International Journal Dental and Medical Sciences Research
Volume 3, Issue 6, Nov-Dec 2021 pp 121-127 www.ijdmsrjournal.com ISSN: 2582-6018

The Management of Localized Intrabony Defect with Class Iii Gingival Recession Using Platelet Rich Fibrin and Bone Graft – A Case Report

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Submitted: 01-11-2021 Revised: 12-11-2021 Accepted: 15-11-2021

ABSTRACT:Periodontitis is an infectious disease of the periodontium that is responsible for the destruction of bone leading to tooth loss. Periodontal regeneration is the process of reproduction or reconstitution of the lost or diseased part in order to restore the architecture and function of the periodontium. Platelet rich fibrin is a rich source of autologous growth factors and cytokines embedded in a matrix which has shown its importance in obtaining periodontal regeneration in recent years. The growth factors present in PRF promotes wound healing and tissue regeneration when combined with alloplastic bone substitutes.

KEYWORDS: Platelet-rich fibrin (PRF), Intrabony defects, Infrabony defects, Bone graft, Class III recession management, Osseous defects.

I. INTRODUCTION:

Periodontitis is a multifactorial disease affecting the periodontium through an inflammatory process. It leads to the loss of support from alveolar bone which usually represents the anatomical sequela to the progression of periodontitis apically[1]. Chronic periodontitis is a common disease that can occur in most age groups, but is most prevalent among adults and seniors worldwide, with approximately 35% of adults (30–90 years) in the US being affected by at least one site with CAL ≥3 mm and probing depth (PD) ≥4 mm[2]. Chronic Periodontitis leads to bone loss that occurs either in single or different combination forms. These alterations in the morphology of the alveolar bone have been termed as periodontal osseous defects. These defects play a crucial role in initiation or progression of periodontal disease which can lead to disease induced osseous defects[1].

The prevalence of vertical defects has been reported to be higher in male patients (14.95%) when compared to female patients (8.2%) and also it was rare in patients with dental awareness (de Toledo et al 2012). Vertical defects are commonly associated with posterior teeth (Baljoon et al.), with the higher prevalence in mandibular posterior teeth (33.8%) (Vrotsos et al., Kasaj et al.) and are commonly associated with molars with higher prevalence of crater formation (26.5%), followed by circumferential defects (23.4%) and 3 wall defects (20.08%) (Wu et al.)[1].

II. PERIODONTAL REGENERATION:

The ultimate aim of periodontal regeneration is restoration of the periodontium to its original form and function. Open flap debridement leads to repair of lost periodontal attachment which occurs primarily through formation of a long junctional epithelium whereas regeneration has been defined as “the reproduction or reconstitution of a lost or injured part to restore the architecture and function of the periodontium.” Periodontal regeneration involves an orchestrated sequence of biologic events such as cell migration, adherence, growth and differentiation, to increase the success and predictability of periodontal regenerative procedures[3]. It involves recruitment of locally derived progenitor cells which subsequently differentiates into periodontal ligament forming cells, cementoblasts or bone forming osteoblasts. Therefore, the key to periodontal regeneration is to stimulate the progenitor cells to reoccupy the defects[4]. The most positive outcome of periodontal regenerative procedures in infrabony defects and furcations has been achieved with a combination of platelet rich fibrin and bone grafts.

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The regenerative potential of platelets was introduced in the 70's, when it was observed that they contain growth factors that are responsible for increase collagen production, cell mitosis, blood vessels growth, recruitment of other cells that migrate to the site of injury, and cell differentiation induction, among others. It is well known that platelets play animportant role in both hemostasis and wound healing processes. Whitman et al, in 1997, introduced the use of platelet-rich plasma in oral surgical procedures, reporting great advantages because it enhances osteoprogenitor cells in the host bone and bone graft[5].

Choukroun et al. in 2004, was the first to describe platelet rich fibrin in France. It has been referred to as a second- generation platelet concentrate because the natural concentrate is produced without any anticoagulants or gelifying agents. PRF is composed of an intimate assembly of cytokines, glycanic chains, and structural glycoproteins enmeshed within a slowly polymerized fibrin network[3]. PRF releases numerous growth factors such as Transforming growth factor-ß (TGF- ß), Platelet-derived growth factor (PDGF), Vascular endothelial growth factor (VEGF), Insulin like growth factor (IGF), that modulate and upregulate growth factor function. It stimulates the cell proliferation of cell proliferation of osteoblasts, gingival fibroblasts and periodontal ligament cells, but suppress the growth of epithelial cells[6]. PRF consists of an autologous leukocyte-platelet-rich fibrin matrix, composed of a tetra molecular structure, with cytokines, platelets, and stem cells within it, which acts as a biodegradable scaffold that favors the development of micro vascularization and is able to guide epithelial cell migration to its surface. It seems to have a sustained release of growth factors in a period between 1 and 4 weeks, stimulating the environment for wound healing in a significant amount of time[5].

