RESEARCH PAPER

Efficacy of a Probiotic Mouthrinse versus 0.2% Chlorhexidine as an Adjunct to Scaling and Root Planing in Stage II and Stage III Grade B Periodontitis

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ABSTRACT

Background

Periodontitis is a chronic inflammatory disease characterized by destruction of the supporting structures of the teeth. Scaling and root planing (SRP) remains the gold standard of non-surgical periodontal therapy; however, bacterial recolonization following SRP often necessitates the use of adjunctive chemotherapeutic agents. Chlorhexidine mouthwash is widely used for chemical plaque control but is associated with several adverse effects on long-term use. Probiotics have emerged as a promising alternative due to their ability to modulate the oral microbiome and host immune response.

Aim

The present randomized controlled trial aimed to compare and evaluate the efficacy of a probiotic mouthrinse with 0.2% chlorhexidine mouthwash as an adjunct to scaling and root planing in the treatment of stage II and stage III grade B periodontitis.

Materials and Methods

This single-blind, parallel-arm randomized controlled trial was conducted on 52 systemically healthy subjects aged 30-60 years diagnosed with stage II or stage III grade B periodontitis. All subjects underwent full-mouth scaling and root planing and were randomly allocated into two groups. Group A received a probiotic mouthrinse containing multiple Lactobacillus strains, while Group B received 0.2% chlorhexidine mouthwash. Clinical parameters assessed included relative attachment level and probing pocket depth at baseline, 1 month, and 3 months, and plaque index and gingival index at baseline, 14 days, 1 month, and 3 months. Standardized acrylic stents were used to ensure reproducibility of measurements. Statistical analysis was performed using appropriate non-parametric tests, and intergroup and intragroup comparisons were carried out.

Results

Both groups demonstrated statistically significant improvements in all clinical parameters over the study period. Intragroup analysis revealed significant reductions in plaque index, gingival index, probing pocket depth, and gains in relative attachment level at all follow-up intervals in both groups. Intergroup comparison showed no statistically significant differences between the probiotic and chlorhexidine groups with respect to most clinical parameters at corresponding time points. The probiotic mouthrinse exhibited comparable efficacy to 0.2% chlorhexidine in reducing plaque accumulation and gingival inflammation and improving periodontal parameters when used as an adjunct to scaling and root planing.

Conclusion

Within the limitations of the study, the probiotic mouthrinse was found to be as effective as 0.2% chlorhexidine mouthwash when used as an adjunct to scaling and root planing in the management of stage II and stage III grade B periodontitis.

Keywords: Probiotics; Chlorhexidine; Scaling and root planing; Periodontitis; Plaque index; Gingival index

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INTRODUCTION

Globally, one of the most prevalent oral conditions is condition affecting both the soft and hard structures periodontal disease.1 After dental caries, the second leading cause of tooth loss is periodontitis. It is an inflammatory condition affecting both the soft and hard structures supporting the teeth.2 There are numerous epidemiological studies showing

the relationship between dental plaque and the severity of gingival and periodontal conditions. It has been observed that improving gingival health and oral hygiene contributes to a decrease in periodontal disease.3 Antimicrobial therapies and plaque management are thus critical components of periodontal disease prevention and treatment.1 According to Cobb (2002), the purpose of periodontal therapy is to remove calculus deposits and unattached bacterial biofilms.4 Mechanical and chemical are the two methods to control plaque. Mechanical plaque control is more popular and cost- effective, but it is not always reliable because it relies on the manual dexterity, thoroughness, and compliance. When chemotherapeutic medications are used in conjunction with a mechanical regimen, the early stages of periodontal disease, plaque and gingivitis, can be effectively treated. Non-surgical periodontal therapy is regarded as the gold standard for treating chronic periodontitis.5 As stated by Claffey et al. (2004), scaling and root planing (SRP) and oral hygiene instructions play a key role in non-surgical therapy 6 The bacterial recolonization that occurs after SRP can be regarded as the primary disadvantage. As a result, other treatments, such as antibiotic therapy, antimicrobial photodynamic therapy, and, more recently, probiotic therapy, have been developed as adjuncts.2 The gold standard agent for chemical plaque control, chlorhexidine (CHX) is used most often.7 It is a bis-biguanide with four chlorophenyl rings and a central hexamethylene bridge connecting two biguanide groups. It acts as an antiseptic and functions effectively as an antimicrobrial agent in vitro against both Gram-positive and Gram-negative bacteria, as well as yeasts, fungi, aerobes, and anaerobes. Due to its binding properties, it exhibits substantivity (up to 12 hours) and a variety of bactericidal and bacteriostatic actions in the oral cavity. Because of its high tissue binding, CHX has a limited rate of absorption in the gastrointestinal tract, thereby reducing its systemic toxicity. After extended use, no teratogenic changes have been found. Since 1970, CHX has been used to treat gingivitis and plaque after it was shown that twice-daily 0.2% chlorhexidine di-gluconate rinses might prevent gingivitis and plaque accumulation. Because of its high substantivity, CHX is safe, stable, and effective in preventing and regulating the formation of new plaque, preventing and slowing the course of gingivitis, and relieving periodontal symptoms.8 The following adverse effects have been reported by various researchers: hypersensitivity reaction9, if the product is inserted into the middle ear, it may cause neurosensory deafness10, taste changes, especially those that impact salty and bitter flavors11-12, are reversible, parotid tumefaction can be unilateral or bilateral13-14, staining of the tongue dorsum, mucosa, teeth, or restorations13, erosion of the mucosa15 changes in the healing process16, and increased calculus formation17. In recent years, probiotics have been proposed as a substitute for treating periodontal diseases.1The term "probiotics" is a Greek word that means "for life". Probiotics are defined by the World Health Organization (WHO) and the Food and Agriculture Organization (FAO) as “live microorganisms which when administered in adequate amounts confer a health benefit on the host.”18 The most commonly used bacterial genera in probiotic formulations include Lactobacillus, Bifidobacterium, Escherichia, Enterococcus, Bacillus, and Streptococcus. Additionally, some Saccharomyces- related fungal strains have been utilized. In 1908, Nobel Prize winner Eli Methnikoff proposed that Bulgarian peasants' lifespan was attributed to their use of fermented milk products, which sparked the concept of probiotics. In 1965, Lilly and Stillwell coined the term "probiotic" to describe substances produced by one organism that aid in the development of another. They were defined as "microbial preparations or components of microbial cells that have a beneficial effect on health and well-being”. However, the concept of probiotics did not come into being until the beginning of the nineteenth century, when scientists hypothesized that beneficial bacteria may be used to replace harmful microorganisms in the gut flora. Probiotics are yeast or bacteria that are controlled as dietary supplements and food additives. They can be found in a range of fermented foods, most commonly yoghurt or dairy products, and can be supplied as capsules, tablets, packages, powders, or other forms. Probiotics are used in the treatment of many systemic conditions such as, Inflammatory Bowel Disease (IBD). Probiotics restore gut homeostasis by increasing protective microorganisms and decreasing inflammatory markers. Strains such as Saccharomyces boulardii, Lactobacillus acidophilus, and Bifidobacterium bifidum are beneficial in the treatment of ulcerative colitis and Crohn’s disease. Probiotics have antibacterial capabilities against dangerous pathogens such as Listeria monocytogenes, Salmonella typhimurium, E. coli, and H. pylori, which contribute in the creation of novel medications. Probiotics may improve symptoms of atopic eczema and respiratory allergies, such as asthma, by affecting the gut mucosa and immune response. Probiotics can aid in the relief of stress-related illnesses like depression and anxiety by promoting the gut-brain axis. Probiotics may help treat hypertension by increasing gut microbial balance, potentially offering an alternative to medicine that has fewer adverse effects. Certain Lactobacillus strains have the ability to bind carcinogens and block bacteria that turn procarcinogens into carcinogens, potentially lowering the risk of recurrent intestinal tumors and bladder cancer. Probiotics, particularly Lactobacillus crispatus and Lactobacillus iners, help maintain vaginal flora, preventing infections like vulvovaginal candidiasis (VVC). Lactobacilli and Bifidobacteria, can help with IBS symptoms including bloating, gas, and irregular bowel movements by lowering bacterial overgrowth in the small intestine and also significantly lower the risk of traveller’s diarrhoea.19 Probiotics have the potential to revolutionize the treatment of periodontal disease.1 It is thought that probiotic bacteria function by strengthening the host immune system, generating antimicrobial chemicals that fight

