to promote regeneration [14]. In cosmetic surgery, implant placement, and periodontal treatments, this homogenous fibrin network speeds healing by improving the regeneration of both hard and soft tissues. [15].

Platelet rich fibrin (PRF)

An autogenous biomaterial, a second-generation platelet concentrate, created in France by Choukronet al. Generally recognized as a substance that speeds up the recovery of both soft and hard tissue injuries. [6]. Platelet rich fibrin (PRF) is a biodegradable scaffold that includes cytokines, platelets, and steam cells; it facilitates micro vascularization, which in turn allows epithelial cells to migrate. Research has shown that PRF has the ability to promote healing, bone development, and maturation whether taken alone or in conjunction with bone transplants. It also has the ability to regenerate soft tissues and bones without causing inflammatory responses [16]. How PRF contributes to the healing of soft tissues PRF promotes fibroblast neo angeogenesis, assists in gingival differentiation, includes a range of growth factors, and may contribute to tissue regeneration via osteoblasts and prekeratinocytes. Hard and soft tissue regeneration are both aided by platelet alpha-granules, which store many growth factors. [17,18].

Factors that promote growth, such as platelet-derived growth factors (PDGFs), transforming growth factor-beta (TGF-beta), elastic growth factor (EGF), and insulin-like growth factor-1 (IGF-1). It is a growth factor that promotes the migration and proliferation of connective tissues and boosts chemotic and mitotic activity. [19,20].

3. Conclusion

The Use of platelet-rich fibrin (PRF) as one of the recession treatment options that provides a more comfortable feeling for patients, helps periodontal regeneration and faster wound healing.

References

[1] Rodas M, De Paula BL et al. Platelet-Rich Fibrin in Coverage of Gingival Recession: A Systematic Review and Meta-Analysis. Dental Investigation Society 2020, 14:315–326.

[2] Cortellini P, Bissada NF. Mucogingival conditions in the natural dentition: Narrative review, case definitions, and diagnostic considerations. Journal of Periodontology 2018, 89 (Suppl 1):S204–S21.

[3] Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. J Am Dent Assoc 2003, 134: 220–225.

[4] Patel M, Nixon PJ, Chan MFWY Gingival recession: part 1 Aetiology and non- surgical management. Br. Dent. J 2011, 211.

[5] Alghamdi H, Babay N, Sukumaran A. Surgical management of gingival recession:

[6] A clinical update. The Saudi Dental Journal 2009, 21: 83-94

[7] Mohan SP, Jaishangar N et al. Platelet Rick-Plasma and Platelet-Rich Fibrin in Periodontal Regeneration: A Review. J Pharm Bioallied Sci 2019, 11 (Suppl 2):

[8] S126-130

[9] Kundapur PP, Bhat KM, Bhat GS. Association of Trauma from Occlusion with Localized Gingival Recession in Mandibular Anterior Teeth. DRJ 2009, 6(2): 71-4.

[10] Chrysanthakopoulos NA. Aetiology and Severity of Gingival Recession in an Adult Population Sample in Greece. Dental Research Journal 2011, 8 (2): 64-70

[11] Susin C, Haas AN, et al. Gingival recession: epidemiology and risk indicators in a representative urban Brazilian population. J Periodontol 2004;75(10):1377–86

[12] Ravipudi S, Appukuttan D et al. Gingival Recession: Short Literature Review on Etiology, Classifications and Various Treatment Options. Journal of Pharmaceutical Sciences and Research 2017, 9(2): 215-220

[13] Baker P, Spedding C. The aetiology of gingival recession. Dent Update 2002, 29: 59–62

[14] Siebert J, Lindhe J: Tetbook of Clinical Periodontology and Implant Dentistry. 2nd ed. Copenhangen: Munksgaard, 1989. 223-225

[15] Harpenau AL, Kao RT, Lundergan WP, Sanz M. Hall's Critical Decisions in Periodontology and Dental Implantology.5th ed. USA: People's Medical Publishing House

[16] Uraz A, Sezgin Y, Yalim M, Taner IL, Cetine D. Comparative evaluation of platelet-rich fibrin membrane and connective tissue graft in the treatment of multiple adjacent recession defects: A clinical study. Journal of Dental Sciences 2013, 10. 36-45

[17] Zhao JH, Tsai CH, Chang YC. Management of radicular cysts using platelet-rich fibrin and bioactive glass: a report of two cases. J Formosan Medical Association 2012, 113 (7). 470-476

[18] Borie E, Olivi DG, Orsi LA et al. Platelet-rich fibrin application in dentistry: a literature review. Int J Clin Exp Med 2015, 8(5). 7922-7929

[19] Dohan DM, Choukron J et al. Platelet-rich fibrin (PRF): A second-generation platelet concentrate. Part II: Platelet-related biological features. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006, 101:E45-50

[20] Saluja H, Dehane V, Mahindra U. Platelet-Rich fibrin: A second generation platelet concentrate and a new friend of oral and maxillofacial surgeons. Annals of Maxillofacial Surgery 2011, 1(1).53-57

[21] Anitua E, Sanchez M, Orive G, Andia I. The potential impact of the preparation rich in growth factors (PRGF) in different medical fields. Biomaterials 2007, 28(31).

[22] Marx RE, Carlson ER, Eichstaedt RM, Schimmele SR, Strauss JE, Georgeff KR. Plateletrich plasma: Growth factor enhancement for bone grafts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998, 85. 638-646.