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# EVALUATION OF THE EFFICACY OF GINGER GEL IN THE TREATMENT OF CHRONIC PERIODONTITIS: A CLINICAL AND **QUALITATIVE ANALYSIS**

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#### **ABSTRACT**

**Introduction:** Periodontitis is a disease of the oral cavity resulting in the inflammation and destruction of the tooth-supporting tissues, primarily caused by the accumulation of complex polymicrobial plaque. Periodontitis can be conventionally treated through mechanical scaling and root planing (SRP). The local delivery of antimicrobials to periodontal pockets has the benefit of administering more drugs at the target site, while minimizing the exposure of the total body. Recently, there is an increased awareness and scientific trials on medicinal plants and their formulations. Hence, the present study was designed to assess the clinical and microbial effects of Ginger gel as a local drug delivery in the treatment of patients with Chronic Generalized Periodontitis.

Materials and Methods: Thirty patients were randomly assigned to study and control groups. Group I - Scaling and Root planing alone, Group II - Scaling and Root planing along with Ginger gel as LDD. Clinical parameters such as gingival index, gingival bleeding index, probing pocket depth, periodontal index along with subgingival plaque samples were collected at baseline and after 1 month for microbial colony forming units. Results: Periodontal therapy with ginger gel as LDD resulted in significant improvement in the clinical parameters and a significant decrease in the microbial colony forming units after 1 month. Conclusion: Ginger gel as LDD was effective clinically and microbiologically, in

reducing the severity of periodontal inflammation and the overall microbial count. Hence, ginger gel can be used as an adjunct to periodontal therapy due to its numerous properties. However further studies need to be conducted on larger samples and a longer follow up to evaluate its effect on periodontal diseases.

## **INTRODUCTION**

Periodontitis is a disease of the oral cavity consisting of inflammation of the tooth-supporting tissues, primarily caused by the accumulation of complex polymicrobial dental plaque. Periodontal inflammation is initiated by Gram-negative tooth-associated microbial biofilms that elicit a host response, resulting in progressive, irreversible bone and soft tissue destruction.<sup>[1]</sup>

Porphyromonas, Prevotella species, and A. actinomycetemcomitans are the principal oral pathogens in periodontitis, as they produce several endodontopathogenic materials. They activate monocytes/macrophages to produce pro-inflammatory vasoactive mediators such as prostaglandin E2 (PGE2) and interleukin-1 (IL-1). These products suppress host defense mechanisms and destroy periodontal tissues.<sup>[2,3,4]</sup>

Therefore, elimination of microbial dental plaque biofilm prevents gingivitis and periodontitis, by controlling bacterial accumulation and plaque formation.<sup>[5]</sup> This can be achieved through the traditional method of mechanical scaling and root planing (SRP) which has shown to be an effective treatment for chronic periodontitis.<sup>[6]</sup> Although mechanical treatment significantly decreases the prevalence and levels of subgingival microorganisms, it does not necessarily eliminate all pathogens, due to the lack of accessibility in deep periodontal pockets.<sup>[7]</sup>

Elimination and suppression of putative periodontopathic microorganisms in the subgingival microbiota is essential for periodontal healing. For effective treatment, the antibiotic must reach the depth of the pocket and produce gingival fluid concentrations higher than the minimum inhibitory concentrations (MIC) of the suspected pathogens.<sup>[8]</sup>

Periodontal therapy with local and systemic antibiotics have been employed to facilitate the elimination of subgingival microflora. However, repeated and long-term use of systemic antibiotics results in resistant strains and superimposed infections. Local administration, therefore, delivers the therapeutic agent into the base of the pocket and is maintained there

like a reservoir for an adequate period until the antimicrobial effect occurs.<sup>[10]</sup> In 1979 Dr. Max Goodson et al first proposed the concept of controlled delivery in the treatment of periodontitis.<sup>[11]</sup>