periodontopathogens, and preventing colonization of pathogenic species. Bifidobacterium and lactobacillus are usually given as tablets, powders or in the form of dairy products. The therapeutic effects of probiotics in periodontology have been researched in invitro and in vivo studies. Recently studies have examined the use of probiotics as an adjunct to supragingival and subgingival scaling in the treatment of periodontal disease.20 Kragen conducted the first research on the use of probiotics to improve oral health in 1954, treating periodontal inflammation. Gingivitis, periodontitis, pregnancy gingivitis, and other periodontal diseases were treated locally using a culture supernatant of a strain of Lactobacillus acidophilus. Almost all of the patients reported significant recovery.21According to Ricci, using probiotic chewing gum that included L. reuteri ATCC 55730 and ATCC PTA 5289 similarly reduced proinflammatory cytokine levels in GCF,46 and using L. brevis reduced salivary MMP (collagenase) activity and other inflammatory indicators.22 A few studies found that probiotic Lactobacillus strains reduced gingival irritation and the quantity of black-pigmented rods, including Porphyromonas gingivalis (Pg), in saliva and subgingival plaque.23 Lactobacillus rhamnosus inhibited both cariogenic species and Gram-negative periodontal pathogens. A study was carried out by Morales et al. (2018) in which a s an adjunct to nonsurgical treatment, the aim was to assess the benefits of probiotic sachets containing Lactobacillus rhamnosus SP1 and azithromycin pillsas an adjunct to nonsurgical treatment with respect to the clinical and microbiological characteristics of chronic periodontitis. They found that all groups exhibited improvements in both clinical and microbiological parameters across all evaluated time points. The probiotic and antibiotic groups demonstrated greater reductions in cultivable microbiota compared to baseline. The placebo group showed a more significant decrease in the number of subjects with P. gingivalis compared to baseline. However, no significant differences were observed between the groups. Hence, the additional use of L. rhamnosus SP1 sachets and azithromycin during initial therapy led to clinical and microbiological improvements comparable to those seen in the placebo group.24Lactobacillus reuteri and Lactobacillus brevis are specific markers for periodontal disease and have the ability to positively impact gingivitis and plaque composition.22,25 In relation to periodontal diseases, Teughels and colleagues demonstrated that the use of beneficial bacteria in conjunction with scaling and root planing (SRP) can prevent infections from recolonizing in periodontal pockets and reduce bleeding on probing in dogs.26 According to Shimauchi et al. the administration of probiotic capsules on a daily basis improved plaque index (PI) and probing depth, while another clinical research showed a decreased prevalence of moderate to severe gingival inflammation.27 As reported by Twetman el al., proinflammatory cytokines were decreased in gingivitis patients who consumed probiotic chewing gums for two weeks.28 Hence, the present study was conducted to compare and evaluate the efficacy of probiotic mouthrinse with 0.2% Chlorhexidine mouthwash, as an adjunct to scaling and root planing in the treatment of Stage II and Stage III Grade B Periodontitis

Materials and Method

Study design: An experimental, parallel, single blind randomized controlled study was conducted in subjects diagnosed with Stage II or Stage III Grade B Periodontitis. **Study population:** The study was performed on subjects reporting to the Out-Patient Department of Periodontology in a tertiary dental care center in accordance to the ADA type 2 specifications which includes dental chair with proper illumination and autoclaved set of instruments. Subjects within the age range of 30-60 years of either sex, reporting to the Out-Patient Department. Department of Periodontology, diagnosed with stage II and stage III grade B periodontitis were recruited for the study. **Sample size collection:** Approximately 26 subjects per group should complete the study at end point follow up. **Methods of data collection:** A total of 52 subjects diagnosed with Stage II and Stage III Grade B periodontitis were included in the study according to the study protocol. All the subjects underwent scaling and root planing. Oral hygiene instructions were given to be followed for the entire duration of the study. A verbal and written informed consent was obtained and subjects willing to participate were included in the study. The subjects were selected on the basis of the following inclusion and exclusion criteria:

Inclusion criteria:

1. Subjects of either sex between the age group of 30-60 years.
2. Subjects having minimum of 20 teeth present. 3. Subjects with Stage II and Stage III Grade B periodontitis having probing depth ≥4mm and ≤6mm with at least two teeth.
4. Systemically healthy and co-operative subjects.
5. Subjects willing to be a part of the study.

Exclusion criteria:

1. Presence of fixed partial dentures or removable partial dentures or any orthodontic appliances.
2. Subjects with history of antibiotic and/or antiinflammatory drugs within 1 month.
3. Pregnant women, women on oral contraceptive medication and lactating mothers.
4. Subjects who are smokers and/or tobacco chewers.
5. Subjects who have undergone any periodontal therapy in past 6 months.
6. Subjects allergic to components of probiotic mouthrinse and/or 0.2% chlorhexidine mouthwash

Withdrawal criteria:

1. Failure to report for reevaluation.
2. Non-compliant subjects
3. Any subjects reporting adverse effects of the drugs if any.

Subjects reporting any adverse reaction to contents of probiotic mouthrinse and 0.2% chlorhexidine mouthwash were withdrawn from the study and rescue drug were given. To match with original sample size fresh subjects were recruited for evaluation. Depending on the inclusion and exclusion criteria the subjects were randomly assigned into 2 groups: **Group A (26 subjects)** who were prescribed post scaling - Probiotic mouthrinse (sachet containing strains of lactobacillus salivarius, lactobacillus acidophillus, lactobacillus rhamnosus, lactobacillus paracasei, lactobacillus plantarum, lactobacillus reuteri) **Group B (26 subjects)** who were prescribed post scaling – 0.2% Chlorhexidine mouthwash.

Assessment of clinical parameters: The clinical parameters like relative attachment level and probing pocket depth were assessed at day 0, 1 month and 3 months and plaque index and gingival index were assessed at day 0, day 14, 1 month and 3 months.Customized acrylic stent was fabricated to standardize the measurements which were recorded by the examiner at specified time intervals using UNC-15 probe.

Methodology

The study was conducted after ethical clearance from the institute. This study was conducted on 52 subjects meeting the inclusion and exclusion criteria who gave verbal and written consent after being informed about the study protocol in the language that is best understood by them. Detailed case history was recorded for all the subjects participating in the study. Scaling and root planing was carried out for all the subjects participating in the study. Standard oral hygiene instructions were given to all the subjects participating in the study to be followed during the entire study duration. After which subjects were allocated randomly into 2 groups. In Group A, Probiotic sachets along with ampules of sterile water were given to the subjects. The subjects were instructed on how to formulate the mouthrinse by mixing 1 full probiotic sachet with 1 full 10ml sterile water ampoule and to use the mouthrinse immediately on formulation and advised to rinse for 1 min twice a day after 30 minutes of tooth brushing. In Group B, 0.2% Chlorhexidine gluconate mouthwash was given to the subjects. The subjects were instructed to follow the mouthwash usage as per instruction and advised to rinse with 10ml of for 1 min twice a day after 30 minutes of tooth brushing. The subjects were then recalled according to the follow-up timeline.

