JACM

ORIGINAL ARTICLE

Evaluation of Turmeric Chip Compared with Chlorhexidine Chip as a Local Drug Delivery Agent in the Treatment of Chronic Periodontitis: A Split Mouth Randomized Controlled Clinical Trial

Abhilasha Singh, MDS,[1] Raja Sridhar, MDS,[2] Ravi Shrihatti, MDS,[3] and Akash Mandloy, MDS[4]

Abstract

Aims and Objective: The aim of this study was to evaluate and compare the effect of chlorhexidine (CHX) chip and turmeric chip as a local drug delivery (LDD) agent in the treatment of patients with chronic periodontitis.

Materials and Methods: A total of 120 sites with pocket depths 5–8 mm were chosen as a split mouth design at 3 sites in the same patient. Selected sites were randomly divided into three groups to receive CHX chip in addition to scaling and root planing (SRP) in group A, turmeric chip in addition to SRP in group B, and SRP only in group C. Clinical parameters, that is, plaque index (PI), gingival index (GI), probing pocket depth (PPD), and relative attachment level (RAL) were recorded at baseline, 1 month, and 3 months interval.

Result: On applying statistical analysis, results revealed that there was a significant reduction in all the clinical parameters, that is, PI, GI, PPD, and gain in RAL from baseline to 1 month and 3 months in all the three groups. These results were found to be significantly high in the CHX group and turmeric group than in the SRP group. Also, the results in both the test groups were maintained till the end of the study periods, but SRP group showed a significant deterioration after 1 month as was seen by increase in PPD and decrease in RAL scores after 3 months in the SRP group.

Conclusion: Both the treatment modalities with the application of LDD as an adjunct to SRP proved to be equally beneficial in the treatment of chronic periodontitis.

Keywords: LDD, chlorhexidine, turmeric, chip, SRP, PPD, RAL

Introduction

PERIODONTAL DISEASE IS a complex multifactorial disease characterized by destruction of the supporting periodontal tissues including the connective tissue and alveolar bone.[1] Elevated proportions of certain subgingival microbial species have been associated with destructive periodontal disease and it is important to keep the pathogenic microflora of the pocket suppressed to maintain health of the periodontal tissues.[1] The primary objective of periodontal therapy is to reduce the microbial load, thereby tending to an improvement in the clinical parameters using nonsurgical and surgical therapies.[1] The most widely used nonsurgical approach has been scaling and root planing (SRP) that effectively decreases the microbial load, but recolonization of the same can occur as early as 60 days after SRP.[1] Also it fails to eliminate the pathogenic bacteria completely especially at the base of the periodontal pocket and the areas inaccessible to periodontal instruments.[2] Consequently, this has led to the adjunctive use of antimicrobials, assuming that chemicals would compensate for technical limitations, prevent early microbial recolonization, and provide a chance for clinical improvements.[3]

Surgical procedures have inherent disadvantages such as greater patient morbidity, marginal bone resorption, and compromised postsurgical aesthetics in the form of gingival recession and interproximal soft tissue cratering.[4]

[1]Delhi Hospital, Bahadurgarh, Haryana, India.
[2]Triveni Institute of Dental Sciences, Hospital and Research Centre, Bilaspur, Chhattisgarh, India.
[3]Maratha Mandal Dental College, Belgaum, Karnataka, India.
[4]Modern Dental College, Indore, India.

Further there are certain individuals in whom surgery is contraindicated such as medically compromised patients, uncooperative individuals, elderly patients, and persons with physical or mental handicaps.[5]

Earlier, a greater emphasis was laid on the microbiologic etiology of periodontal disease. The microbial ecology of human periodontitis suggests therapies with antimicrobial agents in addition to mechanical therapy. Goodson in 1979 first proposed the concept of controlled delivery in the treatment of periodontitis. It has been observed that the local route of drug delivery can attain 100-fold higher concentrations of an antimicrobial agent in subgingival sites than a systemic drug regimen.[6]

Various antimicrobial agents such as tetracycline, 10% doxycycline, 2% minocycline, metronidazole, and chlorhexidine (CHX) gluconate have been used and investigated as local drug delivery (LDD) in the treatment of periodontal disease.[7]

Although local agents are beneficial as adjunct in the treatment of periodontal diseases, some side-effects with their usage have been mentioned in the literature such as placement of tetracycline-containing fibers around 12 or more teeth resulting in oral candidiasis in few patients.[7] Also because of its high cost factor, usage of these LDD agents in routine clinical practice may be restricted. To overcome these disadvantages, research is being conducted on the use of natural products as LDD agents. With the growing interest and increasing knowledge about the medicinal value of natural products, various formulations have been made commercially available. Herbal formulations such as turmeric, aloe vera, neem, tulsi, cocoa husk, and pomegranate are tested widely these days.[8] These products not only have long era of use and better patient tolerance but also act as a renewable source and are cost-effective.

Turmeric (haldi) is a rhizome of Curcuma longa. It has proven properties such as anti-inflammatory, antioxidant, antimicrobial, hepatoprotective, immunostimulant, antiseptic, antimutagenic, and also accelerates wound healing.[9] This natural product is easily available, cost-effective, and can be used as a LDD agent to treat patients with chronic periodontitis effectively.[10] Literature search reveals very few studies performed using turmeric as a LDD agent. Therefore, it adds up to the ongoing research related to herbal products in LDD.

The agent that has shown the most positive antibacterial results to date with pronounced antiseptic properties is CHX,[7] and is considered as "gold standard" in periodontics. Hence the aim of this study was to evaluate the efficacy of turmeric as an LDD agent in the form of chip as compared with only SRP and to compare the efficacy of this herbal chip as an LDD agent with the CHX chip in the treatment of chronic periodontitis.

Materials and Methods

Materials

CHX chip used in this study (*PerioCol-CG*) is a small, orange-brown rectangular chip manufactured by Eucare Pharmaceuticals, Chennai, India. The size of the chip is 4×5 mm with a thickness of 0.25–0.32 mm and weighing 10 mg. Each chip contains 2.5 mg of CHX in a biodegradable matrix of fibrilar collagen.

