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Curcumin and aloe vera chips in stage II Grade A periodontitis: A randomised controlled trial

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Abstract:
Periodontitis is a chronic inflammatory disease in which adjunctive local drug delivery may improve treatment outcomes. Therefore, it is of interest to evaluate the effectiveness of sub gingival curcumin and aloe vera chips used with scaling and root planing in 45 patients with Stage II Grade A periodontitis. All treatment groups showed significant improvements in clinical parameters, while the aloe vera chip produced greater early pocket depth reduction. The curcumin chip showed a significantly greater reduction in *Aggregatibacter actinomycetemcomitans* than scaling and root planing alone after 3 months. Thus, both curcumin and aloe vera chips provided beneficial adjunctive effects and may improve the management of Stage II Grade A periodontitis.

Keywords: Curcumin, aloe-vera chip, local drug delivery, periodontitis, scaling and root planing

Background:
Periodontitis is a chronic inflammatory disease associated with dysbiotic sub gingival microbiota that progressively destroys periodontal tissues and may lead to tooth loss if untreated **[1]**. Stage II Grade A periodontitis represents moderate disease characterized by 4–5 mm probing depths, coronal-third bone loss and low risk of rapid progression **[2]**. Mechanical debridement may not completely eliminate pathogens from deep periodontal pockets, supporting the use of local drug delivery systems for sustained site-specific antimicrobial and anti-inflammatory effects **[3, 4]**. Aloe vera exhibits anti-inflammatory, antioxidant, antibacterial and wound-healing properties, while curcumin shows antimicrobial, antioxidant and anti-inflammatory activities relevant to periodontal therapy **[5, 6]**. Therefore, it is of interest to compare the clinico-microbiological efficacy of curcumin and aloe vera chips as adjuncts to scaling and root planing in Stage II Grade A periodontitis over a 3-month follow-up period.

Materials and Methods:
Study design:
A randomised, controlled clinical trial was conducted in the outpatient Department of Periodontology. The study protocol received clearance from the Institutional Scientific Review Board and Ethics Committee. Written informed consent was obtained from.

Sample size calculation:
Using the formula for comparison of means between 2 independent groups: $n = 2(Z\alpha/2 + Z\beta)\ ^2\sigma^2/d^2$, with 2-sided significance level of $\alpha = 0.05$ ($Z\alpha/2 = 1.96$) and power of 80% ($Z\beta = 0.842$), a minimum of 15 patients per group was calculated. Accounting for a 10% anticipated dropout rate, minimum of 15 patients per group — and 45 patients in total across 3 groups were enrolled; all 45 patients completed the study with no dropouts.

Participant selection:
Inclusion criteria:
Patients aged 25–55 years of either sex; a diagnosis of Stage II Grade A periodontitis **[2]** with at least 1site per quadrant exhibiting probing depth of 4–5 mm following non-surgical therapy; absence of allergy to turmeric or aloe vera; and no use of medicated toothpaste, antibacterial mouthwash, antibiotics or anti-inflammatory medication in the preceding 3 months.

Exclusion criteria:
History of periodontal therapy within the preceding 6 months; medically compromised patients; sites adjacent to recent extraction sockets; current or previous use of tobacco in any form; pregnancy or lactation; and refusal to provide informed consent.

Randomisation and group allocation:
After baseline, microbiological assessment and supragingival and subgingival scaling and root planing, patients were randomly allocated to groups using a computer-generated random number sequence, in a 1:1:1 ratio: Group A (SRP alone, n=15); Group B (SRP + curcumin chip, n=15); and Group C (SRP + aloe vera chip, n=15). Allocation concealment was maintained using sealed, sequentially numbered, opaque envelopes opened only at the time of chip insertion.

Baseline recording:
Case history and full-mouth clinical periodontal examination were recorded at baseline. Clinical parameters were recorded using a UNC-15 probe using customized stent. Parameters recorded were: Plaque Index (PI) using the Silness and Löe index; Gingival Index (GI) using the Löe and Silness index; Probing Pocket Depth (PPD) in millimetres; and Relative Attachment Level (RAL) measured from a fixed reference point on the stent to base of the pocket. Oral hygiene instructions were reinforced.

