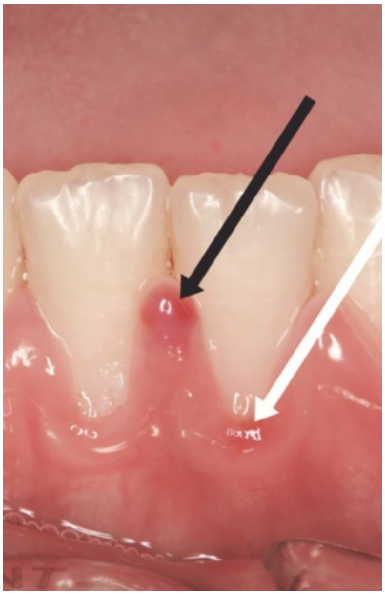
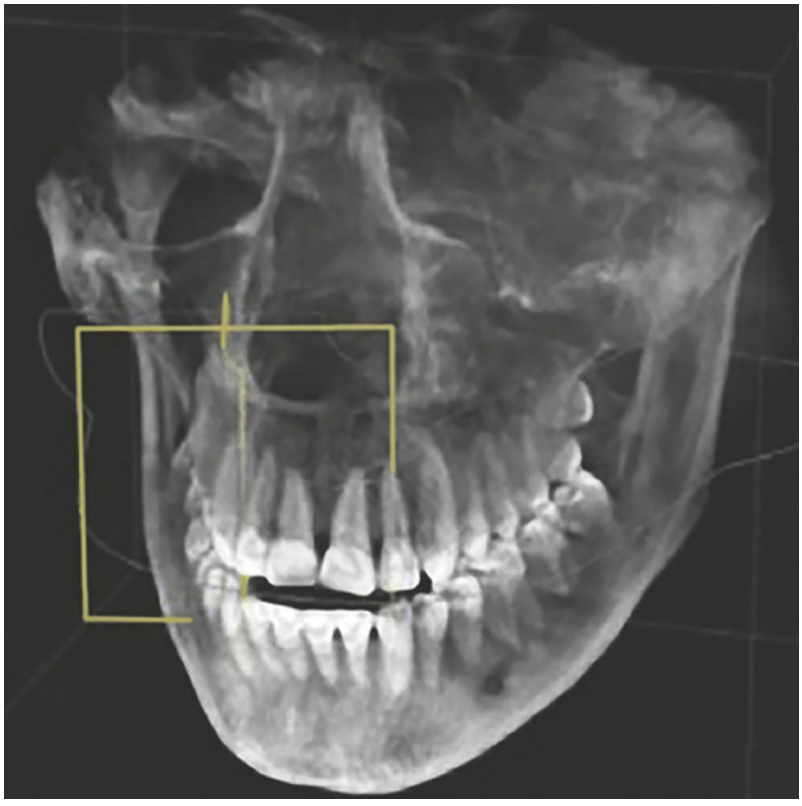
QUINTESSENCE INTERNATIONAL

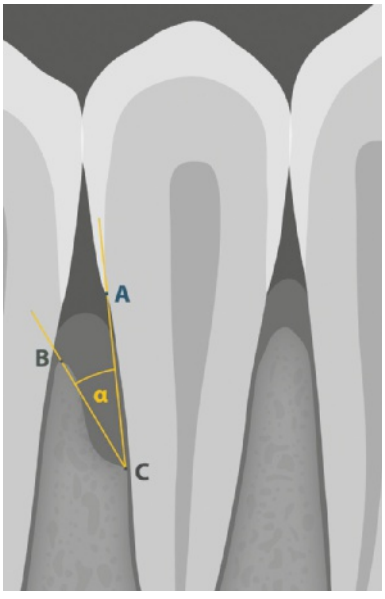
Editorial board's selected articles

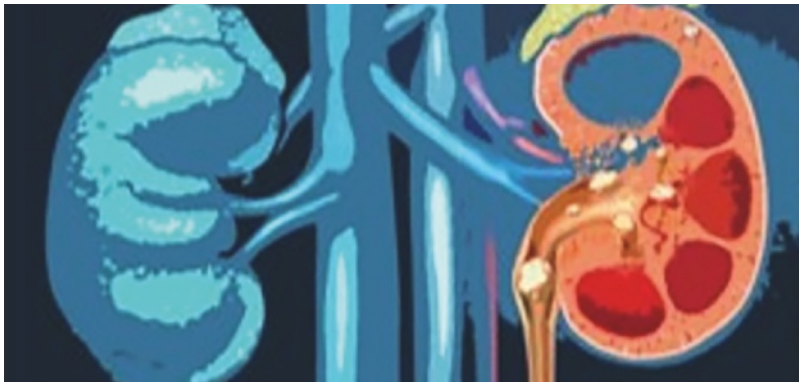
Volume 1

Eli Eliav (ed)









QUINTESSENCE PUBLISHING

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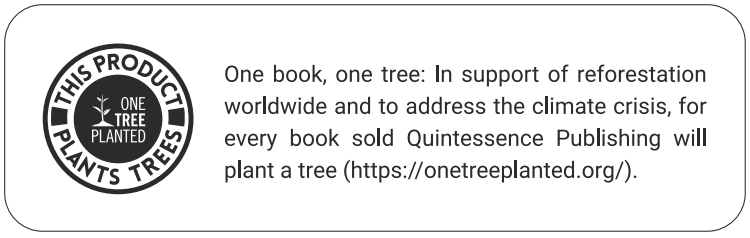

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PREFACE

This year marks a significant milestone, as Quintessence International is now offered exclusively as an online subscription. We are also delighted to introduce this first printed version of our annual yearbook, a compilation of selected articles from the past year.

Quintessence International caters to the global community of general dental practitioners, covering a wide array of topics and dentistry disciplines. Our team of editors, in collaboration with hundreds of reviewers from around the world, perform a remarkable job of reviewing and evaluating these submissions.

A key feature of our review process at Quintessence International is its anonymity. The double-blind process ensures that reviewers do not have access to information about the authors and are requested to assess the manuscripts solely based on their quality and content.

I have had the privilege of serving as the Editor in Chief of Quintessence International since 2008. It is both challenging and incredibly rewarding. Every month, we receive a significant number of manuscript submissions that demand careful review, assessment, and the selection of those suitable for publication.

Choosing manuscripts for publication from the multitude of outstanding submissions is a difficult task. Even more challenging was the selection of the 20 articles to be featured in the yearbook. To achieve this, we endeavored to represent the most significant work from the current year. Each of the Associate Editors selected articles from their respective disciplines and areas of interest, and we reached a collective decision on the final selection. We recognize that there are many other deserving manuscripts.

The articles in the yearbook are organized by disciplines and topics, with the aim of providing a valuable and user-friendly resource for our readers. We sincerely hope that you find it both enjoyable and informative. Looking ahead, we eagerly anticipate the manuscripts that will be submitted to Quintessence International in the coming year.

Thank you for your continued support and readership.

Eli Eliav
Editor in Chief

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RADIOLOGY/IMAGING

Effect of antibiotics as an adjuvant to subgingival instrumentation on systemic inflammation in patients with periodontitis: a randomized clinical trial

Manpreet Kaur, MDS/Rajinder Kumar Sharma, MDS/Shikha Tewari, MDS/Ritika Arora, MDS/Nishi Tanwar, MDS/Aditi Sangwan, MDS

Objectives: The aim of the present study was to evaluate the effect on systemic inflammation of subgingival instrumentation (SI) with or without antibiotics. Moreover, systemic parameters were compared between periodontally healthy (PH) individuals and periodontitis patients. **Method and materials:** Patients with generalized periodontitis: stage III and PH individuals were recruited. Forty eight periodontitis patients were randomly allocated to each treatment group; systemic antibiotics for seven days after completion of SI (AB group), or SI alone (SI group). Periodontal parameters, serum high-sensitivity C-reactive protein (hsCRP), and hematological parameters were assessed at baseline and at week 8. Multivariate analysis was applied to analyze predictive effect of treatment allocated and improvement in periodontal parameters on change in systemic parameters. **Results:** At baseline, hsCRP, total leukocyte count (TLC), neutrophil, and monocyte count were significantly higher in periodontitis patients. There was comparable reduction in neutrophil count in both treatment groups. At week 8, change in periodontal parameters was similar in treatment groups, except for probing pocket depth (PPD). Improvement in both PPD and clinical attachment level (CAL) and CAL alone was predictive of change in TLC and lymphocyte count, respectively. **Conclusion:** This study failed to demonstrate the significant benefit of systemic antibiotics as an adjuvant to SI on improvement in periodontal inflammation and systemic inflammatory parameters, despite significantly higher reduction in PPDs. *(Quintessence Int 2023;54:460–471; doi: 10.3290/j.qi.b3942249)*