The test product turmeric chip used in the study measures 4×5 mm with a thickness of 0.4 mm and weighing 10 mg in a biodegradable 1000 mg hydroxy propyl cellulose matrix with 5% turmeric by weight.

Method

A total of 120 sites in 40 patients aged 30–50 years (each comprising 3 test sites with probing pocket depth [PPD] 5–8 mm) with mild-to-moderate chronic localized periodontitis (when <30% of the sites assessed in the mouth demonstrate attachment loss and bone loss) were selected from the outpatient department of periodontics, Modern Dental College, Indore. The study protocol was approved by the institutional ethical committee. The patients were duly informed about the study and written consent was obtained for their participation in the study. PPD was measured using a graduated probe (UNC-15) by inserting the probe into the gingival sulcus and readings were taken from the gingival margin till the base of the pocket. Alginate impressions were made to prepare customized acrylic stents that would act as a reference guide for recording PPD and relative attachment levels (RALs) at the experimental sites (as explained in flowchart).

Exclusion criteria included pregnant and lactating females, patients with a history of any kind of periodontal therapy within past 6 months, medically compromised patients, sites neighboring recent extraction sites, teeth showing endoperio lesions, restorations and other plaque retentive factors, smokers and tobacco chewers, and, importantly, subjects not compliant with oral hygiene procedures.

Study design

A flowchart indicating chronological order of procedures performed during the study period is shown in Figure 1. For each patient, three contralateral target sites were selected using simple randomization procedure and assigned to one of the three treatment modalities as follows:

Group A: Sites to be treated with CHX gluconate chip (Periocol-CG) in addition to SRP (Fig. 2a, c, e, i).
Group B: Sites to be treated with turmeric chip in addition to SRP (Fig 2b, d, f, j).
Group C: Sites to be treated with only SRP (Fig 2g, h, k).

Randomization was done using predetermined computer-generated randomization scheme to receive balanced random allocation of patients. Each randomization number was randomly assigned to the three treatments—CHX chip, turmeric chip, and SRP alone. One set of envelope was provided to the investigator containing individual randomization codes. The randomized allocation sequence was generated by a statistician.

Statistical analysis

Mean and standard deviations of plaque index (PI), gingival index (GI), PPD, and RAL were calculated for each group. Mauchly's test of sphericity was applied to check whether there was a violation of assumption of sphericity. All significant values in Mauchly's test were subjected to Greenhouse–Geisser corrections and p-values were
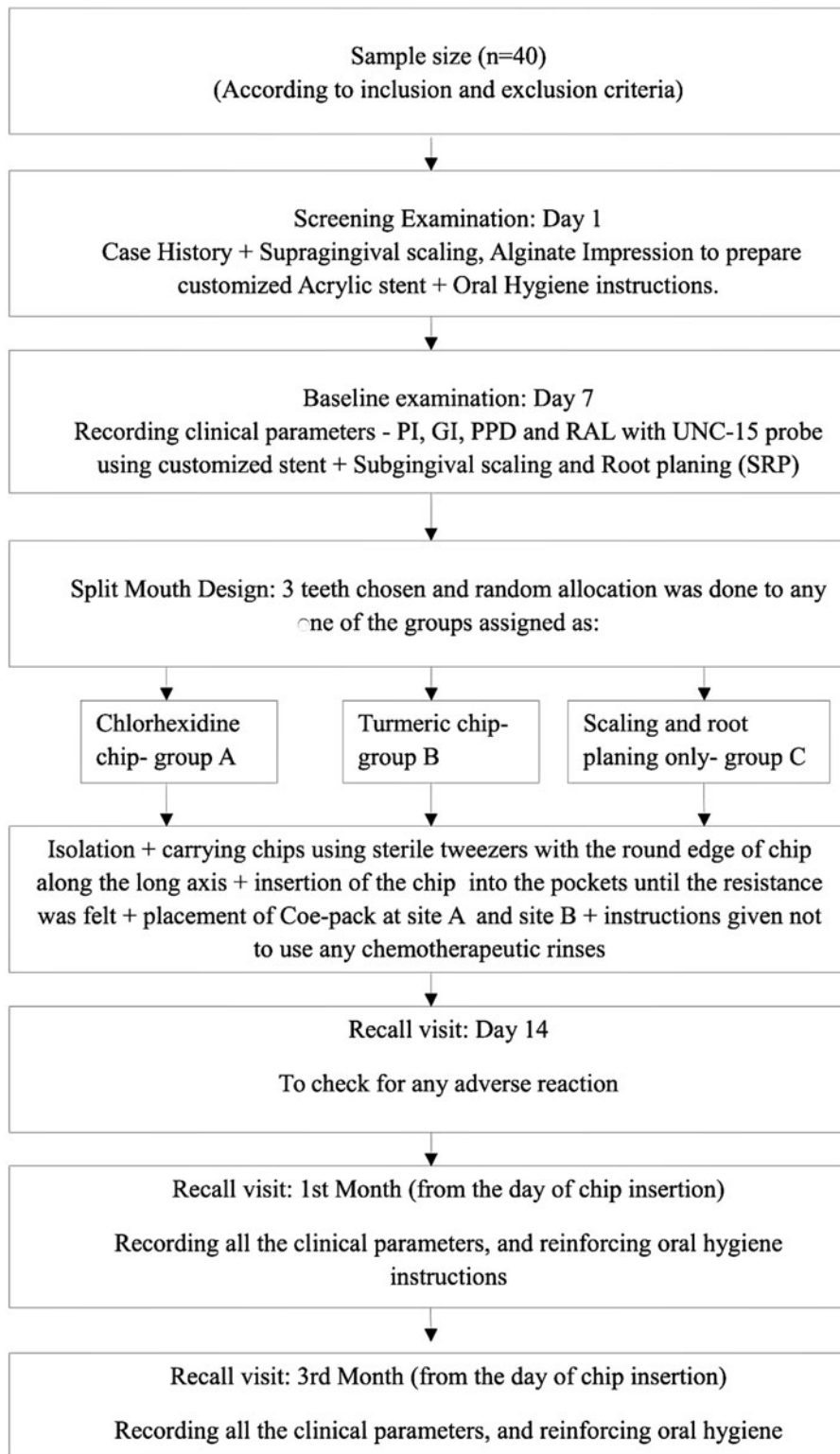
FIG. 1. Study design.