Probiotic Sachet, Sterile Water Ampoules, Prepared Probiotic Mouthrinse and Chlorhexidine Mouthwash

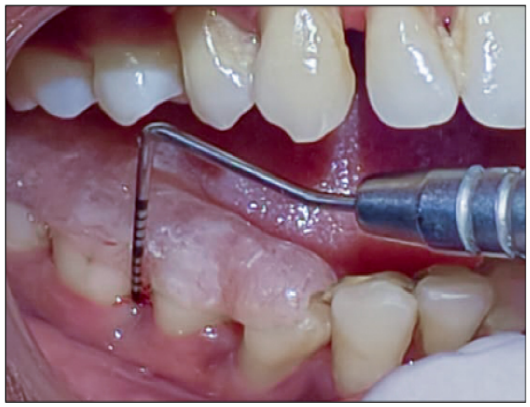
Statistical Analysis

All data were entered into a computer by giving coding system, proofed for entry errors

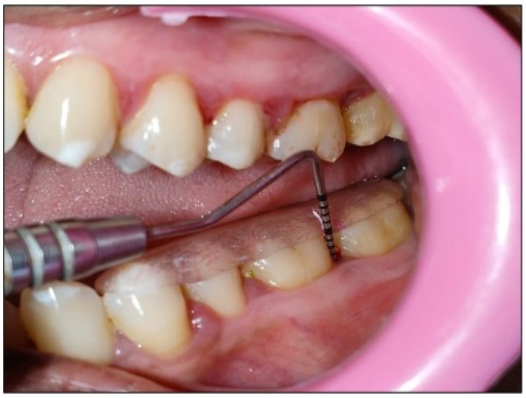
1. Data obtained was compiled on a MS Office Excel Sheet (v 2019, Microsoft Redmond Campus, Redmond, Washington, United States).
2. Data was subjected to statistical analysis using Statistical package for social sciences (SPSS v 26.0, IBM).
3. Descriptive statistics like frequencies and percentage for categorical data, Mean & SD for numerical data has been depicted.

Normality of numerical data was checked using ShapiroWilk test & was found that the data did not follow a normal curve; or for graded data, hence non-parametric tests have been used for comparisons.

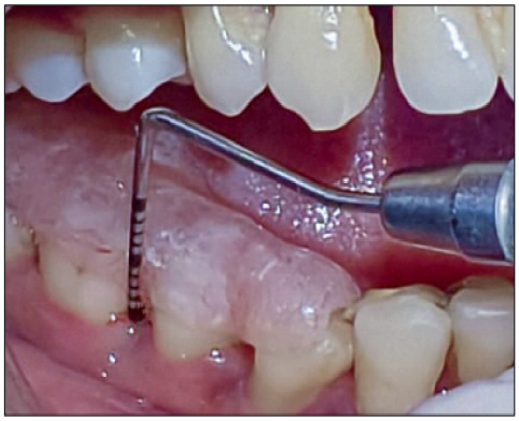
1. Inter group comparison (2 groups) was done using Mann Whitney U test.
2. Intra group comparison was done using Friedman's (for >2 observations) followed by pair wise comparison using Wilcoxon Signed rank test. For all the statistical tests, $p<0.05$ was considered to be statistically significant, keeping α error at 5% and β error at 20%, thus giving a power to the study as 80%. * = statistically significant difference ($p<0.05$) ** = statistically highly significant difference ($p<0.01$) # = non-significant difference ($p>0.05$)



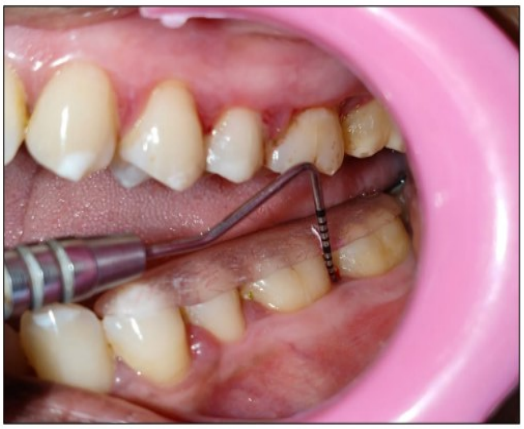
Group A Baseline



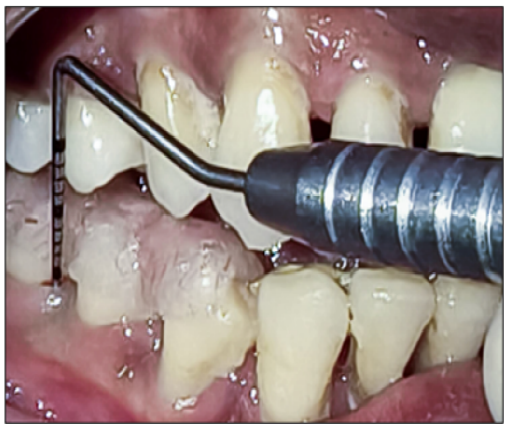
Group B Baseline



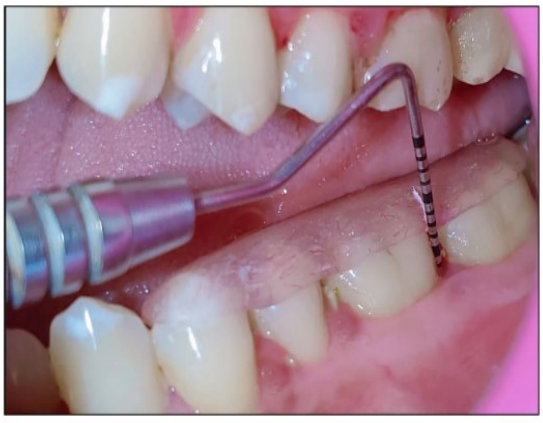
Group A 1 Month Follow-up



Group B 1 Month Follow-up



Group A 3 Months Follow-up



Group B 3 Months Follow-up

Results

Relative attachment level

On intragroup comparison, the mean RAL values for Group A at baseline were 5.92 ± 1.017 mm, at 1 month were 5.04 ± 1.248 mm, and at 3 months were 3.96 ± 1.248 mm. There was statistically highly significant difference seen ($p<0.01$) in RAL between baseline, 1 month and 3 month with higher values at baseline. The change from baseline to 3 months was 1.88 ± 1.092 mm, 1 month to 3 months was 1.04 ± 0.889 mm and baseline to 1 month was 0.84 ± 0.850 mm. The gain in RAL was highly significant. (Table 1, Table 3, Graph 1) The mean RAL values for Group B at baseline were 5.77 ± 0.908 mm, at 1 month were 4. 65 ± 1.093 mm and at 3 months were 3.73 ± 1.313 mm. Similar to group A there was statistically highly significant difference seen ($p<0.01$) in RAL between baseline, 1 month and 3 month with higher values at baseline .The change from baseline to 3 months was 1.96 ± 1.042 mm, 1 month to 3 months was 0.92 ± 0.881 mm, and baseline to 1 month was 1.04 ± 0.995 mm. This gain in RAL was highly significant. (Table 2, Table 3, Graph 1) On intergroup comparison, the mean RAL value in Group A at baseline was 5.92 ± 1.017 mm and at 3 months was 3.96 ± 1.248 mm and the mean RAL values in Group B at baseline was 5.77 ± 0.908 mm and at 3 months was 3.73 ± 1.313 mm. The mean difference in Group A and B from baseline to 3 months was 1.88 ± 1.092 mm and 1.96 ± 1.042 mm respectively. When the two groups were compared, the difference was statistically non-significant ($p>0.05$) at all time intervals. (Table 3, Table 4, Graph 2)

