

Original Research

Evaluation of probing depth with and without platelet rich fibrin membrane in horizontal bone defect -An original Research

¹Shubhangi Sharma, ²Sunny Sharma

¹Senior Lecturer, Department of Periodontology & Implantology, Maharaja Ganga Singh Dental College, Ganganagar, Rajasthan, India;

²Registrar, Department of Prosthodontics and Crown & Bridge, Indira Gandhi Govt. Dental College & Hospital, Jammu, J&K UT, India

ABSTRACT:

Background: To investigate the probing depth in patients with and without platelet rich fibrin membrane in treating periodontal horizontal bone defects. **Method:** A total of 20 patients were selected for relative study consisting of open flap debridement with and without prf membrane for evaluation of probing depth in horizontal bone defect with study duration of 3months. **Result:** Though the mean PPD score decreased in both the group I and group II, the values were very similar in both the groups after 1st, 2nd and 3rd month.

Keywords: PRF:Platelet rich fibrin, PPD; Probing pocket depth

Received: 19 November, 2022

Accepted: 21 December, 2022

Corresponding author: Sunny Sharma, Registrar, Department of Prosthodontics and Crown & Bridge, Indira Gandhi Govt. Dental College & Hospital, Jammu, J&K UT, India

This article may be cited as: Sharma S, Sharma S. Evaluation of probing depth with and without platelet rich fibrin membrane in horizontal bone defect -An original Research. J Adv Med Dent Scie Res 2023;11(1):63-65.

INTRODUCTION

Periodontitis is a chronic infectious disease which results in biofilm formation and subsequent destruction of periodontal tissue because of an accelerated host response to pathogenic bacteria. Periodontitis is mostly promoted by microorganisms which are the primary cause for developing periodontitis.^{1,2} Early studies found smoking^{3,4}, diabetes mellitus^{5,6,7}, or other risk indicators, e.g. genetic predisposition^{8,9,10}, age^{11,12}, sex^{13,14}, social and psychological factors^{14,15,16} to be correlated with periodontitis.

The initial treatment of periodontitis involves controlling its causes, reducing the bacterial load on the supragingival and subgingival surfaces through non-surgical periodontal treatment, drug treatment if needed, and reinforcing oral hygiene education.¹⁷ Chronic periodontitis has been defined as 'an infectious disease resulting in inflammation within supporting tissues of the teeth, progressive attachment loss and bone loss.'¹⁸ Chronic periodontitis demands periodontal therapies that include either non-surgical periodontal therapy or open flap debridement surgical therapy or both to arrest the progression of disease.¹⁹ The objective of this study is to evaluate the PPD

between open flap debridement with prf membrane and open flap debridement without prf membrane in chronic periodontitis.

MATERIAL AND METHODS

STUDY DESIGN

A total of 20 patients were selected for relative study consisting of open flap debridement with prf and without prf membrane in horizontal bone defect patients for evaluating PPD.

CRITERIA FOR PATIENT SELECTION

INCLUSION CRITERIA

- Chronic periodontitis patients with probing depth ranging from 5-8mm
- Subject aged between 20-55 year with horizontal bone defect.
- Systemically healthy patients.

EXCLUSION CRITERIA

- Patients with known systemic illness.
- Pregnant or lactating mothers.
- Patients with history of allergies to drugs
- Subject with habit of smoking and tobacco chewing

- History of periodontal surgery in same area within 6 months.

STUDY PROTOCOL

A proforma was prepared for the study so as to have systemic recording of all observations and information. Clinical parameter i.e probing depth was measured under standard conditions of light using mouth mirror and UNC -15 probe. After completion of initial periodontal treatment including oral hygiene.

STUDY DURATION

3 months

RESULT

Table 1 Comparison of the means of PPD between the group I and group II at baseline, 1st month, 2nd month and 3rd month.

PPD		Baseline	1 month	2 months	3 months
Group I	Mean	5.51	4.30	3.43	2.66
	Std. Deviation	.708	.666	.676	.709
Group II	Mean	5.61	4.51	3.74	2.40
	Std. Deviation	.526	.507	.590	.426
p-value		.643	.578	.437	.729

Table 1 represents the comparison of the means of PPD between the group I and group II at baseline, 1st month, 2nd month and 3rd month. Though the mean PPD score decreased in both the groups, the values were very similar in both the groups after 1st, 2nd and 3rd month.

DISCUSSION

Periodontitis is an inflammatory disease of the periodontium comprising the gingiva, alveolar bone, periodontal ligament and cementum.²⁰ The pathogenesis of periodontitis involves the interaction between the host defense mechanisms and the dental biofilm. Triggered by microorganisms, periodontitis is caused by the chronic immune response leading to inflammatory cytokine production that results in the destruction of the periodontium and subsequent manifestations of periodontitis.^{21,22}

In periodontal disease, not only does the bone that supports the teeth, known as alveolar bone, reduce in height in relation to the teeth but the morphology of remaining bone is altered.²³ Recently, the association of platelet rich fibrin to regenerative procedure have been proposed. The biological plausibility of PRF is related to the increasing concentration of growth factors and other molecules related to angiogenesis, stem cell migration and the osteogenic differentiation on the regenerated site, improving the biological capabilities, tissue formation and healing.^{24,25}

The aim of this study was to evaluate the probing depth with and without PRF membrane in horizontal bone defect. In this study, the mean of PPD decreased in both the group I and group II at baseline, 1 month, 2 months and 3 months of time period but the values were very similar in both the groups after 1st, 2nd, and 3rd month. The second generation platelet derivation, PRF, first appeared in France in 2001 (*choukroun et*

SURGICAL PROCEDURE

After the administration of local anesthesia, buccal and lingual crevicular incisions were made, and mucoperiosteal flaps were reflected. Defect debridement and root planning were carried out using ultrasonic instruments and area specific curettes.