Key words: C-reactive protein, inflammation, lymphocyte count, root planing

Non-communicable diseases (NCDs) are the major cause of mortality, accounting for 74% of the total deaths worldwide.[1] Of all NCD-related mortalities, 43.7% deaths are attributed to cardiovascular diseases (CVDs).[1] Infectious agents are postulated to be either directly involved in the CVD process or increase the systemic inflammation that further mediates its effects.[2] Periodontitis is characterized by microbially infected, host-mediated inflammation that results in loss of periodontal attachment.[3] Periodontitis is detrimental not only to periodontal tissues but also to cardiovascular health.[2] Periodontitis increases the propensity for occurrence of CVDs.[2] This effect is due to involvement of periodontal pathogens in formation and progression of atheromatous plaques.[4] As reported in mice,[5] inflammation induced by periodontal pathogens in vascular walls seems to result in formation of atheromatous plaques. Moreover, in periodontitis patients, periodontal pathogens are isolated from human atheromatous plaques.[6] Additionally, periodontal pathogens entering the systemic circulation stimulate liver to secrete acute phase proteins, such as high-sensitivity C-reactive protein (hsCRP) in circulation. Periodontitis also leads to alterations in total leukocyte count (TLC), differential leukocyte count (DLC), and blood parameters related to platelet activity. Higher blood viscosity due to alterations in TLC, DLC, and platelet parameters associated with periodontitis may be responsible for increased risk of thrombus formation. The altered count of these hematologic parameters due to periodontitis is of concern because of their association with increased risk for CVDs.[2,7-9] Also, serum hsCRP is predictive of

CVDs independent of traditional risk factors.[10] Owing to increased prevalence of severe periodontitis, being the sixth most prevalent condition affecting 11.2% population worldwide,[11] treatment of periodontitis is necessary not only to restore periodontal health but also to attain systemic health.

The periodontal pocket is colonized with anerobic bacteria responsible for chronic nonhealing lesions.[12,13] These morphologic and ecologic alterations are associated with loss of health-associated resilience of normal oral microbiome. It has the potential of creating a vicious cycle of microbially driven periodontal inflammation and more virulent microbial challenge.[14] Subgingival instrumentation (SI) results in resolution of periodontal inflammation as well as reduction in probing pocket depth (PPD). Despite clinical benefits of SI, putative periodontal pathogens cannot be eradicated with SI because of their penetrance into hard tissues of teeth including dentin and cementum,[15] and deeper soft periodontal tissues[16] through ulcerated pocket epithelium. Therefore, systemic antibiotics as an adjuvant to SI may be the treatment of choice. In a review article it was concluded that combination of systemic amoxicillin and metronidazole resulted in greater reduction in the number of periodontal pathogens and better clinical outcomes as compared to SI alone.[17] The suppressive effect of systemic antibiotics on periodontal pathogens was evident even at 12 months post-treatment and resulted in a significantly greater reduction in the number of sites with PPD ≥ 5 mm and a higher percentage of subjects reaching the clinical end point for treatment (≤ four sites with PPD ≥ 5 mm).[18] Furthermore, findings of a meta-analysis revealed that combination of systemic amoxicillin and metronidazole as an adjuvant to SI had an increased effect of about 40% to 50% in PPD reduction, higher percentage of pocket closure, clinical attachment level (CAL) gain, and bleeding on probing (BOP) reduction with maintenance of achieved clinical results up to 12 months of follow-up.[19] The presence of pathogenic bacteria is not only limited to periodontal tissues but is also evident in circulation and atheromatous plaques. Treatment of severe periodontitis with SI and systemic antibiotics,[20] or local drug delivery system[21] has been reported to bear a positive effect on systemic health by reducing systemic inflammation[20,21] and improving endothelial function.[20] However, a meta-analysis revealed that SI resolves systemic inflammation measured in terms of reduction in hsCRP only in patients with other comorbidities.[22]

An additional role of systemic antibiotics as an adjuvant to SI versus SI alone has not been evaluated to date. With this aim, the present randomized trial was conducted to evaluate the effect of combination of systemic amoxicillin and metronidazole as an adjuvant to SI versus SI alone on resolution of systemic inflammation measured in terms of variations in hsCRP, and hematologic parameters including TLC, DLC, platelet count, mean platelet volume (MPV), and platelet distribution width (PDW) in patients with periodontitis.

Method and materials

Study design and ethics statement

The present parallel-design, examiner blinded, randomized clinical trial was conducted in department of Periodontics, Post Graduate Institute of Dental Sciences (PGIDS), Rohtak, Haryana, India. The study protocol was approved by the Biomedical and Health Research Ethics Committee (PGIDS/BHRC/20/13). The study was conducted in accordance with the Declaration of Helsinki 1975, as revised in 2013.

Study population

Individuals aged 35 to 45 years were recruited from the outpatient department of periodontics. The study population included systemically healthy individuals having at least 20 natural teeth excluding third molars. The study population comprised periodontitis patients (25 women and 23 men; mean age 39.67 ± 3.55 years) and periodontally heathy (PH) individuals (10 females, and 8 males; mean age 38.94 ± 3.73 years). Periodontitis criteria included stage III periodontitis[3] with ≥ 30% teeth involved and BOP at > 30% sites.[23] PH individuals were defined as having < 10% bleeding sites with PPD ≤ 3 mm.[23]

Exclusion criteria

Exclusion criteria were as follows:

- history of periodontal treatment in the previous 1 year
- presence of periapical lesions due to caries or periodontitis
- undergoing or requiring an extensive dental or orthodontic treatment
- individuals requiring prophylactic antibiotics prior to dental treatment
- confirmed or assumed allergies or hypersensitivity reactions to amoxicillin and/or metronidazole
- alcohol consumers
- history of systemic medications known to influence periodontal status such as steroids, immunosuppressants, antibiotics, anti-inflammatory drugs, lipid lowering drugs, anti-convulsants, anti-coagulants, anti-hypertensives, or any other host modulatory drugs within 6 months prior to study commencement

- pregnant women or lactating mothers
- current or former users of tobacco in any form
- history of infections within 2 months prior to study commencement.

Individuals meeting the inclusion criteria were explained the purpose of the study, provided with a patient information sheet, and enrolled after obtaining written informed consent.

Sample size calculation

Sample size was estimated using the software G-Power 3.0.10 (Heinrich-Heine University, Dusseldorf, Dusseldorf, Germany). With 80% power and α error of 5%, to detect a difference of 0.5 mg/L in hsCRP between the two treatment groups based on a previous study,[22] for a standard deviation of 0.5 mg/L, the sample size was calculated to be 18 individuals in each group. To compensate for potential drop-outs, 24 individuals were recruited in each treatment group.

Randomization

Patients with periodontitis were randomly allocated to receive one of the two treatments (Fig 1). Randomization was performed by block randomization technique (four-unit block size), with equal allocation between groups by a clinician unrelated to the present study. Allocation sequence was concealed in an opaque envelope. As placebo drug was not allocated, patients were not blinded to the assigned treatment group. However, the researchers (RS) assessing the outcomes of the study, and performing SI (MK) were unaware of treatment allocated.

Periodontal examination

Plaque Index (PI),[24] Gingival Index (GI),[25] BOP, PPD, and CAL were recorded using a periodontal probe (PCP-UNC 15 periodontal probe; Hu-Friedy) at six sites per tooth excluding third molars. Periodontal inflamed surface area (PISA) was calculated.[26] PPD, BOP, CAL, and recession were taken into consideration for calculating the PISA using a freely available spreadsheet (www.parsprototo.info). All periodontal parameters were analyzed at baseline and during the follow-up visit at 8 weeks in both treatment groups. As PH individuals participated only at baseline, periodontal parameters were recorded only at baseline. Periodontal examination was carried out by a single investigator blinded to treatment allocation (RS) to preclude inter-examiner variability. Prior to study commencement, the calibration exercise was performed at two occasions 48 hours apart in ten periodontitis patients not included in the present study. The intraclass correlation coefficient for PPD and CAL was calculated to be 0.92 and 0.90, respectively.

Anthropometric measurement

Body mass index (BMI) was calculated in the study population at baseline.

Evaluation of hsCRP and hematologic parameters

Following overnight fasting, venous blood samples were obtained from all the participants of the study at baseline. Blood samples were again collected at 8 weeks for both treatment groups.

Serum hsCRP was measured using commercial enzyme-linked immunosorbent assay (ELISA) kits (Calbiotech). Assays were carried out according to the manufacturers' recommendations.

Total leukocyte count (TLC), differential leukocyte count (DLC) including neutrophil count, lymphocyte count, monocyte count, eosinophil count, and basophil count; platelet count; MPV; and PDW were assessed. All hematologic parameters were assessed using an automated hematology analyzer (BC-5800, Mindray).

Periodontal treatment

At baseline, after periodontal examination and blood sample collection, oral hygiene instructions were given to periodontitis patients. SI was then performed in various sessions using manual scalers (Hu-Friedy), curettes (Hu-Friedy), and an ultrasonic scaler (EMS, Nylon) by a single researcher (MK), and was completed within 1 week. One treatment group was prescribed amoxicillin (500 mg) and metronidazole (400 mg) thrice daily for 7 days (AB group) at the last session of SI. The SI group received only SI treatment.

Follow-up visits and reevaluation

Patients of both treatment groups were recalled at weeks 4 and 8. Professional plaque control was performed, and reinforcement of oral hygiene instructions was done at week 4. At week 8, periodontal examination was performed. Serum hsCRP and hematologic parameters were again analyzed for both treatment groups.