Probing pocket depth

On intragroup comparison, the mean probing pocket depth in Group A at baseline was 5.27 ± 0.452 mm, at 1 month was 4.38 ± 0.697 mm and at 3 months was 3.38 ± 0.496 mm. There was statistically highly significant difference seen ($p<0.01$) in probing pocket depth between baseline, 1 month and 3 month with higher values at baseline. The change from baseline to 3 months was 1.88 ± 0.666 mm, 1 month to 3 months was 1.00 ± 0.577 mm and baseline to 1 month was 0.88 ± 0.781 mm. The reduction in probing pocket depth was highly significant. (Table 5, Table 7, Graph 3) The mean probing pocket depth in Group B at baseline was 4.92 ± 0.845 mm, at 1 month was 4.19 ± 0.749 mm and at 3 months was 3.31 ± 0.788 mm. Similar to group A there was statistically highly significant difference seen ($p<0.01$) in probing pocket depth between baseline, 1 month and 3 month with higher values at baseline. The change from baseline to 3 months was 1.71 ± 1.042 mm, 1 month to 3 months was 0.92 ± 0.776 mm and baseline to 1 month was 0.79 ± 0.932 mm. This reduction in the probing depth was highly significant. (Table 6, Table 7, Graph 3) On intergroup comparison, the mean probing pocket depth in Group A at baseline was 5.27 ± 0.452 mm and at 3 months was 3.38 ± 0.496 mm and the mean probing pocket depth in Group B at baseline was 4.92 ± 0.845 mm and at 3 months was 3.31 ± 0.788 mm. The mean difference in Group A and B from baseline to 3 months was 1.88 ± 0.666 mm and 1.71 ± 1.042 mm respectively. When the two groups were compared, this difference was statistically non-significant ($p>0.05$). (Table 4, Table 7, Graph 4)

Gingival Index

On intragroup comparison, the mean gingival index values in Group A at baseline were 2.42 ± 1.27, at 14 days were 1.85 ± 0.64, at 1 month were 1.43 ± 0.51 and at 3 months were 0.71 ± 0.26 .There was statistically highly significant difference seen ($p<0.01$) in gingival index between baseline, 14days, 1 month and 3 month with higher values at baseline and the reduction in gingival index values was highly significant at all time follow-ups. (Table 8, Graph 5) The mean gingival index values in Group B at baseline were 2.164 ± 0.97, at 14 days were 1.67 ± 0.40, at 1 month were 1.41 ± 0.57 and at 3 months were 0.74 ± 0.16. There was statistically highly significant difference seen ($p<0.01$) in gingival index between baseline, 14 days, 1 month and 3 month with higher values at baseline. The reduction in gingival index values was statistically significant at all time follow-ups. (Table 9, Graph 5) On intergroup comparison, the mean gingival index value in Group A at baseline was 2.42 ± 1.27 and at 3 months was 0.71 ± 0.26 and the mean gingival index in Group B at baseline was 2.16 ± 0.97 and at 3 months was 0.74 ± 0.16. The mean difference in Group A and B from baseline to 3 months was 1.73 ± 1.16 and 1.45 ± 0.99 respectively. When the two groups were compared, this difference was statistically non-significant ($p>0.05$). (Table 10, Table 11, Graph 6) **Plaque Index**

On intragroup comparison, the mean plaque index values in Group A at baseline were 2.27 ± 0.96, at 14 days were 1.88 ± 0.58, at 1 month were 1.13 ± 0.75 and at 3 months were 0.53 ± 0.24. There was statistically highly significant difference seen ($p<0.01$) in plaque index between baseline, 14days, 1 month and 3 month with higher values at baseline and the change was statistically significant at all time follow ups. (Table 12, Graph 7) The mean plaque index values in Group B at baseline were 2.30 ± 0.95, at 14 days were 1.71 ± 0.43, 1 month were 1.24 ± 0.60 and at 3 months were 0.68 ± 0.26. There was statistically highly significant difference seen ($p<0.01$) in plaque index between baseline, 14days, 1 month and 3 month with higher values at baseline and the change was highly significant at all time follow - ups. (Table 13, Graph 7) On intergroup comparison, the mean plaque index value in Group A at baseline was 2.27 ± 0.96 and at 3 months was 0.53 ± 0.24 and the mean plaque index in Group B at baseline was 2.30 ± 0.95 and at 3 months was 0.68 ± 0.26. The mean difference in Group A and B from baseline to 3 months was 1.74 ± 0.80 and 1.66 ± 0.75 respectively. When the two groups were compared there was a statistically non-significant difference seen ($p>0.05$) at all time follow ups except there was a statistically significant difference between the change in plaque index values from14 days to 3 months with higher reduction in Group B. (Table 11, Table 14, Graph 8)

Tables and charts

* = statistically significant difference ($p<0.05$) ** = statistically highly significant difference ($p<0.01$) # = non-significant difference ($p>0.05$) … for all tables

Table 1: Intra group comparison of Relative Attachment Level in Group A between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Relative Attachment Level	26	5.92	1.017	4	7	6.00	2.79	42.467	0.000**
1 Month Relative Attachment Level	26	5.04	1.248	3	7	5.00	2.10		
3 Months Relative Attachment Level	26	3.96	1.248	2	6	3.00	1.12		

Table 2: Intra group comparison of Relative Attachment Level in Group B between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square Value	P Value of Friedman Test
Baseline Relative Attachment Level	26	5.77	0.908	4	8	6.00	2.83	41.540	0.000**
1 Month Relative Attachment Level	26	4.65	1.093	3	7	4.50	1.98		
3 Months Relative Attachment Level	26	3.73	1.313	2	6	3.00	1.19		

Table 3: Inter group mean diff comparison of Group A vs Group B for Relative Attachment Level using Mann-Whitney U test

	Group	N	Mean	Std. Dev.	Std. Error	Mean Diff	SD of Diff	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U test
bl to 1M	A	26	0.84	0.850	0.170	-0.202	0.258	1.000	266.0	-0.722	0.470
	B	26	1.04	0.955	0.195	-0.202	0.259	1.000			
bl to 3M	A	26	1.88	1.092	0.218	-0.078	0.305	2.000	283.0	-0.356	0.722
	B	26	1.96	1.042	0.213	-0.078	0.305	2.000			
1M to 3M	A	26	1.04	0.889	0.178	0.123	0.253	1.000	268.0	-0.688	0.491
	B	26	0.92	0.881	0.180	0.123	0.253	1.000			

Table 4: Inter group Pair wise comparison of Group A VS B for Probing Pocket Depth, Relative Attachment Level at baseline, 1 month and 3 month using Mann- Whitney U test

	Group	N	Mean	Std. Deviation	Mean Rank	Sum of Ranks	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U
Baseline Probing Pocket Depth	A	26	5.27	0.452	30.15	784	5.000	243.000	-1.955	0.051
	B	26	4.92	0.845	22.85	594	5.000			
1 Month Probing Pocket Depth	A	26	4.38	0.697	28.02	728.5	4.000	298.500	-0.788	0.431
	B	26	4.19	0.749	24.98	649.5	4.000			
3 Months Probing Pocket Depth	A	26	3.38	0.496	27.54	716	3.000	311.000	-0.562	0.574
	B	26	3.31	0.788	25.46	662	3.000			
Baseline Relative Attachment Level	A	26	5.92	1.017	27.88	725	6.000	302.000	-0.690	0.490
	B	26	5.77	0.908	25.12	653	6.000			
1 Month Relative Attachment Level	A	26	5.04	1.248	28.92	752	5.000	275.000	-1.192	0.233
	B	26	4.65	1.093	24.08	626	4.500			
3 Months Relative Attachment Level	A	26	3.96	1.248	27.94	726.5	3.000	300.500	-0.729	0.466
	B	26	3.73	1.313	25.06	651.5	3.000			

Table 5: Intra group comparison of Probing Pocket Depth in Group A between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Probing Pocket Depth	26	5.27	0.452	5	6	5.00	2.85	46.261	0.000**
1 Month Probing Pocket Depth	26	4.38	0.697	3	6	4.00	2.08		
3 Months Probing Pocket Depth	26	3.38	0.496	3	4	3.00	1.08		

Table 6: Intra group comparison of Probing Pocket Depth in Group B between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Probing Pocket Depth	26	4.92	0.845	4	7	5.00	2.67	38.000	0.000**
1 Month Probing Pocket Depth	26	4.19	0.749	3	5	4.00	2.10		
3 Months Probing Pocket Depth	26	3.31	0.788	2	5	3.00	1.23		

Table 7: Inter group mean diff comparison of Group A vs Group B for Probing Pocket Depth using Mann-Whitney U test