calculated. Being a split mouth design, intragroup comparisons were analyzed by repeated measures analysis of variance (ANOVA). Bonferroni test was used for *post hoc* comparisons after the repeated measures ANOVA. Analysis of covariance (ANCOVA) test was applied for intergroup comparisons at 1 month and 3 months adjusting for the baseline score variations as the covariate (adjusted analysis). Both adjusted and unadjusted values are presented in the results. Probability value of <0.001 was considered as statistically significant for all comparisons.

Results

A total of 40 subjects were recruited for this study. The mean age was found to be 34 years. Of these 40 subjects, 18 (45%) were females and 22 (55%) were males.
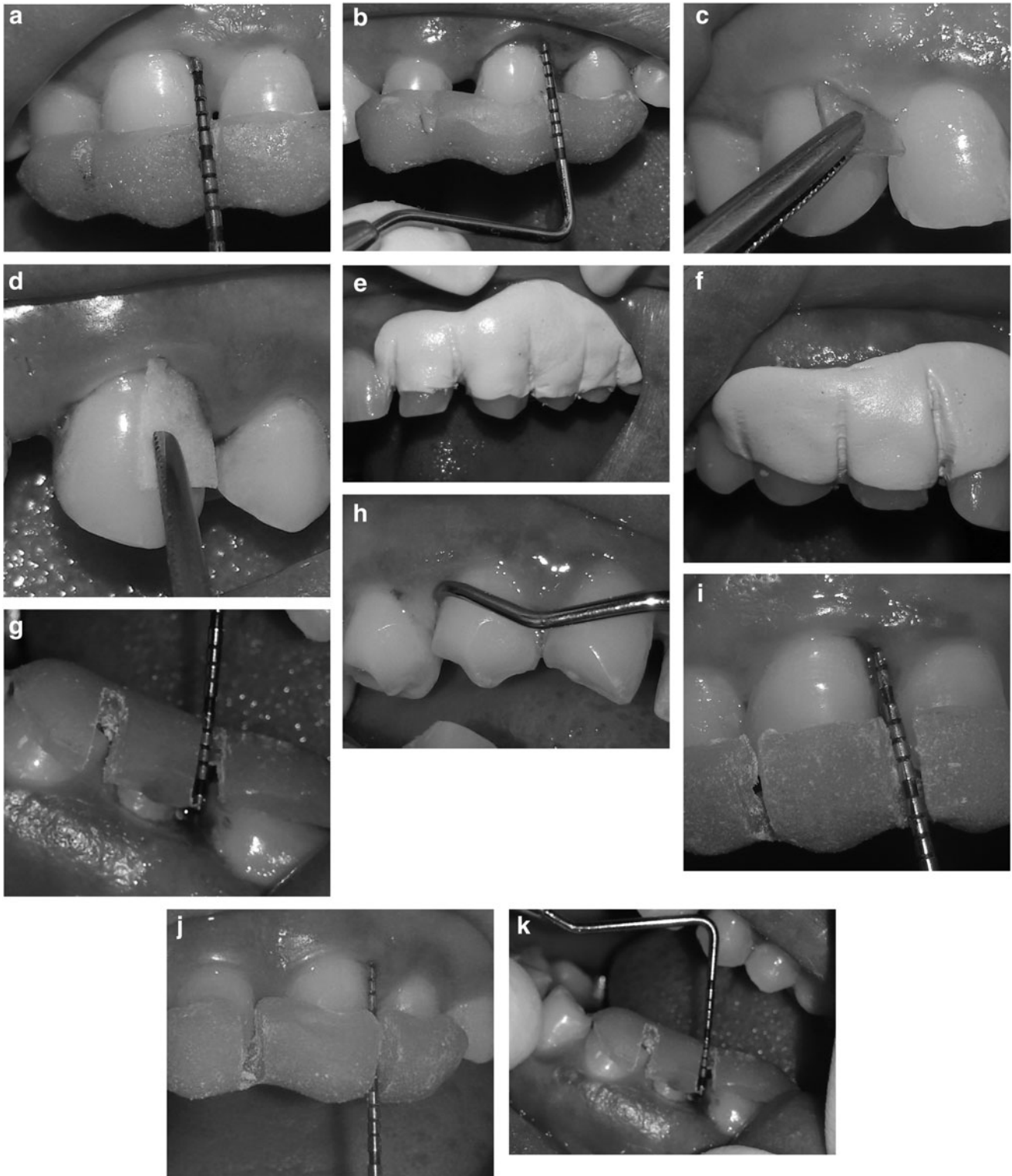
FIG. 2. **(a)** PPD = 4 mm, tooth No. 13(mesial). **(b)** PPD = 6 mm, tooth No. 23(distal). **(c)** Placement of CHX chip with tooth No. 13. **(d)** Placement of turmeric chip with tooth No. 23. **(e)** Placement of periodontal pack at the site of CHX chip insertion. **(f)** Placement of periodontal pack at the site of turmeric chip insertion. **(g)** PPD = 4 mm, tooth No. 35(distal). **(h)** Scaling and root planning with tooth No. 35. **(i)** Three months recall: PPD = 3 mm with tooth No. 13. **(j)** Three months recall: PPD = 2 mm with tooth No. 23. **(k)** Three months recall: PPD = 4 mm with tooth No. 35. CHX, chlorhexidine; PPD, probing pocket depth.