In this report, we present the clinical and radiographic changes of a patient using PRF along with alloplast as grafting material in the treatment of periodontal intrabony defect with endodontic involvement.

III. CASE REPORT:

A. PATIENT INFORMATION:

A 50-year-old female reported to the outpatient department of periodontology at Rajah Muthiah Dental College and Hospital, Annamalai University, Chidambaram, with a chief complaint of receding gums in her lower front tooth region for past 3 months associated with pain which is gradual in onset, non-radiating, aggravates on chewing and relieves at rest for past 10 days. Patient did not give any relevant medical history and there was no systemic condition that could interfere with physiological wound healing. Patient has undergone extraction due to dental caries before 2 years. No abnormalities were detected on extra-oral examination.

B. CLINICAL FINDINGS:

On intra-oral examination, there was generalized bleeding on probing present but no swelling and no pus exudation was noticed. The probing pocket depth (PPD) on the distobuccal aspect of the tooth # 31 was 10 mm and 8 mm on the mesiobuccal aspect, clinical attachment level (CAL) was 10 mm, whereas grade II mobility was detected in relation to 31 and fremitus was found to be positive which denotes the possibility of trauma from occlusion [Figure 1]. The patient also presented with pain in relation to #31 tooth and had pain on percussion. There was no response when subjected to heat test using a heated gutta-percha point. The diagnosis was made to be localized chronic periodontitis with secondary endodontic involvement in relation to left mandibular central incisor (#31).

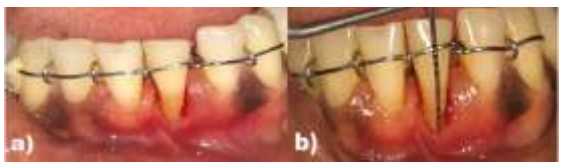

Figure 1: a) Pre-operative view after splinting, b) Pre-operative probing.

An intra-oral periapical radiograph was taken using the standardized techniques, which revealed presence of intrabony defect (IBD) with tooth #31 [Figure 2]. Before planning for the periodontal surgical procedure, patient's platelet count (3.2 lac/mm3), Hemoglobin (12.5 gm/dl), Bleeding time (2.10 min) and Clotting time (3.40 min) were assessed and found to be within normal limits.

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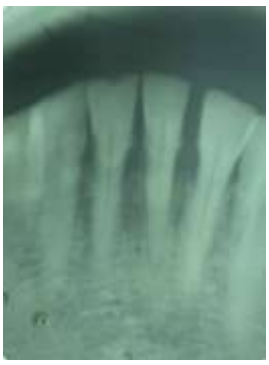

Figure 2: Pre-operative IOPA

C. TREATMENT PLAN:

Keeping all the findings in the mind, a thorough treatment plan was decided, including a series of therapeutic procedures, following non-surgical periodontal therapy by means of conventional scaling and root planning using ultrasonic instrument and curettes. Periodontal splinting was done from #33 to #43 and endodontic treatment was carried out in relation to #31 [Figure 3].

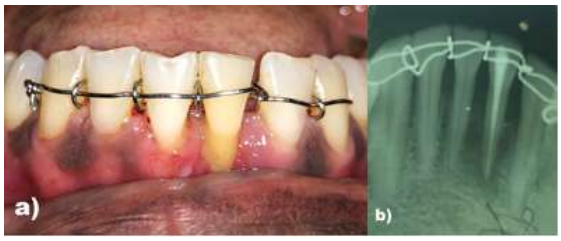

Figure 3: a) Extracoronal splinting, b) Endodontic treatment in #31.

At 4 weeks following phase 1 therapy, a periodontal re-evaluation was performed to confirm the suitability of #31 tooth for this periodontal surgical procedure. Clinical measurements were made using William's periodontal probe with graduation to a precision of 1 mm.