The local delivery of antimicrobial therapy to periodontal pockets has the benefit of administering more drugs at the target site while minimizing the exposure of the total body to the drug and the sustained release of antimicrobial within the periodontal pockets.<sup>[12]</sup>

The common agents used include subgingival chlorhexidine, tetracycline fibers, subgingival minocycline, subgingival doxycycline, and subgingival metronidazole. With the rise in bacterial resistance to antibiotics, the use of herbal products in dentistry is ever increasing. This can be attributed to specific bioactive constituents of plants namely alkaloids, flavonoids, tannins, and phenolic products [13] and their ease of availability, low cost, and less side effects. [14]

Ginger (Zingiber officinale) is a medicinal plant that has been used traditionally in the Chinese and Ayurverdic herbal medicines as an anti-inflammatory agent that can reduce prostaglandin biosynthesis via inhibiting COX-1 and COX-2 enzymes and exert inhibitory effects against periodontal bacterial growth of anaerobic Gram-negative bacteria including Prevotella intermedia, Porphyromonas endodontalis and Porphyromonas gingivalis.<sup>[5]</sup>

Among natural food sources with antimicrobial activities, ginger rhizome (Zingiber officinale Roscoe; family Zingiberaceae) has been used as widely grown food spices and medicinal crops for centuries. Moreover, it is a natural source with no toxicity, which is considered as 'generally recognized as safe' (GRAS) in the Food and Drug Administration (FDA) of the United States. The pungent oil components of ginger harbor a series of polyphenolic ketones called gingerols with many pharmacological activities and possess antibacterial and antifungal, antimicrobial properties.<sup>[4]</sup>

Ginger's pungent components offer powerful anti-inflammatory and antioxidant activities, making it useful in arthritis, Alzheimer's, cancer, prevents blood clots and cardiovascular disease. The active compound responsible for this effect is zingibain, an enzyme that counteracts inflammation. Research suggests this root protects nerve cells in the brain, potentially preventing Alzheimer's disease. Ginger not only increases insulin level but it also helps lower blood pressure, cholesterol, and triglyceride levels, and has anti-diabetic benefits.

The active compounds in ginger are divided into two groups: volatile essential oils which constitute gingerol and shagelol have been accounted for antimicrobial activity of ginger, and fragrant or harsh phenol compounds. [15]

Ginger extract, have been widely used in traditional medicine and human clinical trial without significant side effects. [16]

Till date there are no literatures available with respect to the use of ginger gel as a local drug delivery system in the treatment of periodontal diseases.

Hence, the present study was designed to assess the clinical effects and microbial analysis of Ginger gel as a local drug delivery (LDD) in the treatment of patients with Chronic Generalized Periodontitis.

## MATERIALS AND METHODS

The present randomized, parallel arm, placebo controlled clinical trial was conducted among 30 patients visiting the Department of Periodontology, to evaluate the effect of ginger gel as a local drug with and without scaling and root planing (SRP) on the clinical parameters and the subgingival microbiological analysis in chronic periodontitis patients. The purpose of this study was explained to the patient verbally in a language that he or she could understand, and informed consent was obtained from the patient. The ethical clearance for the study was obtained from the institutional ethical committee.

The subjects included in the study were based on the criteria that the patients were aged between 25 to 65 years of both genders with a minimum of 20 natural teeth and diagnosed with Chronic Generalized Periodontitis, characterized by the presence of > 1 periodontal site with a probing pocket depth of  $\geq$  5mm showing bleeding upon probing, and patients with history of allergy to herbal medications, patients on any medication taken within the last 6 months which may alter the periodontal status and patients who have undergone periodontal treatment within a period of 1 year were excluded from the study.

All the subjects participating in the study received single sitting of full mouth scaling with ultra-sonic scaler and root planing with Gracey curettes. Plaque samples were collected from each subject and were divided into 2 groups as follows:

Group I - Scaling and Root planing alone,

Group II - Scaling and Root planing along with Ginger gel as LDD

## **Clinical Parameters Recorded**

- 1. Gingival index (GI) (Loe H and Silness 1963).
- 2. Gingival bleeding index (GBI) (Ainamo and Bay 1975)
- 3. Probing pocket depth (PPD) measured using graduated Williams periodontal probe from the crest of gingival margin to base of the pocket.
- 4. Periodontal index (PI) (Russell -1956).