TABLE 1. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PLAQUE INDEX AND GINGIVAL INDEX FOR GROUP A (CHX GROUP)

Aspect	PI			GI		
	0	1 m	3 m	0	1 m	3 m
Mean	1.58	0.81	0.76	1.71	1.17	1.14
SD	0.79	0.45	0.56	0.54	0.46	0.45
Repeated measures ANOVA	Greenhouse–Geisser corrected p-value: <0.001, significant			Greenhouse–Geisser corrected p-value: <0.001, significant		
0 vs. 1 m	<0.001, significant			<0.001, significant		
0 vs. 3 m	<0.001, significant			<0.001, significant		
1 m vs. 3 m	>0.05, not significant			>0.05, not significant		

ANOVA and *post hoc* Bonferroni test.
ANOVA, analysis of variance; GI, gingival index; PI, plaque index; SD, standard deviation.

Intragroup changes at baseline, 1 month and 3 months for mean PI and GI for group A (CHX group) are shown in Table 1 and Intragroup changes at baseline, 1 and 3 months for mean PPD and RAL for group A (CHX group) are shown in Table 2.

Intragroup changes at baseline, 1 month and 3 months for mean PI and GI for group B (Turmeric group) are shown in Table 3 and Intragroup changes at baseline, 1 month and 3 months for mean PPD and RAL for group B (Turmeric group) are shown in Table 4.

The mean PI and GI and the mean PPD and RAL displayed a statistically significant reduction in scores at 1 month and 3 months compared to baseline interval for both the groups ($p < 0.001$).

Intragroup changes at baseline, 1 month, and 3 months for mean PI and GI for group C (SRP group) are shown in Table 5. The mean PI and GI displayed a statistically significant reduction in scores when compared from baseline to 1 month and 3 months ($p < 0.001$).

Intragroup changes at baseline, 1 month, and 3 months for mean PPD and RAL for group C (SRP group) are shown in Table 6. The mean PPD and RAL displayed a reduction in scores at 1 month and 3 months interval. The values were statistically significant ($p < 0.001$) when compared from baseline to 1 month for both PPD and RAL. There was

TABLE 2. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PROBING POCKET DEPTH AND RELATIVE ATTACHMENT LEVEL FOR GROUP A (CHX GROUP)

Aspect	PPD			RAL		
	0	1 m	3 m	0	1 m	3 m
Mean	4.04	2.79	2.84	4.11	2.83	2.84
SD	0.55	0.50	0.42	0.53	0.51	0.43
Repeated measures ANOVA	Greenhouse–Geisser corrected p-value: <0.001, significant			Greenhouse–Geisser corrected p-value: <0.001, significant		
0 vs. 1 m	<0.001, significant			<0.001, significant		
0 vs. 3 m	<0.001, significant			<0.001, significant		
1 m vs. 3 m	>0.05, not significant			>0.05, not significant		

ANOVA and *post hoc* Bonferroni test.
PPD, probing pocket depth; RAL, relative attachment level.

TABLE 3. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PLAQUE INDEX AND GINGIVAL INDEX FOR GROUP B (TURMERIC GROUP)

Aspect	PI			GI		
Months	0	1	3	0	1	3
Mean	1.61	0.85	0.72	1.66	1.14	1.17
SD	0.81	0.48	0.47	0.53	0.54	0.44
Repeated measures ANOVA	Greenhouse–Geisser corrected p-value: <0.001, significant			Greenhouse–Geisser corrected p-value: <0.001, significant		
0 vs. 1 m	<0.001, significant			0.001, significant		
0 vs. 3 m	<0.001, significant			<0.001, significant		
1 m vs. 3 m	0.08, not significant			>0.05, not significant		

ANOVA and *post hoc* Bonferroni test.

statistically significant increase in PPD scores and RAL scores from 1 month to 3 months in the SRP group.

Intergroup changes at baseline, 1 month, and 3 months for mean PI and GI between group A (CHX), group B (turmeric), and group C (SRP) are shown in Figure 3.

Intergroup changes at baseline, 1 month, and 3 months for mean PPD and RAL between group A (CHX), group B (turmeric), and group C (SRP) are shown in Figure 4.

The PI, GI, PPD, and RAL scores for group A (CHX), group B (turmeric), and group C (SRP) were compared using ANOVA and ANCOVA to elicit any intergroup changes at baseline, 1 month, and 3 months interval. On applying ANOVA for overall comparison of three groups, there was no significant difference between the baseline scores when compared between the three groups.

After 1 month and 3 months interval, the PI and GI scores displayed no significant difference between the three groups. After 1 month interval and 3 months interval, the three groups significantly differed in PPD and RAL scores when adjusted for baseline values by ANCOVA. When further compared by *post hoc* Bonferroni tests, SRP group showed statistically significant ($p < 0.001$) higher PPD and RAL scores than the other two groups. There was no significant difference between CHX and turmeric groups.

Discussion

The present split mouth randomized controlled clinical trial evaluated two drug forms, that is, CHX and turmeric,

TABLE 4. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PROBING POCKET DEPTH AND RELATIVE ATTACHMENT LEVEL FOR GROUP B (TURMERIC GROUP)

Aspect	PPD			RAL		
Months	0	1	3	0	1	3
Mean	4.04	2.84	2.97	4.10	2.88	3.00
SD	0.49	0.35	0.42	0.50	0.37	0.45
Repeated measures ANOVA	Greenhouse–Geisser corrected p-value: <0.001, significant			Greenhouse–Geisser corrected p-value: <0.001, significant		
0 vs. 1 m	<0.001, significant			<0.001, significant		
0 vs. 3 m	<0.001, significant			<0.001, significant		
1 m vs. 3 m	0.11, not significant			0.13, not significant		

ANOVA and *post hoc* Bonferroni test.