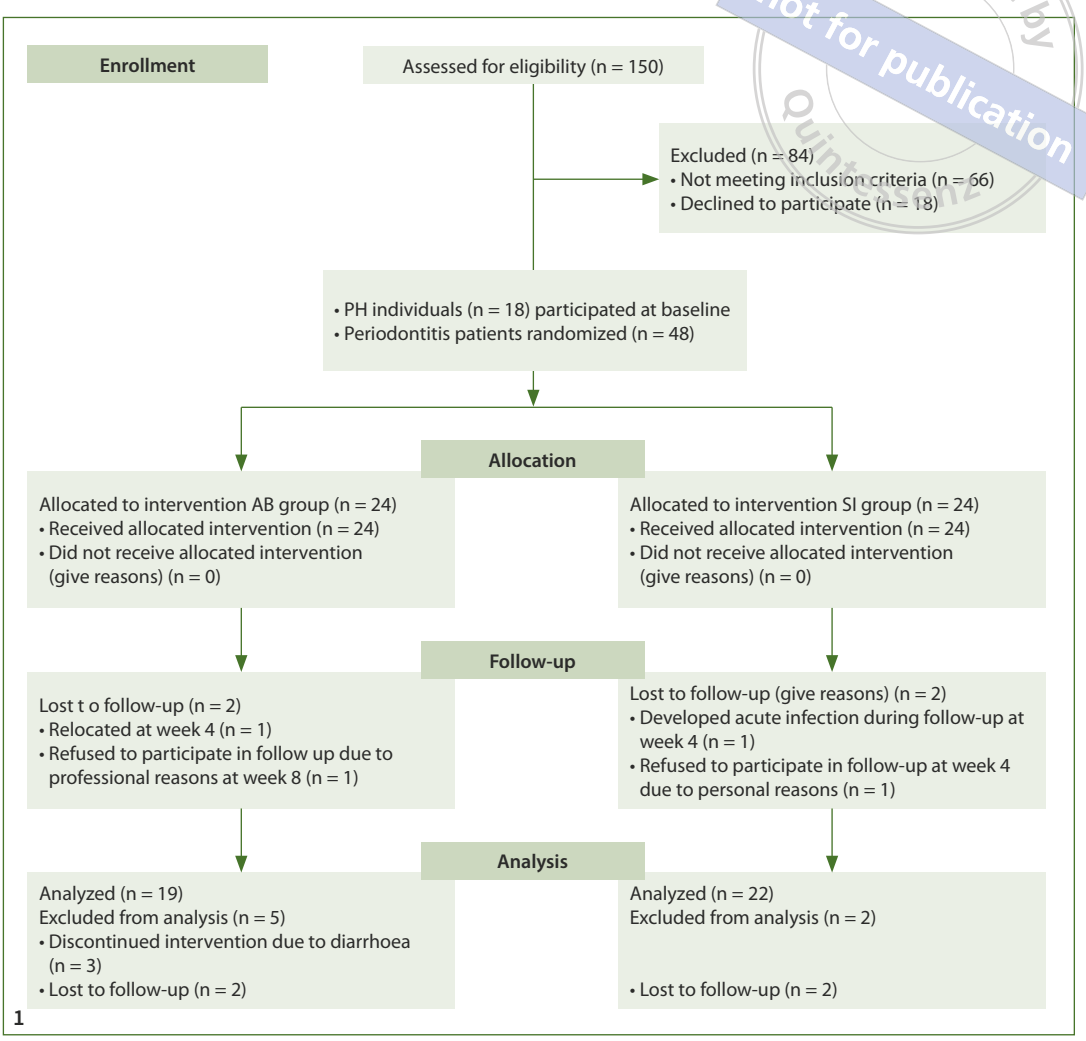

Fig 1 CONSORT flow diagram of the study population.

Outcome variables

Change in hsCRP at week 8 was the primary outcome variable, and changes in hematologic parameters were the secondary outcome variables.

Statistical analysis

Statistical analysis was performed using the statistical software program SPSS (v.20, IBM). Normality of data was determined using the Shapiro–Wilk test. Normally distributed and nonnormally distributed continuous data are presented as mean ± standard deviation (SD) and median (25th; 75th percentile), respectively. Difference in between groups was assessed using the unpaired Student *t* test (for normally distributed data) and the Mann–Whitney U test (for nonnormally distributed data). Intragroup comparison at two different time points was done using the paired *t* test and Wilcoxon signed-rank test for parametric and nonparametric data, respectively. Sex, being a categorical variable, is presented as number (percentage), and was analyzed using the chi-squared test. Spearman rank correlation coefficient was calculated to determine the association between the improvement in periodontal parameters and the change in systemic parameters at week 8. Parameters having significant correlations were further analyzed using multivariate linear regression to assess whether treatment group and improvement in periodontal parameters predicted change in systemic parameters. Statistical significance was set at $P < .05$.

Results

Study sample

The PH group included 18 patients. In total, 24 periodontitis patients were enrolled in each treatment group (Fig 1). However, 19 patients in the AB group and 22 patients in the SI group completed the 8-week follow-up. Details of recruitment and drop-outs of the study population are shown in the CONSORT flow diagram (Fig 1). The actual study start date was 26 November 2021, and the study was completed on 10 June 2022.

Demographic and anthropometric variables

There were no statistically significant differences in any of the variables between periodontitis patients and PH individuals ($P > .05$; Table 1) at baseline. Variables were also comparable between the AB and SI groups ($P > .05$; Table 1).

Periodontal parameters

All the periodontal parameters were significantly higher in patients with periodontitis than in PH individuals at baseline ($P = .000$; Table 1). At baseline, all periodontal parameters were comparable in both the treatment groups ($P > .05$; Table 1), except for a significantly higher PPD in the AB group than in the SI group ($P = .018$; Table 1).

At 8 weeks after periodontal treatment, both treatments (AB and SI) led to significant improvement/change [Δ] in periodontal parameters ($P = .000$; Table 1). ΔPI, ΔGI, ΔBOP, ΔCAL, and ΔPISA were comparable in both the AB group and the SI group, apart from a significantly higher ΔPPD in the AB group than in the SI group ($P = .043$; Table 1).

hsCRP and hematologic parameters

At baseline, compared to PH individuals, periodontitis patients had a significantly higher hsCRP, TLC, neutrophil count, and monocyte count ($P < .05$; Table 2). However, lymphocyte count, eosinophil count, basophil count, platelet count, MPV, and PDW were comparable in periodontitis patients and PH individuals ($P > .05$; Table 2).

At baseline and at week 8, there was no significant difference in hsCRP and other hematologic parameters in both the AB and the SI groups ($P > .05$; Table 2). The effect size for ΔhsCRP was −0.608 with 95% confidence interval (CI) of −1.999 to 1.611. There was a significant decrease in neutrophil count in both the treatment groups at week 8 ($P < .05$; Table 2); however, Δneutrophil count was comparable in the AB group and SI group ($P > .05$; Table 2) with effect size (95% CI) of 0.199 (−0.342 to 0.656). Effect sizes (95% CI) for ΔTLC, Δlymphocyte count, Δmonocyte count, Δeosinophil count, and Δbasophil count were 0.048 (−0.789 to 0.918), −0.130 (−0.516 to 0.340), 0.042 (−0.088 to 0.101), −0.123 (−0.105 to 0.071), and 0.433 (−0.003 to 0.015), respectively. ΔPlatelet count, ΔMPV, and ΔPDW had effect sizes (95% CI) of 0.005 (−31.488 to 31.966), −0.524 (−1.052 to 0.100), and −0.348 (−1.130 to 0.328), respectively.

Correlation and multivariate analysis between Δperiodontal parameters and Δsystemic parameters

A significant positive correlation was observed in the following parameters: ΔPPD/Δbasophil count (rho = 0.437, $P = .004$), ΔCAL/Δbasophil count (rho = 0.439, $P = .004$) (Table 3). A significant negative correlation existed between the following parameters: ΔBOP/Δeosinophil count (rho = −0.323, $P = .039$), ΔPPD/Δeosinophil count (rho = −0.328, $P = .036$), ΔCAL/ΔTLC (rho = −0.316, $P = .044$), ΔCAL/Δlymphocyte count (rho = −0.374, $P = .016$), ΔCAL/ Δmonocyte count (rho = −0.374, $P = .016$), ΔCAL/Δeosinophil count (rho = −0.360, $P = .021$), and ΔPISA/Δeosinophil count (rho = −0.374, $P = .016$) (Table 3). However, ΔhsCRP, Δneutrophil count, Δplatelet count, ΔMPV, and ΔPDW had no association with Δperiodontal parameters ($P > .05$, Table 3).

Multivariate analysis revealed that treatment intervention (AB or SI group) did not predict Δhematologic parameters at week 8 ($P > .05$; Table 4). ΔPPD was a positive predictor whereas ΔCAL was a negative predictor for ΔTLC ($P = .041$, $P = .007$, respectively, Table 4). ΔCAL was a significant negative predictor for Δlymphocyte count ($P = .020$; Table 4).