Microbiological sampling:
Subgingival plaque samples were obtained from the site. Samples were transferred to the microbiology laboratory. Colony counts were determined for *Porphyromonas gingivalis, Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans*. Results were expressed as mean colony-forming units per millilitre (×10⁵ CFU/mL).

Interventions:
All patients in all 3 groups received full-mouth supragingival and subgingival scaling and root planing (SRP) using ultrasonic scalers and curettes. Patients in Groups B and C received SRP and subgingival chip administration. Chip insertion was performed using sterile tweezers. A periodontal dressing (Coe-Pak™) was placed over the site to retain the chip and maintain isolation for 48 hours.

Follow-up:
Patients were recalled at one month and three months from the date of chip administration and SRP. At each recall appointment, clinical parameters (PI, GI, PPD, RAL) were recorded using customised stents and subgingival plaque samples were obtained for microbiological analysis. Oral hygiene instructions were reinforced at all recall appointments.

Statistical analysis:
Statistical analysis was performed using IBM SPSS Statistics version 25. The normality of data distribution was assessed using Levene's test of homogeneity of variance. Intragroup changes in clinical and microbiological parameters from baseline to one month and three months were compared using the paired samples t-test. Intergroup comparisons at each time point were performed using one-way ANOVA with post-hoc Tukey's honestly significant difference (HSD) test for multiple pairwise comparisons. Results were expressed as mean and standard deviation. A p-value of <0.05 (two-tailed) was considered statistically significant for all analyses.

Results:
The three treatment groups were comparable at baseline with respect to age, sex distribution and clinical and microbiological parameters, with all baseline intergroup comparisons showing no statistically significant differences ($p > 0.05$), indicating successful randomisation. The mean age of the study population was 36.8 ± 8.4 years and the sex distribution was similar across the three groups **(Table 1)**. In the combined cohort of 45 participants, all clinical parameters demonstrated statistically significant improvements at one and three months compared with baseline. The plaque index decreased from 2.34 ± 0.29 at baseline to 0.93 ± 0.17 at one month and 0.50 ± 0.13 at three months. Similarly, the gingival index decreased from 2.16 ± 0.25 to 1.06 ± 0.18 and 0.57 ± 0.15, respectively. Probing pocket depth (PPD) decreased from 4.93 ± 0.59 mm at baseline to 3.20 ± 0.56 mm at one month and 2.46 ± 0.51 mm at three months. Relative attachment level (RAL) also improved, decreasing from 8.86 ± 0.74 mm at baseline to 6.20 ± 0.94 mm at one month and 5.13 ± 0.63 mm at three months. All within-group changes were statistically significant ($p < 0.001$; **Table 1**). Intergroup comparisons of clinical outcomes revealed a statistically significant difference in PPD reduction at one month. The mean reductions were 1.73 ± 0.88 mm in Group A (scaling and root planing (SRP) alone), 2.20 ± 0.77 mm in Group B (SRP plus curcumin chip) and 2.33 ± 0.72 mm in Group C (SRP plus Aloe vera chip). The overall intergroup difference was statistically significant ($p = 0.043$), with a significant post hoc difference between Groups A and C ($p = 0.025$). At three months, Group C showed the greatest mean PPD reduction (3.13 ± 0.74 mm), followed by Group B (2.60 ± 0.91 mm) and Group A (2.46 ± 0.74 mm). However, the intergroup difference was not statistically significant ($p = 0.062$). RAL reductions did not differ significantly among the three groups at either one month ($p = 0.162$) or three months ($p = 0.445$), although all groups showed significant within-group improvements ($p < 0.001$; **Table 2**). Microbiological analysis showed significant reductions in the counts of *Porphyromonas gingivalis*, *Fusobacterium nucleatum* and *Aggregatibacter actinomycetemcomitans* in all three treatment groups at one and three months compared with baseline ($p < 0.001$ for all within-group comparisons). For *P. gingivalis*, the counts at three months decreased to 0.22 ± 0.05, 0.40 ± 0.29 and 0.33 ± 0.24 × 10^5 CFU/mL in Groups A, B and C, respectively. For *F. nucleatum*, the corresponding counts were 0.35 ± 0.37, 0.40 ± 0.29 and 0.40 ± 0.26 × 10^5 CFU/mL. For *A. actinomycetemcomitans*, the counts at three months were 0.42 ± 0.19, 0.24 ± 0.14 and 0.37 ± 0.21 × 10^5 CFU/mL in Groups A, B and C, respectively **(Table 3)**. Post hoc Tukey comparisons of microbiological counts at three months revealed no statistically significant differences between the treatment groups for *P. gingivalis* or *F. nucleatum* (all adjusted p-values > 0.05). For *A. actinomycetemcomitans*, the difference between Groups A and B was statistically significant ($p = 0.039$), whereas the differences between Groups A and C ($p = 0.775$) and Groups B and C ($p = 0.165$) were not statistically significant **(Table 4)**.