Group I: 10 patients in which open flap debridement performed

Group II: 10 patients in which open flap debridement performed with prf membrane.

Mucoperiosteal flap repositioned and sutured by interrupted sutures and covered with periodontal dressing.

al,2006). Platelet rich fibrin provides a scaffold for cell migration and growth factors for promoting wound healing and tissue regeneration. Platelet concentrates are made by centrifuging the patient's blood. When used either alone or in combination with bone grafts they have significant impact on periodontal regeneration (*Ehernfest et al.2014*). 'Pocket elimination', defined as pocket depth reduction to gingival sulcus levels is considered as one of the main goals of periodontal therapy. This procedure is indeed essential because of the need to improve accessibility to root surfaces for the therapist during treatment and for the patient during periodontal maintenance and self – performed oral hygiene.²⁶ Conservative surgery encompasses a range of surgical procedures aimed at gaining access to root surface in order to remove plaque/calculus.

CONCLUSION

Horizontal bone loss is the most common problem but receiving scant attention. This study demonstrated that mean PPD decreased in both groups but values were very similar after 1st, 2nd and 3rd month in both the groups.

REFERENCES

- Schaudinn C, Groux A, Keller D, Sedghizadeh PP, Costerton JW. Periodontitis; an archetypical biofilm disease. *J Am Dent Assoc.* 2009;140:978-86.
- Socransky SS, Haffajee AD. The bacterial etiology destructive periodontal disease; current concept. *J periodontol* 1992;63:322-31.
- Calsina G, Ramón J-M, Echeverría J-J. Effects of smoking on periodontal tissues. *J Clin Periodontol.* 2002;29:771-6.
- Johnson GK, Slach NA. Impact of tobacco use on periodontal status. *J Dent Educ.* 2001;65:313-21.

5. Salvi GE, Carollo-Bittel B, Lang NP. Effects of diabetes mellitus on periodontal and peri-implant conditions: update on associations and risks. *J Clin Periodontol*. 2008;35:398–409.
6. Emrich LJ, Shlossman M, Genco RJ. Periodontal-Disease in Non-InsulinDependent Diabetes-Mellitus. *J Periodontol*. 1991;62:123–31.
7. Hodge PJ, Robertson D, Paterson K, Smith GL, Creanor S, Sherriff A. Periodontitis in non-smoking type 1 diabetic adults: a cross-sectional study. *J Clin Periodontol*. 2012;39:20–9.
8. Schaefer AS, Richter GM, Groessner-Schreiber B, Noack B, Nothnagel M, El Mokhtari NE, et al. Identification of a shared genetic susceptibility locus for coronary heart disease and periodontitis. *PLoS Genet*. 2009;5:e1000378.
9. Schaefer AS, Richter GM, Nothnagel M, Laine ML, Ruhling A, Schafer C, et al. A 3' UTR transition within DEFB1 is associated with chronic and aggressive periodontitis. *Genes Immun*. 2010;11:45–54.
10. Schaefer AS, Richter GM, Nothnagel M, Manke T, Dommisch H, Jacobs G, et al. A genome-wide association study identifies GLT6D1 as a susceptibility locus for periodontitis. *Hum Mol Genet*. 2010;19:553–62.
11. Cobb CM, Williams KB, Gerkovitch MM. Is the prevalence of periodontitis in the USA in decline? *Periodontol*. 2009;50:13–24.
12. Locker D, Slade GD, Murray H. Epidemiology of periodontal disease among older adults: a review. *Periodontol*. 1998;16:16–33.
13. Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol*. 1996;67:1041–9.
14. Timmerman MF, der Weijden GAV. Risk factors for periodontitis. *Int J Dent Hyg*. 2006;4:2–7.
15. Heitz-Mayfield LJ. Disease progression: identification of high-risk groups and individuals for periodontitis. *J Clin Periodontol*. 2005;32:196–209.
16. Pistorius A, Krahwinkel T, Willershausen B, Boekstegen C. Relationship between stress factors and periodontal disease. *Eur J Med Res*. 2002;7:393–8.
17. Van der Weijden GA, Timmerman MF. A systematic review on the clinical efficacy of subgingival debridement in the treatment of chronic periodontitis. *J Clin Periodontol*. 2002;29 Suppl 3:55-71.
18. Flemming TF. Periodontitis. *Ann Periodontol*. 1999;4(1);32-7.
19. Graziani F, Karapetsa D, Alonso B, Herrera D. Nonsurgical and surgical treatment of periodontitis; how many options for one disease? *Periodontol*. 2017;75(1);152-88.
20. Albuquerque C, et al. Canine periodontitis; the dog as an important model for periodontal studies. *Vet. J*. 2012;191:299-305.
21. Carranza, FA; Bone Loss and Pattern of Bone Destruction. In Newman, MG; Takei, HH; Carranza clinical periodontology 9th edition 2002, page 363.
22. Miron Rj, Fujioka- Kobayashi M, Bishara M. Platelet rich fibrin and soft tissue wound healing; a systematic review.
23. Al -Hamed FS, Mahri M, AL-Waeli H, Torres J. Regenerative effect of platelet concentrates in oral and craniofacial regeneration.
24. Kribel K, Hieke C, Muller-Hilke B, Nakata M.
25. How KY, Song KP, Chan KG.
26. Wachtel H, Schenk G, Weng Z. *J clin periodontal* 2003.