## **Preparation of Ginger Gel**

The preparation of the ginger gel was carried out at St. John's College of pharmacy, Bangalore, Karnataka, India.

## SAMPLE COLLECTION

The subgingival plaque samples were collected from the deepest part of the periodontal pockets in both test and control sites at baseline and after 1 month to assess the total colony forming viable anaerobic count (CFUs) using sterile Gracey curette, placed into the Eppendorf tubes and transported in fluid thioglycolate medium to be processed immediately.

## MICROBIOLOGICAL ANALYSIS

The subgingival plaque samples were inoculated onto the brucella blood infusion agar plates supplemented with hemin and vitamin K. Streaking was done to spread the inoculums across the plate. A sterile inoculation loop was used with repeated flaming time to maintain sterility. A gap of 5 seconds was given for the loop to cool down after each flaming before streaking. Plates inoculated at the bench and placed in anaerobic jars along with one AnaeroGas Pack Sachet and are incubated for at least 48 hours, and reincubated for another 2 to 4 days to allow slow-growing organisms to form colonies. The Anaerogas Pack 1.5L sachet (HIMEDIA, Ref no. LE002F 5NO) was used. It has a disposable oxygen absorbing and carbon dioxide-generating agents used in anaerobic jars for the preparation of anaerobic media, no catalyst is necessary as no gas pressure is generated. The presence of anaerobic gram-negative microorganisms were determined based on colony observations.

## **TREATMENT**

The treatment in each of the patient included full mouth scaling with ultra-sonic scaler and root planing with Gracey curettes, and the subgingival delivery of ginger gel was dispensed using a blunt syringe. The test sites were isolated with cotton rolls. The tip was inserted till the base of the pocket and the drug was injected slowly while drawing the tip upward till the

gingival margin; excess gel flowing out of the pocket was gently wiped using sterile cotton. Periodontal dressing was placed, and the patients were instructed not to rinse, eat, or drink for the next 20 minutes. Routine oral hygiene instructions were given. After 1 week, removal of periodontal pack and assessment for any adverse effects was done. At 1 month re-evaluation, the periodontal parameters were measured and subgingival plaque samples were collected to assess the microbiological parameter in the form of CFUs. Clinical procedural steps (Figure 1-8)

#### STATISTICAL ANALYSIS

The data collected was analysed using the Statistical Package for Social Science (SPSS version 10.5). Descriptive analysis of all the explanatory and outcome parameters was done using frequency and proportions for categorical variables, whereas in Mean & SD for continuous variables. Student Paired t -Test was used to compare the mean GI, Russels & PPD levels between baseline and 1 month. Mann Whitney U test was used to compare the mean CFUs between the groups at baseline and 1 month. Wilcoxon Signed Rank Test was used to compare the mean CFU levels of different organisms between the groups at baseline and 1 month in each study group. The level of significance was set at P<0.05.

#### RESULTS

The present study was done to assess the clinical and microbial effect of Ginger gel as a local drug delivery (LDD) in 30 patients with Chronic Generalized Periodontitis. A total of 30 subjects were included in the study, where 15 patients underwent ultrasonic scaling and root planing, 15 patients underwent scaling, root planing and ginger gel placement. All subjects completed the study. No adverse effects, such as discomfort, burning sensation, or pain were reported by any of the subjects.

Table 1 gives the age and gender distribution of the study subjects. The study subjects were in the age range of 25-65 years (n=30) with a minimum age of 21 years and the maximum age of 61 years. The gender distribution of the study subjects were males (n=14) making up 45% while females (n=16) making up 55% (Graph 1,2).