Table 1: Mean clinical parameter values and intragroup changes from baseline for the combined cohort (n=45)

Clinical Parameter	Time Point	Mean ± SD	Std. Error of Mean	Mean Change from Baseline	p-value (vs Baseline)
Plaque Index	Baseline	2.34 ± 0.29	0.07	—	—
	1 Month	0.93 ± 0.17	0.45	1.40 ± 0.13	<0.001
	3 Months	0.50 ± 0.13	0.35	1.83 ± 0.17	<0.001
Gingival Index	Baseline	2.16 ± 0.25	0.06	—	—
	1 Month	1.06 ± 0.18	0.04	1.10 ± 0.07	<0.001
	3 Months	0.57 ± 0.15	0.04	1.58 ± 0.11	<0.001
Probing Pocket Depth (mm)	Baseline	4.93 ± 0.59	0.15	—	—
	1 Month	3.20 ± 0.56	0.14	1.73 ± 0.53	<0.001

	3 Months	2.46 ± 0.51	0.13	2.47 ± 0.62	<0.001
Relative Attachment Level (mm)	Baseline	8.86 ± 0.74	0.19	—	—
	1 Month	6.20 ± 0.94	0.24	2.66 ± 0.89	<0.001
	3 Months	5.13 ± 0.63	0.16	3.73 ± 0.79	<0.001

PPD = probing pocket depth; RAL = relative attachment level; PI = plaque index; GI = gingival index. All intragroup comparisons by paired t-test. p<0.001 for all parameters at both follow-up time points versus baseline

Table 2: Mean reductions in probing pocket depth (ppd) and relative attachment level (ral) by treatment group at one month and three months

Parameter / Time Interval	Group A (SRP alone) Mean ± SD	Group B (SRP + Curcumin) Mean ± SD	Group C (SRP + Aloe vera) Mean ± SD	Intergroup p-value (ANOVA)	Post-hoc Significance
PPD — Baseline−1 Month (Mean Reduction, mm)	1.73 ± 0.88	2.20 ± 0.77	2.33 ± 0.72	0.043	A vs C: p=0.025*
PPD — Baseline−3 Months (Mean Reduction, mm)	2.46 ± 0.74	2.60 ± 0.91	3.13 ± 0.74	0.062	NS
RAL — Baseline−1 Month (Mean Reduction, mm)	2.66 ± 0.89	3.26 ± 0.96	3.13 ± 0.74	0.162	NS
RAL — Baseline−3 Months (Mean Reduction, mm)	3.73 ± 0.79	4.06 ± 0.70	4.00 ± 0.75	0.445	NS

*NS = not significant. PPD and RAL reductions shown as mean difference (mm) from baseline. All within-group changes significant at p<0.001 (paired t-test) *p=0.025 by post-hoc Tukey test*

Table: 3 Microbiological counts (×10⁵ CFU/mL) of periodontal pathogens by treatment group at baseline, one month and three months