Discussion

The degree of periodontal inflammation, periodontal destruction, measured in terms of PISA and CAL, respectively, as well as the amount of plaque were comparable in AB and SI groups at baseline. Similar improvement in periodontal parameters was evident in both the treatment groups at week 8. However, in the AB group, a significantly greater improvement in PPD was found than in the SI group, which may be due to a higher PPD at baseline in the AB group, as sites with deeper periodontal pockets undergo better healing than sites with shallower PPD.[27]

In agreement with previous studies,[28,29] serum hsCRP levels were significantly higher in periodontitis patients (mean ± SD 3.36 ± 3.27 mg/L) than in PH individuals (1.01 ± 1.08 mg/L). There

Table 1 Characteristics of study population at baseline, at week 8, and improvement (Δ) in periodontal parameters at week 8 compared to baseline

Variable			Periodontitis patients (n = 41)			
			PH (n = 18)	AB (n = 19)	SI (n = 22)	*P* value
Demographic characteristics	Age (y)	Baseline	38.50 (35.75;42.50)	40.00 (36.00;44.00)		.342†
		Baseline	NA	37.00 (36.00;42.00)	40.00 (36.75;45.00)	.202†
	Sex (M:F), n (%)	Baseline	8 (44.44):10 (55.56)	16 (39.02):25 (60.98)		.696‖
		Baseline	NA	8 (42.11) :11 (57.89)	8 (36.36):14 (63.64)	.707‖
Anthropometric parameter	BMI (kg/m^2)	Baseline	24.59 ± 3.22	25.34 ± 3.86		.473‡
		Baseline	NA	26.50 ± 2.60	24.33 ± 4.50	.072‡
Periodontal parameters	Mean PI	Baseline	0.09 (0.06;0.20)	1.97 (1.71;2.26)		.000†*
		Baseline	NA	1.97 (1.77;2.24)	1.99 (1.64;2.32)	.855†
		Week 8	NA	0.17 (0.07;0.44)	0.28 (0.17;0.56)	.154†
		Δ	NA	1.73 (1.45;1.93)	1.57 (1.30;1.78)	.205†
		P value	NA	.000§*	.000§*	NA
	Mean GI	Baseline	0.05 (0.01;0.06)	1.32 (1.19;1.46)		.000†*
		Baseline	NA	1.35 (1.20;1.48)	1.30 (1.16;1.42)	.340†
		Week 8	NA	0.15 (0.04;0.32)	0.11 (0.05;0.22)	.656†
		Δ	NA	1.22 (1.01;1.33)	1.17 (1.06;1.21)	.556†
		P value	NA	.000§*	.000§*	NA
	Mean BOP (%)	Baseline	3.94 (2.98;6.45)	66.07 (55.93;78.87)		.000†*
		Baseline	NA	67.33 (57.69;79.76)	64.51 (53.72;78.72)	.583†
		Week 8	NA	10.67 (6.79;28.57)	13.03 (8.74;18.16)	.754†
		Δ	NA	50.64 (47.43;63.09)	53.09 (40.40;62.63)	.937†
		P value	NA	.000§*	.000§*	NA
	Mean PPD (mm)	Baseline	1.49 (1.19;1.77)	4.01 (3.68;4.57)		.000†*
		Baseline	NA	4.32 (4.01;4.61)	3.87 (3.63;4.14)	.018†*
		Week 8	NA	2.90 (2.58;3.84)	3.09 (2.69;3.32)	.714†
		Δ	NA	1.18 (0.93;1.64)	0.91 (0.50;1.36)	.043†*
		P value	NA	.000§*	.000§*	NA
	Mean CAL (mm)	Baseline	0.00 (0.00;0.05)	4.49 (4.18;4.95)		.000†*
		Baseline	NA	4.62 (4.24;5.09)	4.38 (4.02;4.75)	.261†
		Week 8	NA	3.34 (3.04;4.25)	3.55 (3.28;4.11)	.574†
		Δ	NA	1.00 (0.88;1.28)	0.91(0.40;1.37)	.308†
		P value	NA	.000§*	.000§*	NA
	Mean PISA (mm^2)	Baseline	40.20 (31.78;72.83)	2,021.04 (1,422.48;2,295.86)		.000†*
		Baseline	NA	2,086.97 (1,528.48;2,412.38)	1,684.31 (1,385.78;2,291.70)	.219†
		Week 8	NA	237.94 (102.96;384.21)	283.18 (207.33;413.19)	.548†
		Δ	NA	1,748.43 (1,202.35;2,043.02)	1,394.32 (1,082.96;1,848.49)	.239†
		P value	NA	.000§*	.000§*	NA

Parametric and nonparametric data are presented as mean ± SD or median (25th; 75th percentile), respectively.
*Statistical significance (*P* < .05).
†Difference between groups at each time point was assessed by the Mann–Whitney U test (nonparametric data)
‡Difference between groups at each time point was assessed by the unpaired Student *t* test (parametric data).
§Intragroup difference over time was analyzed using Wilcoxan signed-rank test (nonparametric data).
‖Sex was compared between groups using chi-squared test.
BMI, body mass index; BOP, bleeding on probing; CAL, clinical attachment level; F, female; GI, gingival index; M, male; NA, not applicable; PI, Plaque Index; PISA, periodontal inflamed surface area; PPD, probing pocket depth.

are contradictory findings on the effect of SI on hsCRP,[29-32] and a meta-analysis[22] reported no effect of SI on hsCRP in systemically healthy individuals. Similarly, the present study reported no change in hsCRP levels in periodontitis patients treated with SI. Treatment with antibiotics as an adjuvant also had no effect on reduction of hsCRP, although previous studies reported that periodontal treatment with SI and systemic[20] or local antibiotics[21] resulted in a decrease in hsCRP.[20,21] However, a recent meta-analysis supports the findings of the present study that SI alone or with local or systemic antibiotics has no advantage in

Table 2 Serum inflammatory marker and hematologic parameters at baseline and at week 8, and their improvement (Δ) at week 8 in the study population

Variables			Periodontitis patients (n = 41)			
			PH (n = 18)	AB (n = 19)	SI (n = 22)	*P* value
Serum inflammatory marker	hsCRP (mg/L)	Baseline	0.41 (0.29;1.74)	2.59 (1.10;4.33)		.001†*
		Baseline	NA	3.21 (1.34;5.17)	2.14 (0.67;3.56)	.229†
		Week 8	NA	3.57 (1.39;5.61)	1.59 (0.81;5.53)	.272†
		Δ	NA	0.49 (−0.84;1.52)	−0.10 (−1.70;1.78)	.794†
		P value	NA	.687§	.935§	NA
Hematologic parameters	TLC (× 10^9/L)	Baseline	5.27 (4.46;6.63)	6.32 (5.33;7.59)		.024†*
		Baseline	NA	6.55 (5.35;7.54)	6.30 (5.23;7.68)	.410†
		Week 8	NA	5.63 (5.21;7.96)	6.00 (5.06;7.25)	.629†
		Δ	NA	0.38 (−0.67;1.32)	0.20 (−0.66;1.58)	.927†
		P value	NA	.165§	.205§	NA
	Neutrophil count (× 10^9/L)	Baseline	2.92 ± 1.02	3.82 ± 1.01		.003‡*
		Baseline	NA	3.99 ± 0.92	3.66 ± 1.08	.302‡
		Week 8	NA	3.46 ± 0.97	3.29 ± 1.14	.608‡
		Δ	NA	0.54 ± 0.77	0.38 ± 0.80	.528‡
		P value	NA	.007¶*	.038¶*	NA
	Lymphocyte count (× 10^9/L)	Baseline	2.12 (1.59;2.39)	2.02 (1.64;2.53)		.974†
		Baseline	NA	1.96 (1.65;2.32)	2.06 (1.58; 2.67)	.744†
		Week 8	NA	2.12 (1.71;2.28)	2.00 (1.43;2.61)	.548†
		Δ	NA	−0.01(−0.34;0.25)	0.19(−0.36;0.56)	.513†
		P value	NA	.615§	.721§	NA
	Monocyte count (× 10^9/L)	Baseline	0.33 ± 0.13	0.41 ± 0.14		.036‡*
		Baseline	NA	0.42 ± 0.15	0.41 ± 0.14	.692‡
		8th week	NA	0.40 ± 0.10	0.39 ± 0.13	.752‡
		Δ	NA	0.03 ± 0.15	0.02 ± 0.15	.895‡
		P value	NA	.451¶	.554¶	NA
	Eosinophil count (× 10^9/L)	Baseline	0.13 (0.07;0.24)	0.16 (0.10;0.34)		.378†
		Baseline	NA	0.18 (0.09;0.34)	0.15 (0.10;0.34)	.875†
		Week 8	NA	0.18 (0.14;0.35)	0.15 (0.10;0.34)	.417†
		Δ	NA	−0.02 (−0.07;0.02)	−0.01(−0.06;0.05)	.556†
		P value	NA	.276§	.782§	NA
	Basophil count (× 10^9/L)	Baseline	0.02(0.02;0.03)	0.02(0.01;0.02)		.171†
		Baseline	NA	0.02 (0.01;0.03)	0.02 (0.010;.02)	.425†
		Week 8	NA	0.02 (0.01;0.02)	0.02 (0.01;0.03)	.639†
		Δ	NA	0.00 (−0.01;0.01)	0.00 (−0.01;0.00)	.205†
		P value	NA	.719§	.173§	NA
	Platelet count (× 10^9/L)	Baseline	229.50 (209.50;284.75)	231.00 (183.50;277.50)		.425†
		Baseline	NA	243.00 (190.00;294.00)	220.50 (176.50;274.00)	.565†
		Week 8	NA	251.00 (219.00;320.00)	259.00 (187.50;278.75)	.705†
		Δ	NA	−9.00 (−58.00;18.00)	−7.50 (−34.25;4.50)	.814†
		P value	NA	.286§	.104§	NA
	MPV (fL)	Baseline	9.50 (8.83;9.88)	9.30 (8.05;10.75)		.993†
		Baseline	NA	8.90 (8.00;9.70)	9.70 (8.60;11.45)	.102†
		Week 8	NA	8.70 (8.00;10.20)	9.80 (8.58;10.55)	.301†
		Δ	NA	0.10 (−0.70;0.40)	0.30(−0.23;0.78)	.158†
		P value	NA	.663§	.127§	NA
	PDW (%)	Baseline	16.00 (15.78;16.15)	16.10 (15.70;16.65)		.265†
		Baseline	NA	16.10 (15.40;16.50)	16.15 (15.78;16.80)	.307†
		Week 8	NA	15.90 (15.60;16.80)	16.10 (15.77;16.80)	.394†
		Δ	NA	0.00 (−0.20;0.30)	−0.05(−0.23;0.30)	.896†
		P value	NA	.793§	.970§	NA