TABLE 5. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PLAQUE INDEX AND GINGIVAL INDEX FOR GROUP C (SCALING AND ROOT PLANING GROUP)

Aspect	PI			GI		
Months	0	1	3	0	1	3
Mean	1.58	0.82	0.80	1.64	1.23	1.21
SD	0.78	0.53	0.56	0.58	0.54	0.42
Repeated measures ANOVA	Greenhouse–Geisser corrected p-value: <0.001, significant			Greenhouse–Geisser corrected p-value: <0.001, significant		
0 vs. 1 m	<0.001, significant			<0.012, significant		
0 vs. 3 m	<0.001, significant			<0.004, significant		
1 m vs. 3 m	>0.05, not significant			>0.05, not significant		

ANOVA and *post hoc* Bonferroni test.

TABLE 6. INTRAGROUP CHANGES AT BASELINE, 1 MONTH, AND 3 MONTHS FOR MEAN PROBING POCKET DEPTH AND RELATIVE ATTACHMENT LEVEL FOR GROUP C (SCALING AND ROOT PLANING GROUP)

Aspect	PPD			RAL		
Months	0	1	3	0	1	3
Mean	3.99	3.66	3.90	4.09	3.80	3.99
SD	0.54	0.61	0.64	0.56	0.67	0.66
Repeated measures ANOVA	Sphericity assumed, p-value: 0.001, significant			Sphericity assumed, p-value: 0.005, significant		
0 vs. 1 m	<0.001, significant			0.006, significant		
0 vs. 3 m	0.752, not significant			0.981, not significant		
1 m vs. 3 m	0.004, significant			0.048, significant		

ANOVA and *post hoc* Bonferroni test.

both in the form of chip as a LDD over SRP alone in chronic periodontitis patients for a period up to 3 months. Split mouth design eliminates interpatient systemic differences (e.g., age, systemic health, and gender) that are known to confound the effects of periodontal treatment (independent variable) on clinical parameters and bacterial counts (dependent variables).[11] Also, maintaining the same oral environment for the experimental and control sites provides similar bacterial load, salivary pH, hygiene maintenance,

etc. so that the effects of the drugs to be evaluated can be determined. Furthermore, to avoid ''carry across'' or ''spillover'' effects while comparing the effects of different drugs within the same dentition in a split mouth design, randomly selected sites were chosen from different quadrants. Hence only one tooth in each quadrant was chosen for the study.[12] To maintain drug uniformity in both the groups, the concentration of turmeric chip was formulated to be 5%, which was equal to 2.5 mg of CHX present in commercially available PerioCol CG chip.

Evaluation of SRP + LDD versus only SRP on the changes in PI and GI scores

The PI and GI scores were significantly reduced in all the three groups (CHX, turmeric, and SRP) when compared from baseline up to 3 months. This reinforces the fact about the importance of a thorough mechanical debridement that can reduce the inflammation of the soft tissues as well as bring about low plaque scores provided the patient maintains a strict oral hygiene regimen.

A significant reduction in the PI and GI scores were noted from baseline to 3 months in all the three groups. This states that all the three treatment modalities were efficient in reducing microbial load (PI) and the degree of inflammation (GI),[13] but none of the treatment modalities superseded the other modalities with respect to PI and GI. The results were similar to those by Pai et al.,[14] wherein the reduction in bleeding on probing (BOP) was similar in both the experimental and control groups tested. Kranti et al.,[12] Grover et al.,[15] Gill et al.,[16] Paolantonio,[17] and Dinca et al.[18] also observed a similar trend in the reduction of GI scores between test and control sites. The findings of this study are in accordance with those of Azmak et al., who found a mean reduction in bleeding index score at 1 month and 3 months for both the combination group and the SRP alone group when compared with the baseline; however, at all periods of observations, a statistically nonsignificant correlation was observed in the bleeding scores between combination groups.[19]

In a similar study by Gottumukkala et al., aimed at comparing CHX and curcumin (CU), the mean PI scores did not show any significant difference between both the test groups at any time interval.[20]
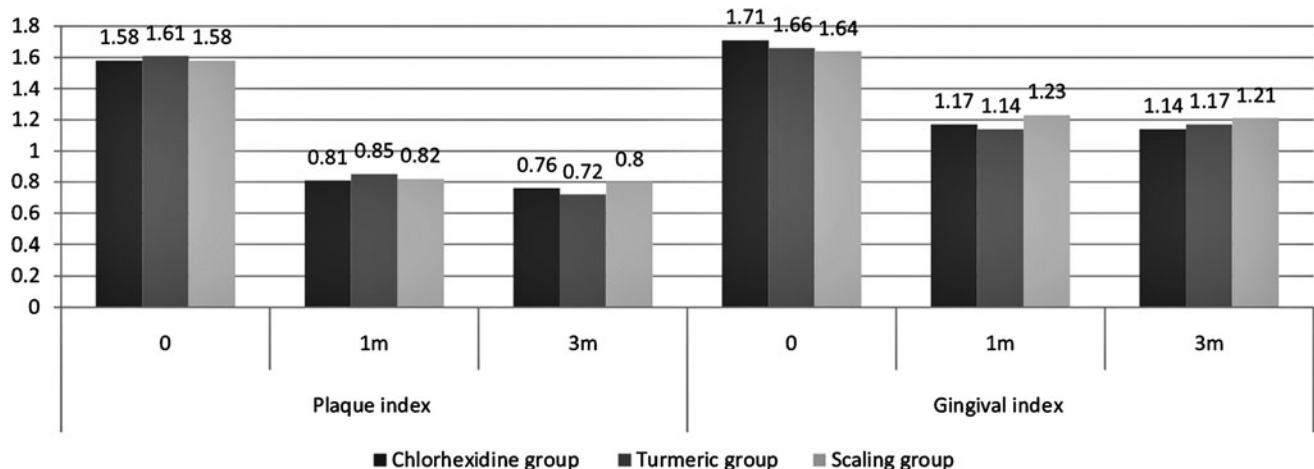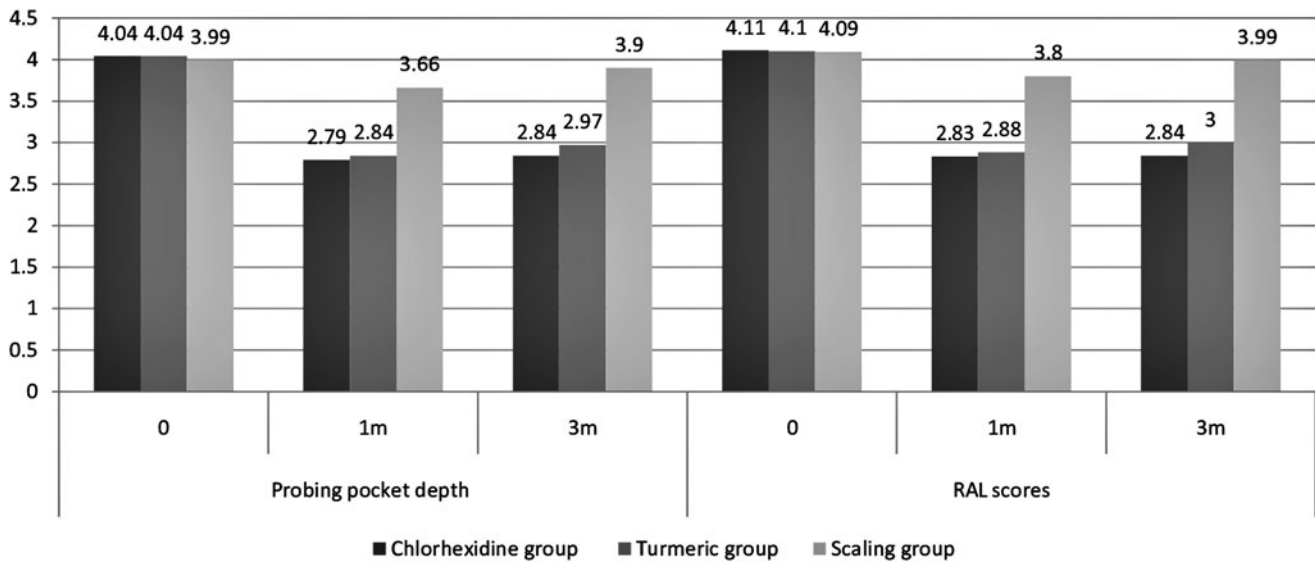