Organism (CFU/mL ×10⁵)	Group	Baseline Mean ± SD	1 Month Mean ± SD	3 Months Mean ± SD	Intragroup p-value*
P. gingivalis	A (SRP)	2.95 ± 0.44	1.18 ± 0.19	0.22 ± 0.05	<0.001
	B (Curcumin)	2.80 ± 0.64	1.24 ± 0.36	0.40 ± 0.29	<0.001
	C (Aloe vera)	2.89 ± 0.57	1.35 ± 0.34	0.33 ± 0.24	<0.001
F. nucleatum	A (SRP)	3.15 ± 0.41	1.07 ± 0.35	0.35 ± 0.37	<0.001
	B (Curcumin)	2.80 ± 0.64	1.24 ± 0.36	0.40 ± 0.29	<0.001
	C (Aloe vera)	2.82 ± 0.50	1.30 ± 0.34	0.40 ± 0.26	<0.001
A. actinomycetemcomitans	A (SRP)	2.82 ± 0.57	1.04 ± 0.20	0.42 ± 0.19	<0.001
	B (Curcumin)	2.78 ± 0.55	1.23 ± 0.30	0.24 ± 0.14	<0.001
	C (Aloe vera)	2.86 ± 0.42	1.22 ± 0.28	0.37 ± 0.21	<0.001

CFU = colony-forming units. All intragroup changes (baseline to 1 month and 3 months) significant at p<0.001 by paired t-test for all organisms in all groups. Group A = SRP alone; Group B = SRP + Curcumin chip; Group C = SRP + Aloe vera chip

Table: 4 Post-hoc tukey test: intergroup comparison of microbiological counts at three months

Organism	Group Comparison (3 Months)	Mean Difference (CFU/mL ×10⁵)	Adjusted p-value (Tukey)
P. gingivalis	A vs B	0.09	0.585
	A vs C	0.17	0.191
	B vs C	0.07	0.717
F. nucleatum	A vs B	−0.05	0.904
	A vs C	−0.01	0.998
	B vs C	0.04	0.928
A. actinomycetemcomitans	A vs B	0.17	0.039*
	A vs C	0.05	0.775
	B vs C	−0.13	0.165

**Statistically significant (p<0.05). All other pairwise comparisons at three months were not statistically significant (p>0.05)*

Discussion:

The present randomised controlled trial evaluated the clinico-microbiological efficacy of curcumin and aloe vera chips as adjuncts to SRP in patients with Stage II Grade A periodontitis over a 3-month follow-up period. All three treatment modalities - SRP alone, SRP + curcumin chip and SRP + aloe vera chip- produced statistically significant improvements in PI, GI, PPD and RAL from baseline to 3 months and significant reductions in the colony counts of all three evaluated periodontal pathogens. The primary differentiating findings were: (i) the aloe vera chip group achieved a significantly greater early reduction in PPD compared with SRP alone at the one-month time point; and (ii) the curcumin chip group demonstrated significantly superior reduction in *A. actinomycetemcomitans* counts at 3 months compared with SRP alone. These targeted advantages of the herbal LDD systems over SRP alone, while modest in magnitude, are clinically relevant and support the growing evidence base for naturally derived adjunctive agents in periodontal therapy **[7].** The seminal investigation of Haffajee *et al.* characterised the microbiological response to SRP in detail, showing significant reductions in the counts of periodontal pathogens including *P. gingivalis*, *F. nucleatum* and *A. actinomycetemcomitans* following non-surgical therapy, while also noting that many sites were recolonised to pre-therapy microbial levels by 3 months in a subset of patients **[8]**. This recolonisation tendency provides the mechanistic rationale for adjunctive LDD: sustained delivery of an antimicrobial agent directly into the treated pocket may suppress residual and re-colonising bacteria beyond the capacity of mechanical debridement alone, particularly in anatomically complex pockets where instrumentation access is limited **[3]**. The local chip formulation avoids the systemic distribution, reduced tissue concentration