Parametric and nonparametric data are presented as means ± SD or median (25th;75th percentile), respectively.
*Statistically significant (*P* <.05).
†Difference between groups at each time point was assessed by the Mann–Whitney U-test (nonparametric data).
‡Difference between groups at each time point was assessed by the unpaired Student *t* test (parametric data).
§Intragroup difference over time was analyzed using Wilcoxon signed-rank test (nonparametric data).
¶Intragroup difference over time was analyzed using paired *t* test (parametric data).
hsCRP, high-sensitivity C-reactive protein; MPV, mean platelet volume; PDW, platelet distribution width, TLC, total leukocyte count.

Table 3 Correlation between improvement (Δ) in periodontal parameters and change (Δ) in systemic parameters at week 8 with respect to baseline

Parameters		ΔPI	ΔGI	ΔBOP	ΔPPD	ΔCAL	ΔPISA
ΔhsCRP (mg/L)	rho	0.103	0.067	−0.145	0.080	0.103	−0.113
	P value	.520	.677	.365	.619	.523	.482
ΔTLC (×10^9/L)	rho	−0.127	−0.090	−0.220	−0.196	−0.316	−0.209
	P value	.429	.576	.168	.220	.044*	.190
ΔNeutrophil count (×10^9/L)	rho	0.001	0.150	−0.122	−0.010	−0.100	−0.136
	P value	.993	.349	.449	.948	.533	.397
ΔLymphocyte count (×10^9/L)	rho	−0.132	−0.200	−0.223	−0.281	−0.374	−0.186
	P value	.411	.210	.161	.075	.016*	.245
ΔMonocyte count (×10^9/L)	rho	0.034	−0.007	−0.218	−0.289	−0.374	−0.182
	P value	.834	.967	.172	.067	.016*	.254
ΔEosinophil count (×10^9/L)	rho	−0.165	−0.297	−0.323	−0.328	−0.360	−0.374
	P value	.303	.059	.039*	.036*	.021*	.016*
ΔBasophil count (×10^9/L)	rho	0.054	0.068	0.186	0.437	0.439	0.298
	P value	.737	.671	.243	.004*	.004*	.059
ΔPlatelet count (×10^9/L)	rho	−0.057	0.088	−0.081	−0.171	−0.199	−0.187
	P value	.726	.584	.616	.285	.213	.242
ΔMPV (fL)	rho	−0.108	−0.202	−0.218	−0.055	0.041	−0.120
	P value	.502	.205	.171	.731	.797	.457
ΔPDW (%)	rho	−0.141	−0.247	−0.252	−0.072	−0.021	−0.162
	P value	.380	.120	.112	.654	.897	.312

*Statistically significant ($P < .05$).
BOP, bleeding on probing; CAL, clinical attachment level; GI, Gingival Index; hsCRP, high-sensitivity C-reactive protein; MPV, mean platelet volume; PDW, platelet distribution width; PI, Plaque Index; PISA, periodontal inflamed surface area; PPD, probing pocket depth; TLC, total leukocyte count.

reducing hsCRP among systemically healthy individuals.[33] Due to the antibacterial and anti-inflammatory effect of systemic antibiotics, their utility in the secondary prevention of CVDs has been assessed in a recent meta-analysis.[34] However, antibiotics do not seem to be beneficial in combating risk associated with CVDs.[34]

Increased TLC, although within normal range, is associated with CVD.[35] Moreover, the increased TLC at or above 7.0×10^9/L as compared to TLC below 4.8×10^9/L predicts the risk for occurrence of CVD and its associated mortality.[36] Relatively higher TLC due to periodontitis,[28,37-39] although within normal range, may raise a concern for its impact on systemic health. In the present study, there was significantly higher mean TLC in periodontitis patients (6.62±1.67×10^9/L) as compared to PH individuals (5.58±1.52×10^9/L). Relatively higher TLC is attributed to an increase in neutrophil count among periodontitis patients as TLC are majorly constituted of neutrophils. In the present study, increased neutrophil and monocyte count, although within normal range, in periodontitis patients contributed to a comparative increase in TLC. In the current study, lymphocyte count did not contribute to any alteration in TLC as its level was similar in both periodontitis patients and PH individuals. In previous studies, increased neutrophil count[28,37-39] and increased lymphocyte count,[37] both within normal range, are responsible for a relative increase in TLC in periodontitis patients. However, no change in lymphocyte count[40] and decreased lymphocyte count[28,39] in periodontitis patients are also reported. The alterations in DLC may have important systemic health implications, as increased neutrophil count and monocyte count as well as decreased lymphocyte count are independent predictors for risk of CVD[7,41] and its associated mortality.[7] Effect of SI on TLC is equivocal with either similar TLC and neutrophil count[32] or a decrease in their count[42] from elevated levels. In the present study, TLC was not reduced at week 8 on treatment with either SI alone or SI with systemic antibiotics. However, treatment with SI and systemic antibiotics in previous studies presented conflicting results.[20,43] In one study, despite improved endothelial function and decrease in hsCRP, there was no effect of SI and systemic antibiotics on TLC.[20] From these findings, it is concluded that further studies with similar research methodology are needed to assess the effect of systemic antibiotics on TLC. In the present study, irrespective

Table 4 Multivariate linear regression analysis evaluating change (Δ) in hematologic parameters (dependent variable) based on improvement (Δ) in periodontal parameters (independent predictor variables) and treatment group (AB/SI)

				95% CI			
Blood parameters	Variable	B	SE	Lower	Upper	Partial η^2	*P* value
ΔTLC (× 10^9 L)	Intercept	1.378	0.678	0.002	2.755	0.106	.050
	ΔBOP	−0.006	0.022	−0.051	0.038	0.002	.770
	ΔPPD	2.958	1.398	0.121	5.796	0.113	.041*
	ΔCAL	-4.286	1.501	−7.333	−1.238	0.189	.007*
	ΔPISA	0.000	0.001	−0.001	0.002	0.008	.603
	AB (SI reference)	−0.285	0.456	−1.211	0.642	0.011	.537
ΔLymphocyte count (× 10^9 L)	Intercept	0.612	0.332	−0.062	1.286	0.088	.074
	ΔBOP	−0.010	0.011	−0.032	0.012	0.025	.353
	ΔPPD	0.979	0.684	−0.409	2.368	0.055	.161
	ΔCAL	−1.788	0.735	−3.280	−0.297	0.145	.020*
	ΔPISA	0.000	0.000	0.000	0.001	0.045	.207
	AB (SI reference)	−0.181	0.223	−0.635	0.272	0.018	.423
ΔMonocyte count (× 10^9 L)	Intercept	0.179	0.078	0.022	0.336	0.132	.027*
	ΔBOP	−0.002	0.002	−0.007	0.003	0.018	.424
	ΔPPD	0.053	0.160	−0.272	0.377	0.003	.743
	ΔCAL	−0.199	0.172	−0.547	0.150	0.037	.255
	ΔPISA	0.000	0.000	0.000	0.000	0.013	.505
	AB (SI reference)	0.014	0.052	−0.092	0.120	0.002	.784
ΔEosinophil count (× 10^9 L)	Intercept	0.104	0.077	−0.053	0.261	0.049	.187
	ΔBOP	−0.003	0.002	−0.008	0.002	0.030	.309
	ΔPPD	0.126	0.159	−0.197	0.449	0.018	.434
	ΔCAL	−0.182	0.171	−0.529	0.165	0.031	.295
	ΔPISA	0.000	0.000	0.000	0.000	0.016	.452
	AB (SI reference)	−0.037	0.052	−0.143	0.068	0.015	.476
ΔBasophil count (× 10^9 L)	Intercept	−0.016	0.007	−0.031	−0.001	0.122	.034*
	ΔBOP	0.000	0.000	−0.001	0.000	0.005	.692
	ΔPPD	−0.005	0.015	−0.036	0.027	0.002	.769
	ΔCAL	0.016	0.016	−0.017	0.050	0.027	.332
	ΔPISA	0.000	0.000	0.000	0.000	0.010	.549
	AB (SI reference)	0.004	0.005	−0.006	0.015	0.022	.381

*Statistically significant (*P*<.05).
BOP, bleeding on probing; CAL, clinical attachment level; η^2, eta-squared; PISA, periodontal inflamed surface area; PPD, probing pocket depth; TLC, total leukocyte count.

of treatment group (AB or SI group), improvement in PPD predicted a decrease in TLC, thereby implicating a positive effect of periodontal treatment on cardiovascular health. Treatment with locally delivered minocycline along with SI resulted in a significant decrease in TLC at week 8.[21] This may be due to the pronounced effect of local delivery of antibiotics in comparison with systemic antibiotics on diseased periodontal pockets.