FIG. 3. Graph showing intragroup comparison of plaque index and gingival index from baseline to 3 months.

FIG. 4. Graph showing intergroup comparison of PPD and relative attachment level from baseline to 3 months.

Evaluation of SRP + LDD versus only SRP on the changes in PPD and RAL scores

Although SRP proved equally beneficial in reducing plaque and gingival inflammation when compared with CHX and turmeric, mechanical debridement alone may not prove very beneficial in sites displaying varying pocket depths and calls for additional therapy to either reduce or eliminate the pocket or cause gain in attachment levels. This was clearly evident in this study wherein the SRP group showed an improvement in the PPD and RAL scores only in the first 1 month after therapy, after which the probing depths and attachment loss significantly increased. It has also been demonstrated that the time spent on therapy, the number of sites that require instrumentation, and the experience of the clinician may influence the success of SRP.[21]

Also, some microbiota simply cannot be mechanically eradicated as bacterial invasion in cementum, radicular dentin, and the surrounding periodontal tissues has been reported.[21] Furthermore, recolonization of pockets can occur as early as 60 days after SRP.[1,2,22] A probable explanation for the deteriorating PPD and RAL scores after 1 month could be attributed to the latter.

The benefits of adjunct therapy such as delivering drugs directly into the pocket in addition to SRP were observed in this study, wherein a significant reduction in PPD and gain in clinical attachment level were evident over a 3-month period. Thus in addition to the benefits of SRP, the effects of the drugs in the pocket placed were evident as reducing PPD scores or gain in RAL values, which was seen maximum in the first 1 month after placement of the drug in the pocket and later remained stable without further deterioration. This was

TABLE 7. COMPARABLE MECHANISM OF ACTION OF CHLORHEXIDINE AND TURMERIC

Chlorhexidine	Turmeric
1. Antimicrobial action—damages the cell membrane of the microorganisms.	1. Curcumin is the active component—anti-inflammatory action.
Chip releases chlorhexidine and maintains drug concentrations in the GCF of <125 μg/mL for at least 7 days, which can inhibit 99% of the cultivable bacteria, on average.[7]	Chip releases 30.24 mg (70%) of turmeric at 24 h, 40.6 mg (94%) at 72 h, and 100% diffusion at 80 h,[10] which can inhibit 99% of the cultivable bacteria, on average.
2. Inhibit microbial proteases (gingipains)[25]	2. Inhibit COX-2, LOX, and iNOS
3. Decreases PGE2 levels.[27]	Decreases PGE2 and inhibits NFκβ pathway but also has an advantage that it selectively inhibits the synthesis of prostaglandins and thromboxane while not affecting the synthesis of prostacycline.[28]
4. Inhibits MMP-8, that is, inhibits collagenolytic activity.[37]	4. Inhibits IL-1β, IL-6, and TNF-α, that is, cytokine activity.[38]
5. Triggers neutrophil degranulation.[37]	5. Decreases neutrophil chemotaxis and phagocytosis[39]
6. Prolonged substantivity	6. Increases TGF-β1 that enhances wound healing and may be responsible in the gain in attachment after its use in periodontal therapy.[40]
7. Binds to intraoral hard and soft tissues.	7. Increases fibronectin and promotes migration of epithelial cells to wounded site, thus helping in re-epithelialization.[38]

GC, gingival crevicular fluid; iNOS, inducible Nitric Oxide synthase; TGF-β1, transforming growth factor.

similar to the previous studies by Pai et al.,[14] Gottumukkala et al.,[23] Nayyar et al.,[24] Mizrak et al.,[25] and Soskolne et al.[26]