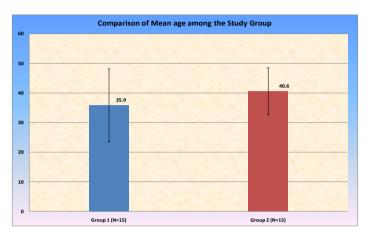
Table 1: Age And Gender Distribution Among Study Subjects.

	Ger	Total	
	Male	Female	Total
GROUP 1	5	10	15
GROUP I	33.3%	66.7%	100%

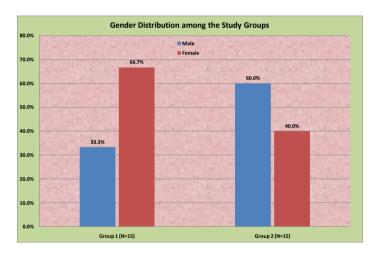
GROUP 2	9	6	15	
UKUUF 2	60%	40%	100%	

		N	Mean	SD	Min.	Max.
AGE	GROUP 1	15	35.9	12.253	21	61
AGE	GROUP 2	15	40.6	7.944	29	54

<sup>\*</sup>Student 't'test



**GRAPH 1.** 



**GRAPH 2.** 

## **GINGIVAL INDEX (GI)**

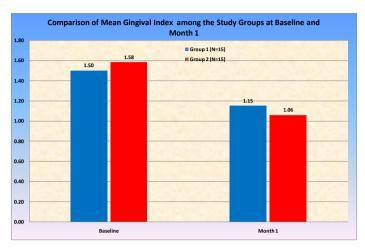
Table 2 gives the mean gingival index scores at baseline and 1 month time interval compared using Student Paired t Test. There was a statistically significant reduction in the GI score from baseline  $(1.50 \pm 0.452)$  to the 1 month recall visit  $(1.06 \pm 0.247)$ . (Graph 3).

Table 2: Comparison of Mean Gingival Index Scores Between The Groups.

Group	Visit	N	Mean	SD	<b>Mean Diff</b>	SE of Diff	P value*
Group 1	Baseline	15 1.50 0.43	0.452	0.25	0.152	0.030	
Group 1	Month 1	15	1.15	0.378	0.55	0.35 0.152	0.030

Group 2	Baseline	15	1.58	0.268	0.52	0.094	<0.001
Group 2	Month 1	15	1.06	0.247	0.32	0.094	

<sup>\*</sup>Paired 't' test



**GRAPH 3.** 

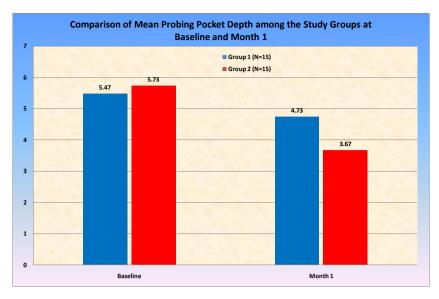
## PROBING POCKET DEPTH (PPD)

Table 3 gives the comparison of mean probing pocket depth (in mm) between the study groups at the recall interval was done using Student Paired t Test. At baseline, the mean probing depth was  $5.47 \pm 0.834$ , and 1 month was  $3.67 \pm 0.724$ , and there was a significant reduction in probing depths at the follow up visit, that was statistically significant (P < 0.001) (Graph 4).

Table 3: Comparison of Mean Probing Pocket Depth Between The Groups.

Group	Visit	N	Mean	SD	<b>Mean Diff</b>	SE of Diff	P value*	
Group 1	Baseline	15	5.47	0.834	0.73	0.264	0.010	
Group 1	Month 1	15	4.73	0.594	0.73	0.204	0.010	
Group 2	Baseline	15	5.73	0.704	2.07	0.261	<0.001	
Group 2	Month 1	15	3.67	0.724	2.07	0.201	<0.001	