D. PRF PREPARATION:

Blood sample was collected from antecubital vein in a 10 ml of sterile tube without anticoagulant on the day of periodontal surgery and PRF was prepared according to the protocol developed by Choukroun et al[7]. The blood was centrifuged immediately using a tabletop centrifuging machine for 12 min at 2,700 rpm. A fibrin clot was formed in the middle of the tube, whereas the upper part contained acellular plasma and the bottom part contained red corpuscles. The fibrin clot was easily separated from the lower part of the centrifuged blood using a sterile tweezers and scissors just after removal of PPP and then transferred onto a sterile dappen dish. One half of PRF clot was gently pressed between two sterile dry gauges to obtain a membrane, while the other half was later minced and added to the graft material [Figure 4].

Figure 4: PRF prepared.

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E. SURGICAL PROCEDURE:

Intraoral antisepsis was performed using 0.2% chlorhexidine digluconate rinse and iodine solution was used to carry out extra oral antisepsis. Following administration of local anesthesia, sulcular incisions were made, and a full thickness mucoperiosteal flap was reflected on labial and lingual sides in relation to #31, 32, 33, 41, 42, 43. The inner surface of the flaps were curetted to remove the epithelium and granulation tissue. Meticulous debridement and root planning was carried out with the help of area specific curettes. The left mandibular central incisor revealed a two walled intrabony defect[1] [Figure 5]. The root surfaces adjacent to the defect was conditioned using tetracycline for 2 minutes. The defect was filled with the mixture of PRF and bone graft and a PRF membrane of required size was placed covering the graft mixture [Figure 6]. The mucoperiosteal flaps were repositioned to their pre-surgical levels and sutured with simple interrupted sutures using resorbable 3-0 vicryl surgical suture [Figure 7]. The surgical area was protected and covered with periodontal dressing [Figure 9].

Figure 5: Mucoperiosteal flap elevated and debridement done.

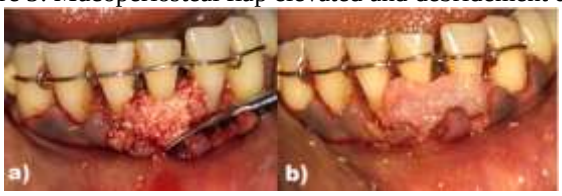

Figure 6: a) PRF + Bone graft placed and b) Covered with PRF membrane.

Figure 7: Suture placed.

Figure 8: Immediate post-operative IOPA.

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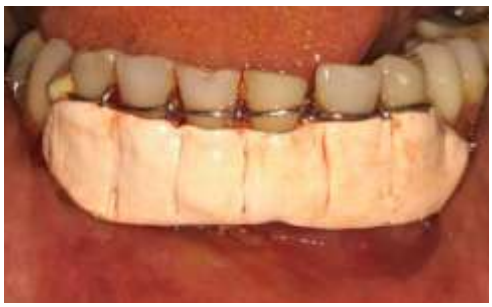

Figure 9: Periodontal dressing placed.

F. POST-OPERATIVE CARE:

After the surgery, post– surgical instructions were given and she was prescribed systemic antibiotics (moxikind CV 625mg, bid, 5 days), non – steroidal anti-inflammatory drugs (zerodol P, bid, 5 days) and 0.2% chlorhexidine mouth rinse (twice a day for 2 weeks). Sutures were removed after 14 days. Clinical healing was normal, with neither infectious episodes nor untoward clinical symptoms. The patient was seen at 1st week, 2nd week, 1st month, 3rd month and 6th month after surgery [Figure 10]. Re-examination at 6 months after the periodontal surgery revealed reduction in PPD with no signs of bleeding on probing and significant radiographic bone fill in the periodontal intrabony defect.

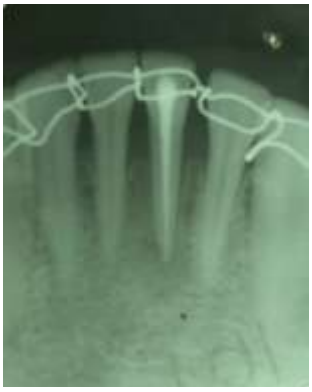

Figure 10: IOPA after 6 months.

G. INVESTIGATIONS:

Complete blood hemogram and Radiograph (intra oral periapical) pre- operative and post – operative.

H. TREATMENT:

PRF with Bone Graft.