The three treatment modalities performed in this study were compared using ANOVA and ANCOVA to unearth the differences if any in their efficacy to reduce PPD or cause a gain in RAL. Accordingly, it was observed that there were significant differences between the groups tested after 1 month and 3 months. Both CHX and turmeric led to significant improvement in reducing pocket depths and gain in attachment levels as compared with only SRP up to the 3-month recall period. Reduction in PPD scores was seen in similar studies conducted by various authors, that is, Kranti et al.,[12] Grover et al.,[15] Paolantonio[17] Azmak et al.,[19] Jaswal et al.,[21] Soskolne et al.,[26] Singh et al.,[27] Suhag et al.,[28] Behal et al.,[29] Varghese et al.,[30] Bhatia et al.,[31] and Rodrigues et al.[32] In this study, the change was significant in the first month but not significant from 1 month to 3 months. This was similar to the results obtained by Pai et al.,[14] Jeffcoat et al.,[33] Heasman et al.,[34] and Grisi et al.[35]

Evaluation of CHX versus turmeric as LDD on clinical parameters

This study also aimed to compare the efficacy of CHX and turmeric chip on the clinical parameters examined. Both the drugs were equally effective not only in reducing plaque and gingival inflammation but also in reducing pocket depths and causing significant gain in attachment level as evident by the significant reduction in all the scores obtained.

This is in accordance with the study by Anitha et al.[36] who found an equivalent effect of CU gel when compared with the CHX gel for the treatment of localized chronic periodontitis at 30 days when compared with the baseline. Similarly, another study by Jaswal et al.[21] who evaluated and compared the clinical effects of 2% whole turmeric gel versus 1% CHX gel as an adjunct to mechanotherapy in the treatment of chronic periodontitis found that turmeric gel showed a comparable improvement in all the clinical parameters as CHX gel. This may be explained on the basis of comparable mechanism of actions of CHX and turmeric as explained in Table 7.

Adverse events and complications

In this study, no adverse events were seen after placement of either of the two chips. Most of the studies that used CHX or turmeric reported no adverse effects, except for a few studies such as by Kaner et al.,[41] in which 50% of the patients reported discomfort, soreness of gingival tissues, and pain after the insertion of CHX chips. According to the authors, they noticed gingival swelling, redness, and gingival exudation, but the signs disappeared after 3–7 days without therapeutic intervention.[41]

Conclusion

This study reveals the excellent clinical properties of turmeric and CHX when evaluated over a period of 3 months. Although CHX is considered as a gold standard agent, this study shows an equally effective agent that can bring about a significant change in inflamed gingival tissues and reduction in PPD as well as gain in attachment. Added to this, turmeric displays very few side-effects as compared with the varying side-effects produced by CHX. Also, the cost of CHX chip is substantially expensive as compared with that of turmeric chip. Hence, turmeric chip may show a promising result as an LDD agent in the treatment of periodontitis.

Author Disclosure Statement

No competing financial interests exist.

References

1. Puri K, Dodwad V, Bhat K, Puri N. Effect of controlled release periochip on clinical and microbiological parameters in patients of chronic periodontitis. J Indian Soc Periodontol 2013;17:605–611.
2. Rams TE, Slots J. Local delivery of antimicrobial agents in the periodontal pocket. Periodontol 2000 1996;10:139–159.
3. Jeffcoat MK, Bray KS, Ciancio SG, et al. Adjunctive use of a subgingival controlled release chlorhexidine chip reduces probing depth and improves attachment level compared with scaling and root planing alone. J Periodontol 1998; 69:989–997.
4. Lindhe J, Westfelt E, Nyman S, et al. Long term effect of surgical/non-surgical treatment of periodontal disease. J Clin Periodontol 1984;11:448–458.
5. Paul GT, Hemalata M, Faizuddin M. Modified widman flap and nonsurgical therapy using chlorhexidine chip in the treatment of moderate to deep periodontal pockets: A comparative study. J Indian Soc Periodontol 2010;14:252–256.
6. Goodson J. Antimicrobial strategies for treatment of periodontal diseases. Periodontol 2000 1994;5:142–168.
7. Jolkovsky DL, Ciancio S. Chemotherapeutic agents. In: Newman MG, Takei HH, Klokkevold PR, Carranza KA, eds. Carranza's Clinical Periodontology, 10th edition. Missouri Saunders: Elsevier, 2006:798–812.
8. Kukreja BJ, Dodwad V. Herbal mouthwashes: A gift of nature: Int J Pharm Biol Sci 2012;3:46–52.
9. Lawande SA. Therapeutic applications of turmeric (Curcuma Longa) in Dentistry: A promising future. J Pharm Biomed Sci 2013;27:586–591.
10. Kudva P, Tabasum ST, Gupta S. Comparative evaluation of the efficacy of turmeric and curcumin as a local drug delivery system: A clinicomicrobiological study. Gen Dent 2012;60:e283–e287.
11. Stabholtz A, Nicholas AA, Zimmerman GJ, et al. Clinical and antimicrobial effects of a single episode of subgingival irrigation with tetracycline HCl or chlorhexidine in deep periodontal pockets. J Clin Periodontol 1998;25: 794–800.
12. Kranti K, Seshan H, Zope S. Clinic evaluation of topical subgingival application of the biodegradable xanthan based 1.5% chlorhexidine gel for treatment of periodontal pockets. JoADR 2010;1:47–54.
13. Machtei EE, Hirsh I, Falah M, et al. Multiple applications of flurbiprofen and chlorhexidine chips in patients with

chronic periodontitis: A randomized, double blind, parallel, 2-arms clinical trial. J Clin Periodontol 2011;38:1037–1043.

14. Pai BSJ, Rajan SA, Srinivas M, et al. Comparison of efficacy of chlorhexidine varnish and chip in the treatment of chronic periodontitis. Contemp Clin Dent 2013;4:156–161.

15. Grover V, Kapoor A, Malhotra R, et al. To access the effectiveness of a chlorhexidine chip in the treatment of chronic periodontitis: A clinical and radiographic study. J Indian Soc Periodontol 2011;15:139–146.

16. Gill JS, Bharti V, Gupta H, Gill S. Non surgical management of chronic periodontitis with two local drug delivery agents- a comparative study. J Clin Exp Dent 2011;3:e424–e429.