<sup>\*</sup>Paired 't' test



**GRAPH 4.** 

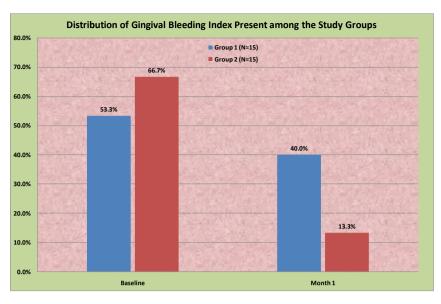
## **GINGIVAL BLEEDING INDEX**

Table 4 gives the comparison of mean gingival bleeding scores between the study groups was done using Chi Square Test. At baseline, the mean gingival bleeding score was 53.3% and 66.7% in Group 1 & 2 respectively, and 1 month the score was 40% and 13.3%, there was a statistically significant reduction in the gingival bleeding scores in Group 2 subjects at the follow up visit. (Graph 5).

Table 4: Comparison of Mean Gingival Bleeding Index Between The Groups.

Croup	Gingival bleeding index	Vi	sit	Total	P value*	
Group	Gingivai bleeding index	Baseline   Month		Total	1 value	
	Planding Absent	7	9	16		
Group 1	Bleeding Absent	46.7%	60.0%	53.3%	0.358	
	Planding Progent	8	6	14	0.558	
	Bleeding Present	53.3%	40.0%	46.7%		
	Bleeding Absent	5	13	18	0.004	
Group 2	Bleeding Absent	33.3%	86.7%	60.0%		
	Planding Progent	10	2	12	0.004	
	Bleeding Present	66.7%	13.3%	40.0%		

<sup>\*</sup>Chi Square Test



**GRAPH 5.** 

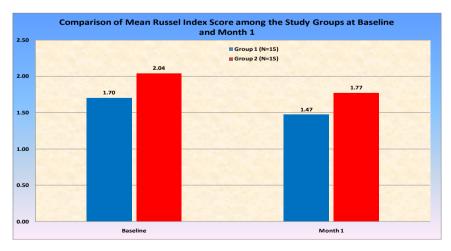
## **RUSSEL INDEX**

Table 5 gives the comparison of mean Russel index scores at baseline and 1 month time interval was done using Student Paired t Test. At baseline, the mean russel score was  $2.04 \pm 0.708$ , and 1 month was  $1.77 \pm 0.720$ , and there was no significant differences in the scores between the groups (Graph 6).

Table 5: Comparison of Mean Russel Index Scores Between The Groups.

Group	Visit	N	Mean	SD	<b>Mean Diff</b>	SE of Diff	P value*	
Group 1	Baseline	15	1.70	1.077	0.23	0.388	0.564	
	Month 1	15	1.47	1.048	0.23	0.366		
Group 2	Baseline	15	2.04	0.708	0.27	0.261	0.303	
	Month 1	15	1.77	0.720	0.27	0.201	0.303	

<sup>\*</sup>Paired 't' test



**GRAPH 6.** 

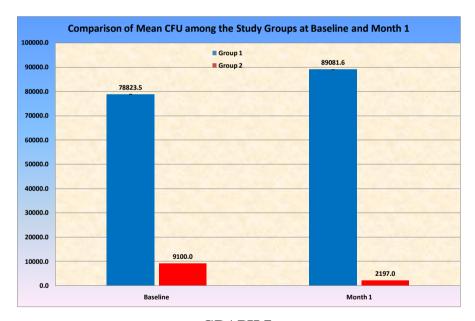
## MICROBIOLOGICAL FINDINGS

Intergroup comparison of mean Colony Forming Units (CFUs) of different microorganisms was done using Wilcoxin Sign Rank test. The significantly found microorganisms were Bacteriods, Fusobacterium Nucleatum, Micromonas Micros, Porphyromonas Gingivalis, Prevotella Intermedia, Prevotella Melanigenica. The intergroup comparison of the group 2 (ginger gel) and group 1 showed a reduction in microbiological parameters in the form of CFUs, however there was a statistically significant reduction in group 2 at 1 month recall period when compared with group 1. (Table 6,7 & Graph 7,8)

Table 6: Comparison of Mean Cfus (Cfu/MI) Between Groups.