In the present study, there was a significant decrease in neutrophil count in both the AB and SI groups. However, the improvement in neutrophil count was similar in both the treatment groups. Similar improvement in periodontal inflammation measured in terms of PISA may be responsible for comparable findings in both groups. A previous study reported a similar finding of reduction in neutrophil count on treatment with systemic antibiotics and SI.[43] Greater improvement in neutrophil count (16.87%) in a previous study compared to the AB group of the present study (13.28%) further suggests a significant decrease in TLC in the previous study.[43]

In the present study, similar eosinophil and basophil counts were found in periodontitis patients and PH individuals. This

finding is similar to the finding previously reported.[37] In the present study, change in lymphocyte count, monocyte count, eosinophil count, and basophil count at week 8 was not evident in both the groups. Similar results are reported after treatment with SI.[32] In the present study, despite no change in lymphocyte count at week 8, improvement in CAL due to SI with or without systemic antibiotics was found to be a negative predictor for change in lymphocyte count. This association might have contributed to negative predictability of improvement in CAL for change in TLC. The proportional increase in lymphocyte count due to improvement in CAL may have some implication in reducing risk for CVDs, but needs to be confirmed by conducting long-term follow-ups.

MPV and PDW are related to the degree of platelet activation. Due to the association of increased platelet activation with CVDs, it is intriguing to evaluate the impact of both treatment modalities on parameters of platelets. In the present study, platelet count, MPV, and PDW were similar in periodontitis patients and PH individuals. Previous study reported similar platelet count,[43,44] but decreased[43] to similar MPV[44] and increased PDW[44] in periodontitis patients compared with PH individuals. Contrary findings with similar platelet count[39,40] and MPV[39] as well as decreased platelet count[38] and decreased MPV[38] have been reported in patients with aggressive periodontitis as compared to PH individuals. SI has been reported to result in a decrease in platelet count in generalized aggressive periodontitis patients; however, its value at baseline was not compared with platelet count in PH individuals.[42] This finding[42] hints towards the importance of periodontal treatment, as increased platelet count is associated with increased incident for CVDs and its associated mortality.[9] However, in the present study, neither the AB group nor the SI group presented with a change in platelet count. Furthermore, there was no change in MPV or PDW in either treatment group. This finding is in contrast to a previous study that reported an increase in MPV at 1 month after treatment with SI and antibiotics in severe periodontitis patients, despite no change in platelet count.[43] Moreover, an increase in MPV is also correlated with improvement in PPD.[43] As association of increased MPV with risk for CVD is due to increased thrombotic potential of large-sized platelets, increase in MPV after periodontal therapy in severe periodontitis patients[43] represents a contradictory finding. Increased utilization of platelets in severe periodontitis is responsible for decreased MPV (although within normal range) in severe periodontitis, and this number increased within normal range after periodontal treatment.[43] Hence, increase in MPV beyond normal values may hold importance when comparing its association with both CVD and periodontitis rather than comparison within normal ranges.

As increased BMI is associated with risk for CVDs,[45] Comparable BMI among groups, strict well-defined inclusion and exclusion criteria, assessment of periodontal inflammation measured in terms of PISA as well as its correlation with systemic parameters, and consistency of blood collection with regards to fasting status, were the potential strengths of the present study. Dosage and duration of amoxicillin and metronidazole delivered to the AB group was based on meta-analysis.[46] The duration of combination therapy with amoxicillin and metronidazole for 7 days and 14 days was evaluated in this meta-analysis.[46] It was suggested that administration of antibiotics (400/500 mg or 500/500 mg combinations of amoxicillin and metronidazole, respectively) for 7 days seems appropriate as comparable improvement in periodontal parameters is observed in both regimens.

Post intervention time point of assessment at week 8 in the present study was based on the findings of continued periodontal healing till 8 weeks after nonsurgical periodontal intervention.[47] However, the impact of systemic antibiotics as an adjuvant to SI on systemic inflammation would merit evaluation at multiple timepoints so that immediate and long-term effects may be assessed. Another limitation of the study was noninclusion of microbiologic assessment in the study design. Longitudinal studies on patients with varying staging of periodontitis, assessment at multiple time points, and inclusion of other hard endpoints of CVD such as endothelial function, and carotid intima media thickness, would benefit the present study.

Conclusions

Within the limits of the present study, the following conclusions can be drawn. Irrespective of the adjunctive use of systemic antibiotics, change in hematologic parameters is accompanied by improvement in periodontal parameters. SI with or without antibiotics had a marked effect on the reduction of neutrophil count. Change in lymphocyte count is correlated with improvement in CAL. Therefore, systemic antibiotics did not have an additive beneficial effect on the reduction of systemic inflammation.

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The role of home care therapy in periodontal disease treatment and management

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Home care therapy is indispensable to manage periodontal disease successfully. Often, during and following initial periodontal treatment, it is unclear how much of the clinical improvement was due to patients' home care or to professional intervention, as these two therapeutic components are often amalgamated in clinical practice as well as in studies. In this case series, four patients with periodontal disease received education on using oral hygiene devices and used them competently prior to initiation of professional periodontal treatment. The changes in their clinical presentations, solely attributed to their home care therapy, were documented. The rationale and suggested clinical guidelines are also presented. **Conclusion:** Home care therapy is an indispensable but often overlooked step in the successful management of periodontal diseases. Ideally, this step should be solidified prior to proceeding with any professional treatment. By motivating patients to participate in the treatment more actively, clinicians can significantly improve the outcome and longevity of their professional interventions. *(Quintessence Int 2023;54:288–295; doi: 10.3290/j.qi.b3773959)*

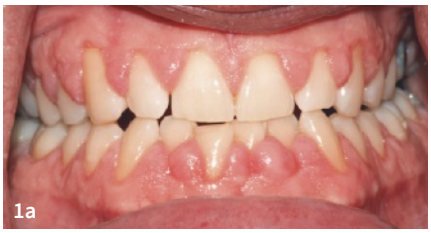
Key words: bone loss, caries, dental plaque, gingivitis, oral hygiene, periodontitis

Optimizing oral hygiene is an important component not only of phase I periodontal therapy but also of cause-related therapy.[1] Cause-related therapy is achieved by therapeutic interventions that suppresses the etiologic factors and by the constant review of home care therapy with the patient.[1,2] The primary etiology of the two most common diseases of the oral cavity, dental caries and periodontal disease, is plaque bacteria on a susceptible host.[3] In cases of periodontal disease, insufficient removal of dental plaque leads to a microbiologic shift of the predominant "red complex" bacteria triggering a pro-inflammatory cascade.[4-6] Consequently, irreversible periodontal attachment loss occurs.[5-7]

Optimal oral hygiene is not only a necessary component in cause-related therapy, but it is also prerequisite before performing periodontal surgeries. Undergoing scaling and root planing and oral hygiene instructions leads to a reduction in periodontal pathogens and a change in the microbiome.[8,9] This results in an improved oral environment preceding surgical intervention, leading to a more optimal healing response.[1,8,9] Furthermore, good oral hygiene is needed for the long-term success of periodontal treatment.[10,11] Unfortunately, a recent study reported that the oral hygiene phase was variable in length and content, variable in the consequential result, insufficiently instructed, and invariably amalgamated with the scaling and root planing, which is the intervention or part of an intervention.[12] Thus, the aim of the present case series was to report the improvement of periodontal conditions solely from patients' home care therapy prior to initiating any active professional intervention.