	Group	N	Mean	Std. Dev.	Std. Error	Mean Diff	SD of Diff	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U test
bl to 1M	A	26	0.88	0.781	0.156	0.088	0.245	1.000	272.0	-0.601	0.548
	B	26	0.79	0.932	0.190	0.088	0.246	0.000			
bl to 3M	A	26	1.88	0.666	0.133	0.172	0.249	2.000	259.0	-0.872	0.383
	B	26	1.71	1.042	0.213	0.172	0.251	1.000			
1M to 3M	A	26	1.00	0.577	0.115	0.083	0.195	1.000	279.0	-0.467	0.641
	B	26	0.92	0.776	0.158	0.083	0.196	1.000			

Table 8: Intra group comparison of Gingival Index in Group A between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Gingival Index	26	2.42692	1.275896	1.000	4.850	2.00000	3.60	63.012	0.000**
14 Days Gingival Index	26	1.85135	0.647759	1.000	3.100	1.89500	3.17		
1 Month Gingival Index	26	1.43808	0.512913	0.890	2.420	1.26000	2.23		
3 Months Gingival Index	26	0.71000	0.261916	0.280	1.140	0.67500	1.00		

Table 9: Intra group comparison of Gingival Index in Group B between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Gingival Index	26	2.16404	0.972077	1.000	4.140	1.70000	3.56	59.384	0.000**
14 Days Gingival Index	26	1.67896	0.403558	1.000	2.330	1.68500	3.12		
1 Month Gingival Index	26	1.41346	0.576673	.890	2.850	1.29000	2.33		
3 Months Gingival Index	26	0.74192	0.161370	.430	1.000	0.71000	1.00		

Table 10: Inter group mean diff comparison of Group A vs Group B for Gingival Index using Mann-Whitney U test

	Group	N	Mean	Std. Dev.	Std. Error	Mean Diff	SD of Diff	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U test
bl to 14D	A	26	0.559	0.915	0.183	0.063	0.256	0.740	285.5	-0.290	0.772
	B	26	0.496	0.877	0.179	0.063	0.256	0.360			
bl to 1M	A	26	0.996	0.822	0.164	0.217	0.205	0.800	279.0	-0.421	0.674
	B	26	0.780	0.590	0.120	0.217	0.204	0.540			
bl to 3M	A	26	1.734	1.168	0.234	0.275	0.311	1.380	276.0	-0.481	0.631
	B	26	1.458	0.998	0.204	0.275	0.310	0.990			
14D to 1M	A	26	0.438	0.369	0.074	0.154	0.134	0.460	254.5	-0.911	0.363
	B	26	0.284	0.553	0.113	0.154	0.135	0.245			
14D to 3M	A	26	1.175	0.562	0.112	0.212	0.141	1.085	236.0	-1.280	0.200
	B	26	0.963	0.407	0.083	0.212	0.140	0.925			
1M to 3M	A	26	0.737	0.472	0.094	0.059	0.154	0.550	244.5	-1.115	0.265
	B	26	0.678	0.601	0.123	0.059	0.155	0.525			

Table 11: Inter group Pair wise comparison of Group A VS B For Gingival Index, Plaque Index at baseline, 14days, 1 month and 3 month using Mann- Whitney U test

	Group	N	Mean	Std. Deviation	Mean Rank	Sum of Ranks	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U test
Baseline Gingival Index	A	26	2.42692	1.275896	28.31	736	2.000	291.000	-0.864	0.387
	B	26	2.16404	0.972077	24.69	642	1.700			
14 Days Gingival Index	A	26	1.85135	0.647759	27.85	724	1.895	303.000	-0.647	0.517
	B	26	1.67896	0.403558	25.15	654	1.685			
1 Month Gingival Index	A	26	1.43808	0.512913	27.23	708	1.260	319.000	-0.349	0.727
	B	26	1.41346	0.576673	25.77	670	1.290			
3 Months Gingival Index	A	26	0.71000	0.261916	24.38	634	0.675	283.000	-1.011	0.312
	B	26	0.74192	0.161370	28.62	744	0.360			
Baseline Plaque Index	A	26	2.27077	0.961627	26.87	698.5	2.000	328.500	-0.176	0.860
	B	26	2.30538	0.950698	26.13	679.5	1.660			
14 Days Plaque Index	A	26	1.88231	0.585466	27.67	719.5	1.700	307.500	-0.563	0.573
	B	26	1.71242	0.432258	25.33	658.5	1.725			
1 Month Plaque Index	A	26	1.13192	0.751707	24.62	640	0.900	289.000	-0.899	0.368
	B	26	1.24308	0.609680	28.38	738	0.950			
3 Months Plaque Index	A	26	0.53654	0.240748	23.29	605.5	0.585	254.500	-1.533	0.125
	B	26	0.68308	0.264602	29.71	772.5	0.630			

Table 12: Intra group comparison of Plaque Index in Group A between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Plaque Index	26	2.27077	0.961627	0.660	3.570	2.00000	3.75	66.642	0.000**
14 Days Plaque Index	26	1.88231	0.585466	1.160	3.080	1.70000	3.17		
1 Month Plaque Index	26	1.13192	0.751707	0.250	2.420	0.90000	1.96		
3 Months Plaque Index	26	0.53654	0.240748	0.200	1.000	0.58500	1.12		

Table 13: Intra group comparison of Plaque Index in Group B between baseline, 1 month and 3 months

	N	Mean	Std. Deviation	Minimum	Maximum	Median	Mean Rank	Chi-Square value	p value of Friedman Test
Baseline Plaque Index	26	2.30538	0.950698	1.410	3.570	1.66000	3.73	65.907	0.000**
14 Days Plaque Index	26	1.71242	0.432258	0.660	2.830	1.72500	3.08		
1 Month Plaque Index	26	1.24308	0.609680	0.620	2.570	0.95000	2.19		
3 Months Plaque Index	26	0.68308	0.264602	0.320	1.140	0.63000	1.00		

Table 14: Inter group mean diff comparison of Group A vs Group B for Plaque Index using Mann-Whitney U test

	Group	N	Mean	Std. Dev.	Std. Error	Mean Diff	SD of Diff	Median	Mann-Whitney U value	Z Value	p value of Mann-Whitney U test
bl to 14d	A	26	0.374	0.818	0.164	-0.248	0.232	0.500	269.0	-0.620	0.535
	B	26	0.622	0.804	0.164	-0.248	0.232	0.351			
bl to 1M	A	26	1.140	0.355	0.071	0.046	0.143	1.150	250.5	-0.993	0.321
	B	26	1.094	0.616	0.126	0.046	0.144	0.790			
bl to 3M	A	26	1.748	0.806	0.161	0.079	0.224	1.600	249.0	-1.022	0.307
	B	26	1.668	0.757	0.154	0.079	0.223	1.100			
14D to 1M	A	26	0.766	0.556	0.111	0.295	0.170	0.615	232.0	-1.360	0.174
	B	26	0.472	0.634	0.129	0.295	0.171	0.440			
14D to 3M	A	26	1.374	0.559	0.112	0.328	0.139	1.335	196.5	-2.071	0.038*
	B	26	1.046	0.397	0.081	0.328	0.138	1.000			
1M to 3M	A	26	0.607	0.633	0.127	0.033	0.155	0.550	279.0	-0.421	0.674
	B	26	0.574	0.431	0.088	0.033	0.154	0.390			