Group	Visit	N	Mean	SD	P value*
Croup 1	Baseline	34	78823.53	38750.539	<0.001
Group 1	Month 1	30	9100.00	17742.118	<0.001
Crown 2	Baseline	38	89081.58	158477.124	<0.001
Group 2	Month 1	30	2197.00	3573.301	<0.001

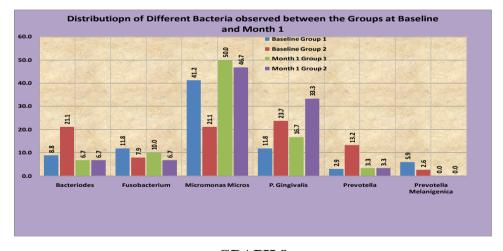
<sup>\*</sup>Wilcoxin Sign Rank test



GRAPH 7.

Table 7: Comparison of Mean Cfus of Different Organisms Between Groups At Baseline and 1 Month.

Visit	Group	Type of Microorganisam	N	Mean	SD	Median	Min.	Max.
		Bacteriodes	3	100000.00	0.000	100000.00	100000	100000
		Fusobacterium	4	77500.00	45000.000	100000.00	10000	100000
	Group 1	Micromonas Micros	14	74285.71	42192.651	100000.00	10000	100000
	Group 1	P. Gingivalis	4	77500.00	45000.000	100000.00	10000	100000
		Prevotella	1	100000.00	•	100000.00	100000	100000
Baseline		Prevotella Melanigenica	2	55000.00	63639.610	55000.00	10000	100000
Daseillie		Bacteriodes	8	62762.50	51393.383	100000.00	100	100000
		Fusobacterium	3	100000.00	.000	100000.00	100000	100000
	Group 2	Micromonas Micros	8	43750.00	46579.425	10000.00	10000	100000
	Gloup 2	P. Gingivalis	9	58000.00	49907.414	100000.00	1000	100000
		Prevotella	5	62200.00	51857.497	100000.00	1000	100000
		Prevotella Melanigenica	1	100000.00	•	100000.00	100000	100000
		Bacteriodes	2	1000.00	.000	1000.00	1000	1000
		Fusobacterium	3	7000.00	5196.152	10000.00	1000	10000
	Group 1	Micromonas Micros	15	13600.00	24245.176	10000.00	1000	100000
	Group 1	P. Gingivalis	5	4600.00	4929.503	1000.00	1000	10000
		Prevotella	1	1000.00	•	1000.00	1000	1000
Month 1		Prevotella Melanigenica	1	10000.00	•	10000.00	10000	10000
Monui		Bacteriodes	2	1000.00	.000	1000.00	1000	1000
		Fusobacterium	2	1000.00	.000	1000.00	1000	1000
	Group 2	Micromonas Micros	14	3378.57	4360.871	1000.00	100	10000
	Group 2	P. Gingivalis	10	1441.00	3041.843	550.00	10	10000
		Prevotella	1	100.00	•	100.00	100	100
		PrevotellaMelanigenica	1	100.00	•	100.00	100	100



**GRAPH 8** 

## **DISCUSSION**

Periodontitis is a chronic inflammatory disease of the tooth-supporting tissues, primarily caused by the accumulation of complex polymicrobial dental plaque resulting in progressive

destruction of the supporting structures. Traditional periodontal therapy includes mechanical debridement to disrupt the subgingival flora and to provide smooth, clean, and biologically compatible root surfaces. Unfortunately, in some instances, the complex anatomy of the root and the contours of the lesion may hinder the treatment and prevent sufficient reduction of the bacterial load to make the tooth surface biologically acceptable. [17]

Effective treatment of periodontitis requires the adequate removal of subgingival plaque by scaling and root planing. As the pockets deepen, plaque control measures become less effective. Retention of plaque in inaccessible sites can be a nidus for re-infection, which may allow the return of microflora and recurrence of the disease.<sup>[18]</sup>