Case 1

A 46-year-old man was referred to the clinic for evaluation of gingival overgrowth and persistent halitosis. His medical history revealed hypertension for which he was taking a calcium channel blocker (nifedipine). A comprehensive periodontal evaluation revealed generalized marginal gingival erythema, gingival hyperplasia, periodontal probing depth of 3 to 7 mm, and gener-



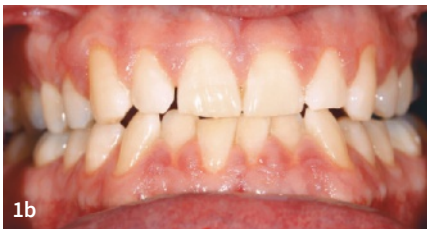


Fig 1a Initial presentation: Generalized marginal gingival erythema and gingival hyperplasia were noted, and they were more pronounced in the interproximal sites and the mandibular anterior sextant.

Fig 1b Two-week follow-up. Generalized reduction in marginal gingival erythema and gingival hyperplasia were noted at 2 weeks following home care therapy initiation.

Fig 1c 12-week follow-up: Further reduction in marginal gingival erythema and gingival hyperplasia were noted at 12 weeks. Mild gingival erythema and hyperplasia were noted on mandibular anterior sextant while a complete resolution was noted at the rest of the oral cavity.

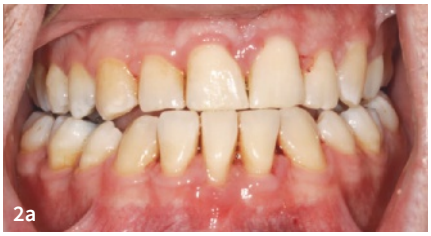
alized bleeding on probing, with no radiographic evidence of apparent alveolar bone loss. The gingival hyperplasia appeared more pronounced in interproximal sites and the mandibular anterior sextant (Fig 1a). There were moderate deposits of supragingival and subgingival dental plaque. His halitosis appeared to be associated with the chronic presence of dental plaque in the pseudo periodontal pockets.

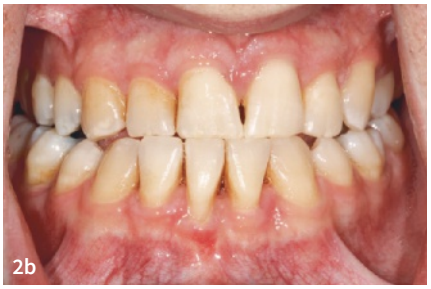
Periodontal diagnosis of generalized biofilm-induced gingivitis with generalized moderate drug-influenced gingival enlargements was made.[13,14]

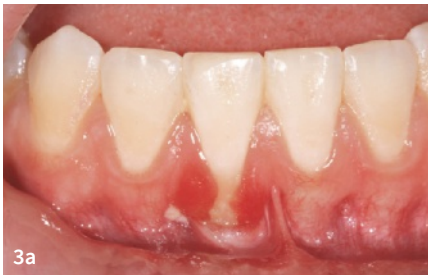
The patient was recommended intensive home care therapy in combination with repeated professional nonsurgical periodontal debridement, followed by reevaluation, and when appropriate, maintenance visits every 3 months. Medical consultation was conducted with his treating physician for switching his anti-hypertensive medication to another class, for which the physician agreed. He was prescribed an angiotensin II receptor antagonist (losartan), which was as effective as the calcium channel blocker in managing his hypertension. Prior to proceeding with nonsurgical periodontal debridement, an intensive home care therapy was executed. Based on the principles of cause-related therapy, the patient was specifically informed about the dental plaque as the primary etiologic factor for his periodontal disease. The modified bass technique was demonstrated. He was recommended to spend 4 seconds on each surface of the teeth (ie, buccal, lingual, and occlusal) throughout his oral cavity using an oscillating electric toothbrush twice daily. In addition to daily flossing, the patient was recommended to use a rubber tip stimulator three times a day. He was instructed to insert a rubber tip stimulator interproximally until resistance was felt, then massage the interproximal gingiva with circular motions applying firm apical pressure until his gingiva blanched. After 12 weeks of home care therapy (Figs 1b and 1c), a significant resolution of the marginal gingival erythema and gingival overgrowth was noted. Throughout this period, the patient was fully compliant with the suggested home care therapy with minimally visible residual dental plaque. Furthermore, significant reduction in probing depth was achieved, with all sites exhibiting 3 to 5 mm with minimal to no bleeding on probing.

Case 2

A 27-year-old man was referred to the clinic for evaluation of his periodontal condition. His medical history revealed schizophrenia for which he was taking haloperidol (Hadol decanoate), lithium, and quetiapine (Seroquel). A comprehensive periodontal evaluation revealed generalized marginal gingival erythema and gingival edema, which were more pronounced in the maxillary and mandibular anterior sextants (Fig 2a). There were generalized periodontal probing depths of 3 to 6 mm, with a localized probing depth of 9 mm on the mandibular right first molar, with generalized bleeding on probing, generalized mild horizontal bone loss, and localized vertical bone loss on the mandibular right first molar. There were moderate deposits of supragingival and subgingival dental plaque. Periodontal diagnosis of localized stage III grade C periodontitis with mucogingival defects, recession type 2 (RT2), was determined for the patient.[15,16]







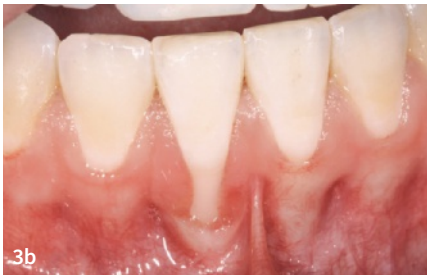


Fig 2a Generalized marginal gingival erythema and gingival edema were more pronounced in the maxillary and mandibular anterior sextants.

Fig 2b Generalized reduction in marginal gingival erythema and edema were noted at 2 weeks following home care therapy.

Fig 3a Marginal gingival erythema and gingival edema around the mandibular right central incisor were noted, with bleeding on probing and moderate deposits of supragingival and subgingival dental plaque. Furthermore, there was mucogingival deformity with lack of attached gingiva and buccal gingival recession.

Fig 3b A significant resolution of gingival erythema and edema were noted following 2 weeks of home care therapy. There was minimally visible dental plaque, suggesting effective plaque removal by the patient.

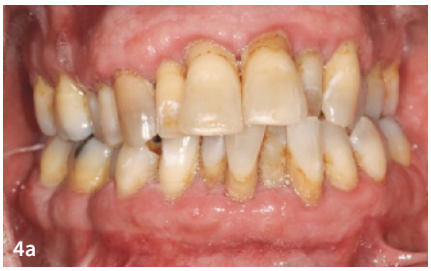
He was recommended intensive home care therapy in combination with scaling and root planing as part of the initial periodontal therapy. After explaining the importance of removing dental plaque, which was the main etiologic factor for his periodontal disease, home care therapy techniques were reviewed in a similar manner as described in Case 1. After 2 weeks of home care therapy, a significant resolution of marginal gingival erythema and gingival overgrowth was noted in the maxillary and mandibular anterior sextants (Fig 2b). Throughout this period, the patient was compliant with the suggested home care therapy, resulting in minimally visible residual dental plaque. Thereafter, the patient's periodontal disease was controlled and maintained through scaling and root planing, followed by reevaluation and surgical periodontal treatment and maintenance therapy.

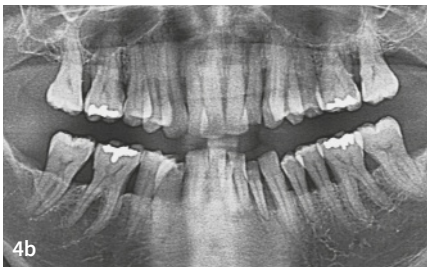
Case 3

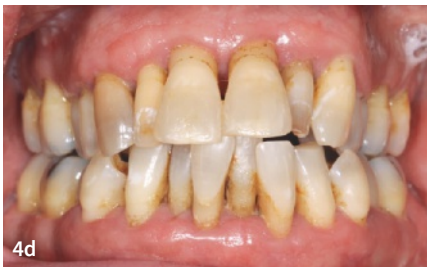
A 19-year-old woman was referred to the clinic for evaluation of her gingival recession on the mandibular right central incisor. Her medical history was noncontributory. A comprehensive periodontal evaluation revealed localized marginal gingival erythema and gingival edema around the mandibular right central incisor with periodontal probing depth of 4 to 5 mm, bleeding on probing, and moderate deposits of supragingival and subgingival dental plaque. Furthermore, the mandibular right central incisor exhibited 5 mm buccal gingival recession with a complete lack of attached gingiva (Fig 3a). Periodontal diagnosis of gingival health on a reduced periodontium with mucogingival deformity on the mandibular right central incisor, RT1, and lack of keratinized gingiva was made.[13,16]

The patient was recommended an intensive home care therapy regimen to significantly resolve the severe gingival inflammation around the mandibular right central incisor prior to proceeding with mucogingival surgery.

After explaining the importance of removing dental plaque, which was the main etiologic factor for her periodontal disease, home care therapy techniques were reviewed with an emphasis on performing gingival line toothbrushing around the receded marginal gingiva with her right hand while retracting her lower lip with her left hand. This allowed her to visualize and gain better access to the receded gingiva while brushing. After 2 weeks of home care therapy, a significant resolution of marginal gingival erythema and edema were noted in the mandibular right central incisor, with minimally visible residual dental plaque (Fig 3b). Thereafter, it was determined that she was ready to proceed with her gingival graft surgery to correct the mucogingival deformity on the mandibular right central incisor.