Table 15: Tests of Normality

	Group	Shapiro-Wilk		
		Statistic	df	p value
BL Gingival Index	A	.857	26	.002
	B	.868	26	.003
14d Gingival Index	A	.923	26	.054
	B	.890	26	.010
1M Gingival Index	A	.829	26	.001
	B	.716	26	.000
3M Gingival Index	A	.938	26	.122
	B	.911	26	.028
BL Plaque Index	A	.867	26	.003
	B	.703	26	.000
14d Plaque Index	A	.856	26	.002
	B	.943	26	.159
1M Plaque Index	A	.871	26	.004
	B	.828	26	.001
3M Plaque Index	A	.917	26	.038
	B	.916	26	.036
BL Probing Pocket Depth	A	.557	26	.000
	B	.842	26	.001
1M Probing Pocket Depth	A	.833	26	.001
	B	.800	26	.000
3M Probing Pocket Depth	A	.619	26	.000
	B	.847	26	.001
BL Relative Attachment Level	A	.836	26	.001
	B	.889	26	.009
1M Relative Attachment Level	A	.919	26	.042
	B	.898	26	.014
3M Relative Attachment Level	A	.814	26	.000
	B	.863	26	.003

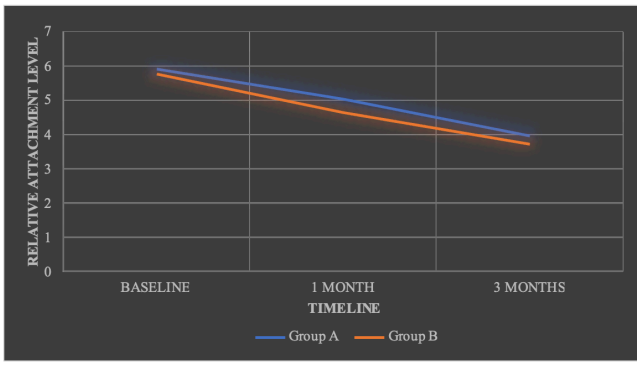
Table 16: Pair wise comparison in Group A between Gingival Index, Plaque Index, Probing Pocket Depth, Relative Attachment Level at baseline, 14days, 1 month and 3 months

Pairs	Z value	p value of Wilcoxon Signed Ranks
14 Days Gingival Index - Baseline Gingival Index	-2.758	0.006**
1 Month Gingival Index - Baseline Gingival Index	-4.204	0.000**
3 Months Gingival Index - Baseline Gingival Index	-4.463	0.000**
1 Month Gingival Index - 14 Days Gingival Index	-3.809	0.000**
3 Months Gingival Index - 14 Days Gingival Index	-4.458	0.000**
3 Months Gingival Index - 1 Month Gingival Index	-4.464	0.000**
14 Days Plaque Index - Baseline Plaque Index	-2.383	0.017*
1 Month Plaque Index - Baseline Plaque Index	-4.469	0.000**
3 Months Plaque Index - Baseline Plaque Index	-4.464	0.000**
1 Month Plaque Index - 14 Days Plaque Index	-4.259	0.000**
3 Months Plaque Index - 14 Days Plaque Index	-4.459	0.000**
3 Months Plaque Index - 1 Month Plaque Index	-3.930	0.000**
1 Month Probing Pocket Depth - Baseline Probing Pocket Depth	-3.944	0.000**
3 Months Probing Pocket Depth - Baseline Probing Pocket Depth	-4.574	0.000**
3 Months Probing Pocket Depth - 1 Month Probing Pocket Depth	-4.400	0.000**
1 Month Relative Attachment Level - Baseline Relative Attachment Level	-3.630	0.000**
3 Months Relative Attachment Level - Baseline Relative Attachment Level	-4.425	0.000**
3 Months Relative Attachment Level - 1 Month Relative Attachment Level	-3.746	0.000**

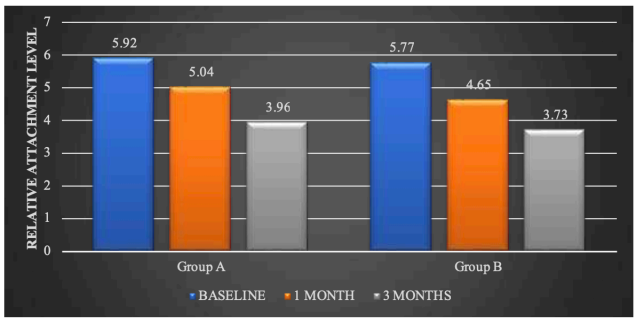
Table 17: Pair wise comparison I Group B between Gingival Index, Plaque Index, Probing Pocket Depth, Relative Attachment Level at baseline, 14days, 1 month and 3 months

Pairs	Z Value	P Value Of Wilcoxon Signed Ranks Test
14 Days Gingival Index - Baseline Gingival Index	-2.585	0.010*
1 Month Gingival Index - Baseline Gingival Index	-4.215	0.000**
3 Months Gingival Index - Baseline Gingival Index	-4.465	0.000**
1 Month Gingival Index - 14 Days Gingival Index	-2.517	0.0120*
3 Months Gingival Index - 14 Days Gingival Index	-4.460	0.000**
3 Months Gingival Index - 1 Month Gingival Index	-4.465	0.000**
14 Days Plaque Index - Baseline Plaque Index	-3.102	0.002**
1 Month Plaque Index - Baseline Plaque Index	-4.465	0.000**
3 Months Plaque Index - Baseline Plaque Index	-4.465	0.000**
1 Month Plaque Index - 14 Days Plaque Index	-3.138	0.002**
3 Months Plaque Index - 14 Days Plaque Index	-4.460	0.000**
3 Months Plaque Index - 1 Month Plaque Index	-4.465	0.000**
1 Month Probing Pocket Depth - Baseline Probing Pocket Depth	-3.126	0.002**
3 Months Probing Pocket Depth - Baseline Probing Pocket Depth	-4.266	0.000**
3 Months Probing Pocket Depth - 1 Month Probing Pocket Depth	-3.758	0.000**
1 Month Relative Attachment Level - Baseline Relative Attachment Level	-3.804	0.000**
3 Months Relative Attachment Level - Baseline Relative Attachment Level	-4.434	0.000**
3 Months Relative Attachment Level - 1 Month Relative Attachment Level	-3.692	0.000**

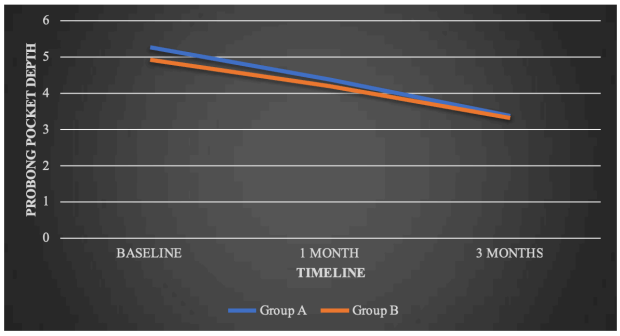
Graph 1: Intragroup comparison of Relative Attachment Level in Group A and Group B



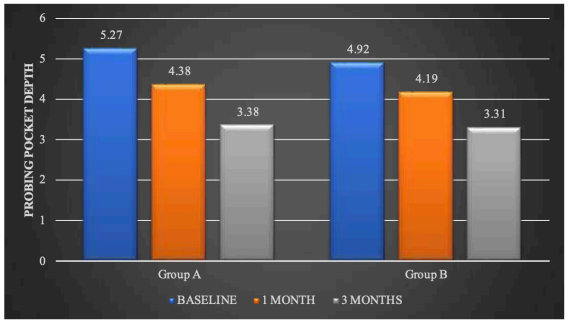
Graph 2: Inter Group Comparison of Relative Attachment Level



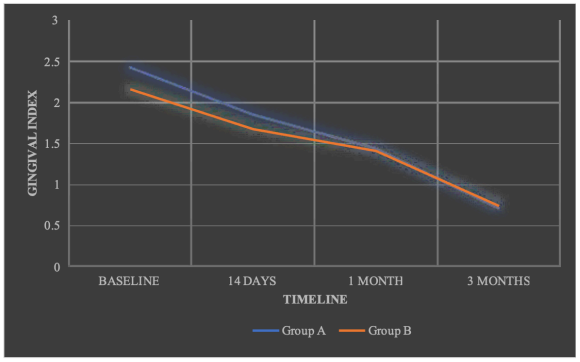
Graph 3: Intragroup comparison of Probing Pocket Depth in Group A and Group B