Long-term adjunctive use of systemic and topical chemotherapeutic agents when used with scaling and root planing for the treatment of periodontitis is contraindicated because of adverse effects and development of bacterial resistance. [19]

Topical administration of antibacterial agents in various forms such as mouthwashes, dentifrices, and gels can be used effectively in controlling supragingival plaque. Irrigation devices deliver agents into deep pockets but clinically may not be effective in halting the progression of periodontal disease. [20] Local drug delivery can provide 100-fold higher therapeutic doses of the agent in subgingival areas than systemic therapy. [19]

Ginger (Zingiber officinale) is a medicinal plant that has been used traditionally in the Chinese and Ayurverdic herbal medicines as an anti-inflammatory agent that can reduce prostaglandin synthesis and exert inhibitory effects against periodontal bacterial growth of anaerobic Gram-negative bacteria including Prevotella intermedia, Porphyromonas endodontalis and Porphyromonas gingivalis.<sup>[5]</sup>

Ginger is a natural food source with antimicrobial activities, that is a widely grown food spices and medicinal crops with no toxicity, and considered as 'generally recognized as safe' (GRAS) by the Food and Drug Administration (FDA) of the United States. The pungent oil components of ginger harbor a series of pharmacological activities and possess antioxidant, antibacterial and antifungal, antimicrobial and anti-diabetic properties.<sup>[4]</sup>

A meta-analysis assessing the effect of the LDD system of chlorhexidine (CHX) as an adjunct to SRP in chronic periodontitis showed significant reduction of probing depth but no significant gain in the attachment level. Similarly meta-analysis of local delivery of tetracycline, metronidazole, and minocycline as an adjunct to conventional mechanical therapy suggested improved clinical outcomes. However considerations regarding the adverse effects of widespread use of antibiotics should be taken into account when choosing a therapeutic strategy for chronic periodontitis.<sup>[21]</sup>

In the present randomized, clinical and microbiological trial there was a significant reduction in all the clinical and microbial parameters such as gingival index, gingival bleeding index, pocket probing depth and microbial colony forming units with the use of Ginger gel as a local drug delivery (LDD) in patients with Chronic Generalized Periodontitis.

The improvement in clinical parameters can be due to antiinflammatory and antioxidant properties of the ginger because of allicin that inhibited the production of pro-inflammatory cytokine messengers in a study of inflammatory bowel disease, apparently by inactivating the pro-inflammatory factor NFkB via its IkB inhibitor. By virtue of sulphur-based antioxidants found in ginger, NFkB was maintained in its inactive state, thus preventing synthesis of excess cyclooxygenase (COX)/lipooxygenase (LOX).<sup>[22]</sup>

Study done by Mahyari et al (2016) studied and compared the efficacy of herbal mouthwash containing hydroalcoholic extracts of Z. officinale, Rosmarinus officinalis, and Calendula officinalis (5% v/w) with chlorhexidine. reported significant improvement in all the clinical parameters such as gingival index, gingival bleeding index and plaque index scores from baseline to the end of trial in both polyherbal and chlorhexidine mouthwash group. Moreover, the efficacy of polyherbal mouthwash was comparable to that of chlorhexidine mouthwash. <sup>[5]</sup> This reduction in the gingival and gingival bleeding scores can be attributed to the anti-inflammatory effect of ginger as it inhibits the synthesis of inflammatory mediators such as prostaglandins by acting on enzymes cyclooxygenase-1 and cyclooxygenase-2. <sup>[22]</sup> It also inhibits 5-lipoxygenase enzyme which is involved in the biosynthesis of leukotrienes.