17. Paolantonio M. Clinical, microbiological and biochemical effects of subgingival administration of a Xanthan-based Chlorhexidine gel in the treatment of periodontitis: A randomized multicenter trial. J Periodontol 2009;80:1479–1492.

18. Dinca A, Mesaros M, Rusu D, et al. Initial periodontal therapy using a new Xanthan-based Chlorhexidine Gel–Chlosite. Int Poster J Dent Oral Med 2005;7:e-283.

19. Azmak N, Atilla G, Luoto H, et al. The effect of subgingival controlled release delivery of chlorhexidine chip on clinical parameters and matrix metalloproteinase-8 levels in gingival crevicular fluid. J Periodontol 2002;73:608–615.

20. Gottumukkala S, Sudarshan S, Satyanarayana RM. Comparative evaluation of the efficacy of two controlled release devices: Chlorhexidine chips and indigenous curcumin based collagen as local drug delivery systems. Contemp Clin Dent 2014;5:175–181.

21. Jaswal R, Dhawan S, Grover V, Malhotra R. Comparative evaluation of single application of 2% whole turmeric gel versus 1% chlorhexidine gel in chronic periodontitis patients: A pilot study. J Indian Soc Periodontol 2014;18:575–579.

22. Bruce LP, William FA. American academy of Peridontology: Treatment of gingivitis and periodontitis (position paper). J Periodontol 1997;68:1246–1253.

23. Gottumukkala S, Koneru S, Mannem S, et al. Effectiveness of sub gingival irrigation of an indigenous 1% curcumin solution on clinical and microbiological parameters in chronic periodontitis patients: A pilot randomized clinical trial. Contemp Clin Dent 2013;4:186–191.

24. Nayyar N, Dodwad V, Arora K. Comparative evaluation of 1% curcumin solution and 0.2% Chlorhexidine Gluconate irrigation as an adjunct to scaling and Root planing in the management of chronic Periodontitis: A clinico-microbiology study. J pharm Biomed Sci 2012;14:1–7.

25. Mizrak T, Guncu GN, Caglayan F, et al. Effect of controlled-release chlorhexidine chip on clinical and microbiological parameters and prostaglandin E2 levels in gingival crevicular fluid. J Periodontol 2006;77:437–443.

26. Soskolne AW, Proskin HM, Stabholz A. Probing depth changes following 2 years of periodontal maintenance therapy including adjunctive controlled release of chlorhexidine. J Periodontol 2003;74:420–427.

27. Singh M, Shreehari AK, Garg PK, Singh S. Clinical efficacy of chlorhexidine chips and tetracycline fibers as an adjunct to non surgical periodontal therapy. European J Gen Dent 2014;3:34–39.

28. Suhag A, Dixit J, Dhan P. Role of curcumin as a subgingival irrigant: A pilot study. PERIO 2007;4:115–121.

29. Behal R, Mali AM, Gilda SS, Paradkar AR. Evaluation of local drug delivery system containing 2% whole turmeric gel used as an adjunct to scaling and root planing in chronic periodontitis: A clinical and microbiological study. J Indian Soc Periodontol 2011;15:35–38.

30. Varghese MK, Nagarathna DV, Scariya L. Curcumin and metronidazole in periodontal therapy. Int J Res Ayurveda Pharm 2014;5:680–684.

31. Bhatia M, Urolagin SS, Pentyala KB, et al. Novel therapeutic approach for the treatment of periodontitis by curcumin. J Clin Diagn Res 2014;8:ZC65–ZC69.

32. Rodrigues GF, Machion L, Casati MZ, et al. Clinical evaluation of the use of locally delivered chlorhexidine in periodontal maintenance therapy. J Periodontol 2007;78:624–628.

33. Jeffcoat MK, Palcanis KG, Weatherford TW, Reese M. Use of a biodegradable chlorhexidine chip in the treatment of adult periodontitis: Clinical and radiographic findings. J Periodontol 2000;71:256–262.

34. Heasman PA, Heasman L, Stacey F, Mc Cracken GI. Local delivery of chlorhexidine gluconate (Periochip) in periodontal maintenance patients. J Clin Periodontol 2001;28:90–95.

35. Grisi DC, Salvador SL, Figueiredo LC, et al. Effect of a controlled-release chlorhexidine chip on clinical and microbiological parameters of periodontal syndrome. J Clin Periodontol 2002;29:875–881.

36. Anitha V, Rajesh P, Shanmugam M, et al. Comparative evaluation of natural curcumin and synthetic chlorhexidine in the management of chronic periodontitis as a local drug delivery: A clinical and microbiological study. Indian J Dent Res 2015;26:53–56.

37. Gendron R, Grenier D, Sorsa T, Mayrand D. Inhibition of the activities of matrix metalloproteinases 2, 8 and 9 by chlorhexidine. Clin Diagn Lab Immunol 1996;6:437–439.

38. Sidhu GS, Singh AK. Enhancement of wound healing by curcumin in animals. Wound Repair Regen 1998 6:167–177.

39. Gopinath D, Ahmed MR, Gomathi K, et al. Dermal wound healing processes with curcumin incorporated collagen films. Biomaterials 2004;25:1911–1917.

40. Satoskar RR, Shah SJ, Shenoy SG. Evaluation of anti-inflammatory property of curcumin (diferuloyl methane in patients with postoperative inflammation. Int J Clin Pharmacol Ther Toxicol 1986;24:651–654.

41. Kaner D, Bernimoulin JP, Hopfenmuller W, et al. Controlled delivery chlorhexidine chip versus amoxicillin/metronidazole as adjunctive antimicrobial therapy for generalized aggressive periodontitis: A randomized controlled clinical trial. J Clin Periodontol 2007;34:880–891.

Address correspondence to:
Abhilasha Singh, MDS
Consultant at Delhi Hospital
391 First Floor
City Homes
Bahadurgarh
Haryana
India

E-mail: abhilashasingh05@gmail.com