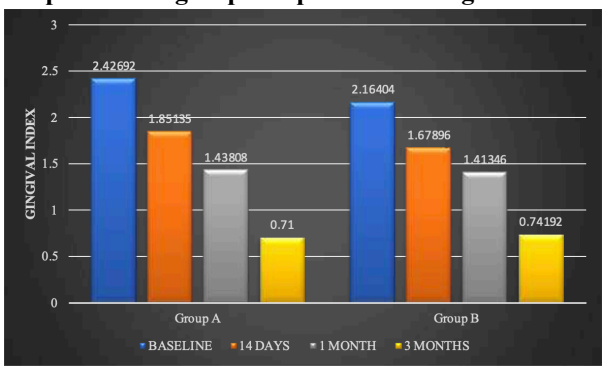
Graph 4: Inter group comparison of Probing Pocket Depth



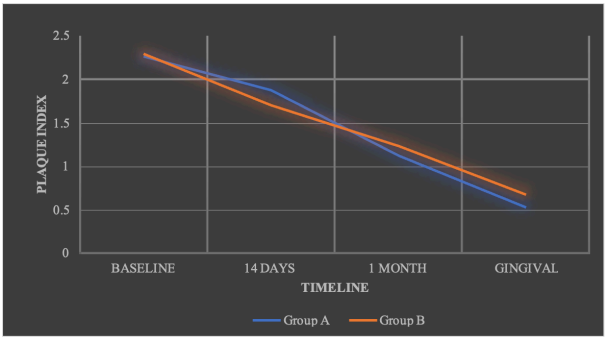
Graph 5: Intragroup comparison of Gingival Index in Group A and Group B



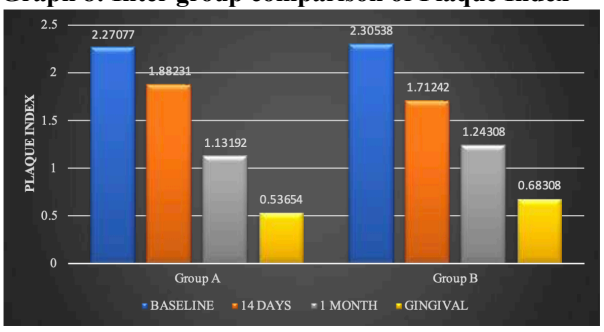
Graph 6: Inter group comparison of Gingival Index



Graph 7: Intragroup comparison of Plaque Index in Group A and Group B



Graph 8: Inter group comparison of Plaque Index



Discussion

Periodontal disease is a common chronic inflammatory condition affecting the tissues supporting the teeth and characterized by a high inflammatory response to oral microbiome dysbiosis. Microbiological dysbiosis is caused by pathogenic bacteria such as Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans, causing an excessive host inflammatory response, resulting in tissue destruction. Understanding how oral microbiota dysbiosis contributes to the initiation and progression of periodontal disease is essential in developing effective therapeutic strategies to reduce the bacterial load and restore the microbiological balance.29 Mechanical therapies, such as non-surgical periodontal therapy (NSPT), successfully decrease bacterial burden. Ultrasonic and manual devices are used to thoroughly remove the supra and subgingival biofilm and calculus as part of non-surgical periodontal treatment (NSPT). But they frequently fall short of reestablishing the oral microbiota's natural equilibrium, which is necessary for sustained periodontal health.29 According to Jones (1997), Chlorhexidine is considered the gold standard against which other antiplaque and gingivitis treatments are compared.30 In the 1940s, Imperial Chemical Industries in England created chlorhexidine. In 1950, it was marketed as a general antiseptic. Chlorhexidine was first made available to the public in Britain in 1957 as a skin antiseptic. Later, it was extensively employed in surgery and medicine. Chlorhexidine is a symmetrical compound. It consists of two biguanide groups joined by a central hexamethylene bridge and four chlorophenyl rings.31 Chlorhexidine's greater antiplaque activity is attributed to its long-term availability-substantivity. The effects of chlorhexidine vary depending on the concentration. In low doses, it is bacteriostatic; in high ones, it is bactericidal. Different bacterial species have different concentrations. Saliva itself has antibacterial properties for around five hours and reduces salivary bacterial counts for more than twelve hours following a single rinse with chlorhexidine. Saliva's aerobic and anaerobic species can be lowered by 80–90% after many chlorhexidine treatments. In the oral cavity, chlorhexidine has also been discovered to be a strong antifungal agent. Chlorhexidine inhibits plaque by 3 mechanisms – 1)

By preventing pellicle formation and lowering glycoprotein adsorption onto the tooth surface by inhibiting acidic groups on salivary glycoproteins, chlorhexidine decreases plaque. 2) Preventing the bacterial cell wall from adhering to the tooth surface by attaching itself to the bacterium. 3) By causing agglutination components to precipitate in the saliva and removing calcium from the plaque matrix, mature plaque cannot bond.32 Long-term use of chlorhexidine mouthwash, can cause brownish staining of the teeth and tongue, oral mucosal erosion, and taste disturbances, as well as rare unilateral or bilateral parotid swellings.33 Therefore, there was a need felt to develop other chemical plaque control agents as an adjunct to mechanical treatment. Probiotics, a type of helpful bacteria, have been used successfully in gastrointestinal diseases and are now being introduced into dentistry. The human gut is home to ten times as many microorganisms as compared to other parts of the body.

This biomass is made up of more than 400 recognized bacterial species that contribute significantly to human health through a variety of metabolic processes. When exposed to toxins, such as through the unrestrained use of medications like antibiotics or contaminated food and water, these microflora activities are disturbed. Probiotics can help restore normal functions disturbed by bacterial toxins. The first study conducted by Holocombh in 1991 recognised Bifidobacterium bifidum as a probiotic species. When routinely prescribed antibiotics become ineffective due to development of resistance.in 1994 World Health Organization declared that probiotics are the second most essential immune defence system.34 Probiotics are defined by the World Health Organization (WHO) and the Food and Agriculture Organization (FAO) as “live microorganisms which when administered in adequate amounts confer a health benefit on the host.”18 It has been suggested that Regulatory T lymphocytes (Tregs), are essential for lowering inflammation in response to non-pathogenic antigens.34 According to the research conducted by Boden and Snapper (2008), interactions between mast cells, T lymphocytes, and dendritic cells may be mediated by toll-like receptor (TLR) pathways.35 Their interaction aids in regulating allergic immune responses. The TLR signaling by the commensals in intestinal microbiota is crucial for immune control and intestinal epithelium hemostasis and protection against epithelial damage. TLRs trigger the synthesis of epithelial repair components by identifying pattern recognition molecules from commensal microbes. This is a key method by which probiotics work. probiotics interact with immune and epithelial cells and change signaling pathways. Probiotics include yeast, bacteria, and molds, with bacterial species being the most frequent. The two genera Lactobacillus and Bifidobacterium species are the primary sources of the most widely utilized probiotics. Numerous species of Lactobacillus, including Lactobacillus acidophilus, Lactobacillus brevis, Lactobacillus casei, and Lactobacillus salvarius, have been found. These species produce digestive enzymes that aid in the metabolism of proteins and carbohydrates.34 The Lactobacillus species aid in the breakdown of bile salts as well as the creation of vitamins B and K. They aid in the suppression of pro-inflammatory mediators and the improvement of both innate and acquired immunity. Porphyromonas gingivalis, Treponema denticola, Aggregatibacter actinomycetemcomitans, and Tannerella forsythia are the primary pathogenic organisms linked to periodontitis. These bacteria exhibit a range of virulent traits, including colonization at subgingival locations, evasion of the host's defences, and tissue destruction. The disease's progression is also influenced by how long the host's immunological response lasts. According to numerous research conducted worldwide, oral probiotic use may also help treat periodontal disease. Probiotic species like Lactobacillus can inhibit the growth of P. gingivalis, Prevotella intermedia, and A. actinomycetemcomitans. The Study demonstrated that lactobacilli play a critical role in preserving the equilibrium of the oral microbiota. Probiotics to treat periodontal disease are available in the form of tablets, chewing gum, lozenges.34 Teughels et al. (2013) conducted a randomized placebo-controlled clinical trial to evaluate the adjunctive effect of Lactobacillus reuteri-containing probiotic lozenges with scaling and root planing (SRP) in chronic periodontitis. They found that the SRP + probiotic group exhibited significantly greater pocket depth reduction, attachment level gain, and a more substantial decrease in Porphyromonas gingivalis levels at 12 week when compared to SRP alone