Srivastava et al. (1992) studied effects of ginger on rheumatism or musculoskeletal disorders and suggested that the anti-inflammatory activity can be ascribed to prostaglandin and leukotriene suppression as well as dual inhibition of eicosanoid biosynthesis. [24]

A study done by Park et al., 2007, which signified that the ethanol and n-hexane ginger extracts exhibited antibacterial effect against three of the anaerobic Gramnegative bacteria, Porphyromonas endodontalis, Porphyromonas gingivalis and Prevotella intermedia, that are

responsible of periodontal diseases, they found that [10]-gingerol and [12]-gingerol effectively inhibited the growth pattern of these periodontal pathogens at a MIC range of 6-30  $\mu g/ml$ . [26]

Shirin Adel and Prakash (2010) observed the antioxidant activity of ginger root by analyzing its components such as vitamin C,  $\beta$  carotene, flavonoids, polyphenols, and tannins and concluded that maximum antioxidant activity is present in the alcoholic (methanolic) extracts. Hence, ginger can be considered as a functional food that protects against cell oxidation and delays the onset of chronic diseases.<sup>[27]</sup>

Although there are studies related to the use of ginger in the form of mouthwash and oral supplements on oral microorganisms in vitro and in gingivitis and diabetic patients, no literature exists till date on the in vivo effect of ginger gel as a local delivery agent on periodontopathic microorganisms and in the treatment of patients with chronic generalized periodontitis.

This study is the first of its kind, wherein ginger gel as a local drug delivery agent was evaluated for its effect on the clinical parameters and anaerobic microbial culture, it has resulted in significant reduction in clinical parameters and CFUs of periodontopathogens at the recall period. This indicates that ginger gel was effective in reducing the severity of periodontal inflammation and periodontitis by reducing the overall microbial count. However further studies can be conducted on larger samples and for a longer follow up duration to evaluate its effect on periodontal diseases.

## ETHICAL COMPLIANCE

- **1. Source of Funding:** This study was self-funded by the authors and did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.
- **2. Disclosure of Interest:** The authors declare that they have no conflict of interest.
- **3. Informed Consent:** Informed consent was obtained from each individual participant involved in this study.
- **4. Statement of Human Right:** This study was conducted in accordance with the 1964 Declaration of Helsinki and its subsequent amendments.
- 5. Statement of Animal Welfare: No animals were involved in the study



Figure 1: Pre-Operative Clinical Picture.



**Figure 2: Pre-Op Pocket Probing Depth** 



Figure 3: Gel Prepared From Ginger.



Figure 4: Delivery of Ginger Gel as LDD



Figure 5: Post-Op Pocket Probing Depth.



Figure 6: Streaking Of The Inoculated Plaque Samples.



Figure 7: Anaerobic Jar With Gas Pack.

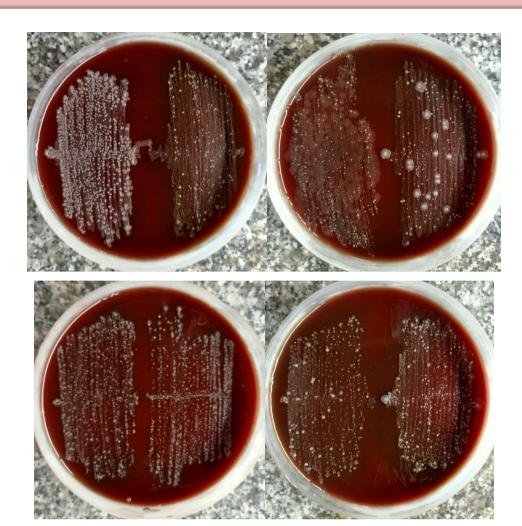


Figure 8: Microbial Colony Forming Units Observed.

## **CONCLUSION**

The present study demonstrated a statistically significant reduction in the clinical parameters from baseline to 1 month (P < 0.001) suggesting that the treatment was effective and showed significant improvement in reducing the inflammatory components due to its antiinflammatory property. Statistically significant reduction in the colony forming units of the periodontal pathogens was observed post therapy which can be attributed to the anti-bacterial property of ginger. Hence, within the limits of the present study it can be concluded that Ginger gel as LDD can be potentially useful in the treatment of periodontal disease.

## **ACKNOWLEDGEMENT**

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