IV. DISCUSSION:

Periodontitis is a disease characterized by irreversible loss of connective tissue attachment and supporting alveolar bone[8]. The interpretation of Periodontal osseous defects as oral buccal/facial and interproximal defects is essential as the treatment protocol has to be extended to both of them[1]. Regenerative periodontal therapy aims at the regeneration of the lost periodontal structures in order to restore the health, function and esthetics of periodontium[6]. The complete regeneration of the periodontium following periodontal therapy has been difficult due to differences in the healing abilities among periodontal tissues. Although there are a broad range of treatment options available, only some may be regarded as regenerative procedures.

In this case report, a combination of minced PRF with bone graft was placed in the defect covered by PRF membrane. The intended role of PRF was to deliver the growth factors to the surgical site during the early phase of healing. This evaluated the clinical efficacy of PRF + bone graft in the treatment of infrabony defects in patients with chronic periodontitis and showed a significant improvement in clinical and radiographic parameters. There was a significant reduction in PPD and gain in CAL with no signs of bleeding on probing. Also, there was a significant radiographic bone fill on both the mesial and distal aspects of #31 in the periodontal intrabony defect.

PRF which is in the form of gel, when used in conjunction with bone graft, offers several advantages including better wound healing, bone growth and maturation, graft stabilization, wound

sealing and hemostasis and better handling of graft material[3]. It consists of an assembly of cytokines, glycan chains and structural glycoproteins enmeshed within a slow releasing fibrin network[9]. Recently studies have demonstrated that, there was a significantly slow-sustained release of key growth factors for at least 7 days and up to 28 days was observed with PRF membrane[6]. In a study where PRF when used in combination with bone mineral had the ability in increasing the regenerative effects in infrabony defects. Also, amorphous PRF when used in combination with bio-oss for augmentation in maxillary atrophic cases showed reduced healing time and favorable bone regeneration[4].

Anuj Sharma et al. (2011)[10] and Thorat et al. (2011)[11] conducted a study to explore the clinical and radiographic effectiveness of autologous PRF in the treatment of intrabony defects in patients with chronic periodontitis and found that, there was greater pocket depth reduction, clinical attachment level (CAL) gain, and bone fill at the sites treated with PRF + conventional open-flap debridement when compared to conventional open-flap debridement alone.

Simonpieri et al. (2009)[12] described the four main advantages of the use of PRF during bone grafting. They include,

1. Fibrin clot plays an important mechanical role in maintaining and protecting the graft and PRF fragments serve as biological connectors between bone particles.
2. Fibrin network facilitates cellular migration, vascularization, and survival of the graft.
3. The growth factors (PDGF, TGF-β, IGF-1) are gradually released as the fibrin matrix is resorbed, thus creating a perpetual process of healing.
4. The presence of leukocytes and cytokines in the fibrin network can play an important role in the self-regulation of inflammatory and infectious phenomena within the grafted material.

Several studies have examined the suitability of autologous PRF for treatment of human intra-bony defects and revealed that PRF improves clinical and radiographical parameters[13]. PRF could improve the periodontal osseous defect healing, as they can up regulate phosphorylated extracellular signal regulated protein kinase expression and suppress the osteoclastogenesis by promoting secretion of osteoprotegerin (OPG) in osteoblasts cultures. PRF can also stimulate the osteogenic differentiation of human dental pulp cells by upregulating OPG and alkaline phosphatase (ALP) expression. It increases cell attachment, proliferation and collagen related protein expression of human osteoblasts and also enhances the phosphorylated – extracellular signal regulated kinases, OPG and ALP expression which benefits periodontal regeneration by influencing human periodontal ligament fibroblasts[4].

Above studies show that when PRF is used alone or in combination with bone graft, there was significant amount of pocket depth reduction, CAL gain, and radiographic bone fill with better healing of the periodontal tissues. PRF has become the simplified, easy, fast and cost-effective treatment modality for regenerative periodontal therapy due to its sustained release of growth factors. Hence, it is considered to be the first in fibrin technology.

V. CONCLUSION:

The present case report suggests that, PRF when combined with bone graft is efficacious both clinically and radiographically in the treatment of intrabony defects in patients with chronic periodontitis. Also, there were no adverse effects seen with this mode of treatment. The autologous PRF preparation is both clinically effective and economical when compared to any other regenerative materials including PRP. However, the long-term results associated with this modality of therapy will be required to know its clinical and radiographic effect over bone regeneration.

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