and resistance implications associated with systemically administered antibiotics, while achieving sustained therapeutic concentrations within the pocket milieu **[3].** In the present study, the curcumin chip group showed a consistent trend toward superior clinical outcomes relative to SRP alone across all time points, with the greatest mean PPD reduction at three months (2.60 ± 0.91 mm vs 2.46 ± 0.74 mm) and significantly superior reduction in *A. actinomycetemcomitans* at three months (p=0.039). These findings are consistent with the multiple a pharmacological mechanism through which curcumin exerts its periodontal benefits. Curcumin's primary anti-inflammatory mechanism involves suppression of the nuclear factor kappa-B (NF-κB) signalling pathway and downstream reduction of pro-inflammatory mediators including COX-2, TNF-α, IL-1β and IL-6; **[9]** additional mechanisms include suppression of Toll-like receptor (TLR) signalling pathways that are directly implicated in the inflammatory cascade in periodontitis **[10]**.And promotion of cellular migration and fibrinolysis during wound healing through modulation of urokinase plasminogen activator expression **[11]**. Heggers *et al.* showed in a split-mouth randomised controlled trial that a turmeric chip was equally effective as a chlorhexidine chip as an adjunct to SRP in the treatment of chronic periodontitis, producing significant reductions in PPD and clinical attachment level gain over 3 months **[12].** The aloe vera chip group showed the numerically greatest mean PPD reduction at both one month (2.33±0.72 mm) and three months (3.13±0.74 mm), with a statistically significant advantage over SRP alone at one month (p=0.025), suggesting a beneficial early therapeutic effect of the aloe vera LDD system on pocket depth reduction. Ajmera *et al.* showed the antibacterial and wound-healing-promoting properties of aloe vera gel *in vivo*, showing that elimination of bacteria with high inflammatory potential directly facilitated the wound healing process — a mechanism potentially operative in the enhanced gingival and periodontal tissue repair observed in the aloe vera group at the one-month time point **[13]**. The anti-gingivitis properties of aloe vera, reviewed by Ashifa *et al.*, are attributable to its combined antiplaque activity and potent anti-inflammatory characteristics, including direct inhibitory effects on bradykinin, histidine, COX-1 and COX-2 enzymes that collectively reduce gingival oedema and tissue engorgement, contributing to apparent reduction in probing depth **[14]**. The aloe vera gel additionally contains collagen-supporting elements — including vitamin C, mannose-6-phosphate, dermatan sulfate and hyaluronic acid — that support gingival connective tissue repair and complement the structural remodelling that accompanies PPD reduction **[14]**. The recent systematic review and meta-analysis by Jain *et al.* confirmed that aloe vera gel as an adjunct to mechanical debridement significantly improves periodontal clinical parameters compared with debridement alone, providing meta-analytic support for the findings of the present study **[15]**. Furthermore, aloin compounds present in aloe vera extracts exhibit structural similarity to tetracyclines and have been shown to inhibit MMP-2 and MMP-9, enzymes critically implicated in periodontal connective tissue destruction **[16]** representing a host-modulation benefit analogous to that of tetracycline-class periodontal adjuncts. The significantly greater reduction in *A. actinomycetemcomitans* at 3 months in the curcumin group relative to SRP alone (p=0.039) represents the most clinically relevant intergroup microbiological finding of the present study. The superior curcumin-mediated clearance of this organism at three months may reflect curcumin's documented antimicrobial activity against Gram-negative periodontal pathogens, potentially operating through membrane disruption and inhibition of bacterial virulence factor expression at the cellular level **[9]**. The aloe vera chip group demonstrated numerically lower *P. gingivalis* counts at three months (0.33 ± 0.24×10⁵ CFU/mL) compared with the SRP group (0.22 ± 0.05×10⁵ CFU/mL) and curcumin group (0.40 ± 0.29×10⁵ CFU/mL), though these differences were not statistically significant, consistent with the broader literature showing that the differential microbiological advantages of herbal LDD systems are organism-specific and may reflect the distinct antimicrobial mechanisms of curcumin versus aloe vera compounds. Limitations include the relatively small sample size, the single-centre design and the 3-month follow-up period which may not fully capture sustained or delayed differences in disease progression between groups

Conclusion:

Curcumin and aloe vera chips as adjuncts to scaling and root planing (SRP) significantly improved clinical and microbiological outcomes in Stage II Grade A periodontitis over 3 months. While all treatments showed progress from baseline confirming SRP as the gold standard for non-surgical care both herbal systems provided target benefits. Specifically, aloe vera chips led to greater initial reductions in probing pocket depth at 1 month, while curcumin chips proved superior in eliminating *A. actinomycetemcomitans* by the 3 month mark.

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