group.36 Another study conducted by Nadkerny et al. (2015) evaluated and compared the antiplaque and anti-inflammatory effects of a probiotic mouthwash, 0.2% chlorhexidine, and saline. They concluded that probiotic mouthrinses, when used alongside mechanical plaque control, effectively reduced plaque and gingivitis.37 Hence, the present study was conducted to compare and evaluate the efficacy of probiotic mouthrinse with 0.2% Chlorhexidine mouthwash, as an adjunct to scaling and root planing in the treatment of Stage II and Stage III Grade B Periodontitis. Based on the inclusion and exclusion criteria a total of 52 subjects were selected for final statistical analysis after considering drop outs in each group. All the subjects reported for the follow up and there were no dropouts. The subjects were divided in 2 groups: Group A and Group B consisting of 26 subjects each. In Group A, probiotic sachets along with ampules of sterile water were given to the subjects to be used as a mouthrinse. In Group B, 0.2% chlorhexidine gluconate mouthwash was given to the subjects. The following clinical parameters: relative attachment level, probing pocket depth, gingival index and plaque index were assessed at baseline and the changes in relative attachment level and probing pocket depth were assessed at 1 month and 3 months while gingival index and plaque index were assessed at 14 days, 1 month and 3 months.

Relative attachment level (RAL)

A statistically significant gain in RAL was observed in both the probiotic group (Group A) and the chlorhexidine group (Group B) at 3 months. While there was a significant improvement within each group, the intergroup comparison showed no significant difference. These findings are in agreement with the results reported by Costacurta et al. (2018), where the use of a Lactobacillus reuteri probiotic lozenge led to significant clinical attachment gains when used adjunctively with mechanical therapy.38 Similarly, in a study conducted by Teughels et al. (2013), where probiotics in the form of lozenges supplemented with SRP showed gain in clinical attachment. In this study the lozenges were compared with placebo, hence significant difference was observed between the test and control groups.36 In accordance with the study results in the study conducted by, Vishnupriya et al. (2022), probiotic and chlorhexidine were used in the form of local drug delivery agents and statistically non-significant difference in Clinical attachment level (CAL) was found between the two groups.39 The observed RAL improvements can be attributed to the ability of probiotics to modulate the inflammatory response and enhance periodontal healing.

Probing pocket depth (PPD)

The mean PPD showed statistically significant reductions within both groups from baseline to 3 months. However, intergroup differences remained statistically non- significant. Costacurta et al. (2018) similarly reported notable reductions in PPD with probiotic use over 3 months, showing comparable effectiveness to chlorhexidine.38 These findings are consistent with Sachelarie et al. (2025) and Vanditha et al. (2025), who demonstrated that probiotics as adjuncts to periodontal therapy led to significant pocket depth reductions.40,41 In the study conducted by Vishnupriya et al. (2022), probiotic and chlorhexidine were used in the form of local drug delivery agents. They found statistically non-significant difference in PPD between the two groups.39 Probiotics may exert an inhibitory effect on periodontal pathogens, thereby reducing inflammation and pocket depth over time.

Gingival index (GI)

The GI scores significantly reduced in both groups over the 3-month follow-up period, with no significant difference between groups. These findings are in agreement with the study conducted by Costacurta et al. (2018), who noted a decrease in gingival inflammation with probiotic use.38 Nadkerny et al. (2015) further validated these findings by showing that probiotic and chlorhexidine mouthrinses had comparable reductions in gingival index values.37 Moreover, Harini and Anegundi (2010) demonstrated that a probiotic mouthrinse containing Lactobacillus species significantly reduced gingival scores in pediatric subjects over a two-week period when compared with Chlorhexidine mouthwash. They observed a significant reduction in the gingival index values in the test group as compared to the control which was contrasting with the results of this study.42 Additionally, Krasse et al.

(2006) reported significant reductions in gingival bleeding and inflammation after probiotic use.25

Plaque index (PI)

Both groups demonstrated a significant reduction in plaque index scores. Although both treatments were effective, chlorhexidine showed a slightly greater reduction at certain time intervals, consistent with its established efficacy. The results are in line with findings by Costacurta et al. (2018) and Singh et al. (2021), who highlighted that probiotic significantly reduced plaque formation, although chlorhexidine remains marginally more effective short-term.38,43 In the study by Nadkerny et al. (2015), probiotic mouthrinses led to a significant reduction in PI comparable to that of chlorhexidine.37 Similarly, in a study by Harini and Anegundi (2010) and Noordin and Kamin (2007) demonstrated substantial reductions in plaque after probiotic mouthrinse use.42,44 In a study by Boyapati et al. (2024) they found a non-significant difference in probiotic group when compared to the chlorhexidine group as both groups showed similar reduction in plaque scores as seen in the present study.45 Overall, the study findings reinforce that while chlorhexidine remains highly effective, probiotics provide a promising alternative with comparable clinical outcomes. Moreover, probiotics offer the added advantage of fewer side effects like staining and taste alteration, often associated with long-term chlorhexidine use. The probiotic mouthrinse was well accepted by all subjects, with no reported adverse effects such as allergic reactions or oral lesions, whereas subjects in the chlorhexidine group reported an increase in tooth staining. Despite these findings, the study had several limitations. The follow-up period was limited to three months, preventing assessment of long-term clinical outcomes. Additionally, no direct microbiological analysis was performed, restricting insights into changes in pathogen load or the oral microbiome induced by probiotic use. Although the sample size was statistically adequate, the study was conducted at a single tertiary dental center with a relatively homogeneous population, which may limit the generalizability of the results to broader populations. The study also depended on patient compliance, requiring daily reminders, and the probiotic mouthrinse needed to be freshly prepared before each use, which could affect consistency. Furthermore, the study design was single-blinded, with only the statistician blinded, while both the examiner and subjects were aware of group allocation, introducing the possibility of bias in the findings.

Conclusion

The present randomized controlled clinical trial was designed to evaluate and compare the efficacy of a probiotic mouthrinse and 0.2% chlorhexidine mouthwash as an adjunct to scaling and root planing (SRP) in the treatment of Stage II and Stage III Grade B periodontitis. Within the limitations of the study, it can be concluded that both treatment modalities significantly improved the clinical outcomes, namely, Relative Attachment Level (RAL), Probing Pocket Depth (PPD), Gingival Index (GI), and Plaque Index (PI) over a period of three months. While chlorhexidine has been long established as the gold standard for chemical plaque control due to its potent antibacterial properties, the probiotic mouthrinse demonstrated comparable clinical improvements. The probiotic group showed statistically significant reductions in probing depths, plaque accumulation, and gingival inflammation, similar to the chlorhexidine group, with no statistically significant differences between the groups in most parameters at three months. Moreover, the results corroborate existing literature, including studies by Nadkerny et al. (2015), Vivekananda et al. (2010), and Teughels et al. (2013), affirming that probiotics exhibit anti-inflammatory properties thus promoting periodontal health. Importantly, probiotics bring forth a novel therapeutic approach without the side effects often associated with chlorhexidine, such as mucosal irritation, altered taste sensation, and dental staining. This reflects a shift towards natural, biologically safe, and sustainable options in periodontal therapy. Thus probiotics offer a promising adjunctive therapy for the management of periodontal diseases. However, further research with larger sample sizes, longer follow-up periods, and microbiological investigations is warranted to validate and optimize probiotic therapy protocols.

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