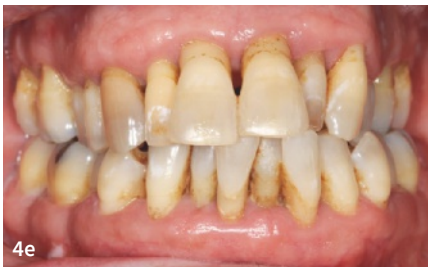


Fig 4a Generalized marginal gingival erythema and marginal gingival edema were noted. There were heavy deposits of supragingival and subgingival dental plaque.

Fig 4b Radiographically, there was generalized moderate to severe alveolar bone loss.

Fig 4c A reduction in gingival erythema and edema was noted 2 weeks after home care therapy initiation.

Fig 4d A continuous reduction in gingival erythema and edema was noted 5 weeks after home care therapy initiation.

Fig 4e A significant resolution of gingival erythema and edema was noted 9 weeks after home care therapy initiation. Although supragingival calculus was present, there were minimal deposits of soft dental plaque, suggesting effective home care by the patient.

Case 4

A 47-year-old man was referred to the clinic for evaluation of periodontal disease. His medical history was noncontributory. A full periodontal evaluation revealed generalized marginal gingival erythema, marginal gingival edema, periodontal probing depth of 5 to 9 mm, generalized bleeding on probing, and radiographic evidence of moderate to severe alveolar bone loss (Figs 4a and 4b). There were heavy deposits of supragingival and subgingival dental plaque. A periodontal diagnosis of generalized stage III, grade B periodontitis with mucogingival defects, RT 2, was made.[15,16]

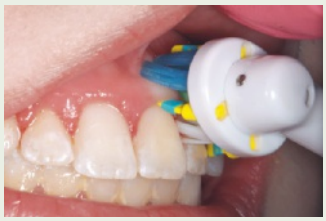
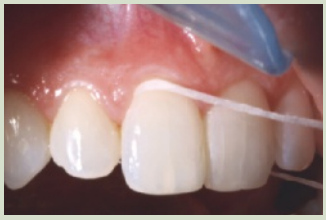
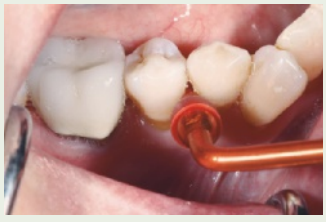
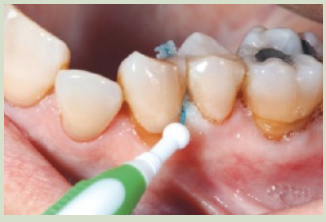
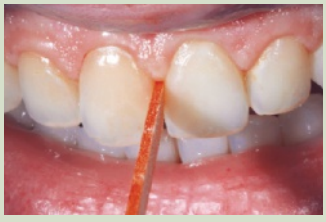
The patient was recommended initial periodontal therapy, including intensive home care therapy in combination with scaling and root planing. Home care therapy techniques were reviewed in a similar manner as described in Case 1. The patient was recalled for home care therapy review at 2 weeks, 5 weeks, and 9 weeks (Figs 4c to 4e). Throughout this period, a continuous reduction in marginal gingival erythema and edema were noted. The patient was fully compliant with the suggested home care therapy as evident by minimally visible dental plaque in 9 weeks. Thus, it was decided that adequate home care was achieved and that he was ready for scaling and root planing followed by reevaluation.

Discussion

Successful long-term management of periodontal disease requires behavioral changes of patients to attain and sustain a high level of daily plaque removal, life long.[17] Treating clinicians should educate and help their patients develop good oral hygiene habits, predominantly toothbrushing and interproximal cleaning, prior to initiating any professional intervention.[18] Not only does it lead to a more optimal healing response,[1,8,9] but it is also needed for long-term success of periodontal treatment and oral health.[10,11,19] Patients with poor oral hygiene exhibited further attachment loss regardless of receiving surgical or nonsurgical treatment.[20,21] Furthermore, maintaining low plaque levels may help reduce the severity and recurrence rate of gingival hyperplasia in patients with medication-induced gingival hyperplasia, such as in Case 1.[22]

The literature suggests that a power-driven toothbrush was more effective than manual toothbrush, especially in reducing Plaque Index and Gingival Index.[23-25] In addition, a recent meta-analysis suggested that an oscillating-rotating power-driven toothbrush was more effective than other electric toothbrushes as measured in whole-mouth Plaque Index, Interproximal Plaque Index, and the number of sites with bleeding.[26] For patients with periodontal disease, a common 2-minute brushing technique may not be long enough.[17]

Table 1 Oral hygiene recommended devices and the suggested techniques

Devices	Instructions	Notes and examples
Oscillating electric toothbrush	Place or park the brush head around the buccal surface of each tooth, leave it for 4 seconds, and move it to another tooth. Place the lower half of the brush head on the gingiva and the upper half in the cervical third of the tooth. When the buccal surfaces are completed, repeat the same process for the lingual surface of each tooth in the same manner. Lastly, place or park the brush head on the biting/occlusal surface of each tooth, apply apical pressure, leave the brush head for 4 seconds, move to another tooth, and repeat the same technique until completion of all occlusal surfaces.	Perform twice a day. Three time daily for severe periodontal patients or high caries risk patients. 
Floss	Once passing the interproximal contact, hug/wrap around the distal side of the anterior tooth, slide the floss apically until the floss disappears 2–3 mm subgingivally. Move the floss coronally and apically 2–3 times. Repeat the same on the mesial side of the posterior tooth. When completed, slide the floss out by pulling it buccally. Repeat the same technique for all interproximal contacts.	Perform twice a day. Suitable for patients with healthy periodontium or low caries risk as well as for patients with periodontitis, who have very high dexterity skills and motivation. 
Rubber tip stimulator	Place the pointy tip interproximally from the buccal side. Press lingually until the tip is fully engaging. Then, draw 5 circles especially with apical pressure until the gingiva blanches. Repeat the same technique for all interproximal surfaces. Once completed, repeat the same from the lingual side.	For patients with healthy periodontium or gingivitis, perform once a day. Excessive use may induce interproximal recession. For patients with periodontitis, perform 2–3 times a day. 
Interdental brush/triangular-shaped wooden toothpick	Place the brush/toothpick between the two teeth from the buccal aspect. Gently push and pull five times from the buccal to the lingual aspect. Ensure the brush/toothpick completely passes through the buccal embrasure to the lingual embrasure. For the toothpick, the apex or the tip of the triangle should be pointing occlusally while the base of the triangle should be in contact with interproximal papilla.	Perform twice a day. Suitable for patients with periodontitis, who have difficulty with effective flossing. Help patients identify the size that fits their embrasures.  

For interproximal cleaning, oral hygiene education should be tailored to the patient depending on the motivation level, dexterity, and anatomical factors such as embrasure size.[27] In general, for patients who are less motivated, who do not have good manual dexterity, or who have open interproximal embrasures, an interdental brush or a triangular-shaped wooden toothpick should be used instead of floss.[17,27] The interdental area can be further strengthened by means of gingival stimula-

Table 2 Commonly asked questions from patients and the suggested answers

Commonly asked questions	Suggested answers
"I noticed bleeding while performing my home care therapy. I stopped as I thought that I was damaging my gums making them bleed."	Bleeding is a reliable sign, suggesting the presence of inflammation and disease around your gums. With proper home care therapy techniques, healthy gums should not bleed.
	Thus, instead of avoiding home care in the bleeding sites, I want you to focus more on these sites while performing home care even if it bleeds. In 7–10 days, you will notice the resolution of bleeding in these sites, suggesting the resolution of inflammation or disease.
"How much pressure should I apply while using an electric toothbrush?"	Oscillating-rotating electric toothbrush already has a set torque. Thus, there is no need to apply too much pressure. Instead, park and hold the brush against the teeth/gums and let the brush head remove the plaque. Some electric toothbrush systems come with pressure sensitive heads while brushing that notifies you when you apply too much pressure, which may be helpful as well.
"I rinse my mouth multiple times a day; however, my mouth doesn't seem to be getting better."	Bacteria in the oral cavity live on your tooth surfaces, and are known as biofilm. The biofilm adheres to your teeth strongly. Thus, vigorous swishing or rinsing will not be effective enough to dislodge the biofilm. Instead, the biofilm needs to be mechanically dislodged or removed using toothbrush and interproximal cleaning tools. Furthermore, the antimicrobial ingredients in your mouthwash won't reach bacteria in biofilm effectively as the biofilm creates a wall or barrier through which the antimicrobial ingredients cannot effectively cross.
"I am afraid of brushing my gums, which can cause gum recession."	As long as you brush gently, but thoroughly, gum line brushing would not result in gingival recession. When instructed to brush gently, patients often brush less effectively around their gums, leaving dental plaque behind. The remanent of dental plaque can initiate gum disease, which can result in a further initiation or worsening of gingival recession. Thus, please hold your toothbrush lightly and use a repeated circular motion over the gum line. This would ensure a more effective removal of the dental plaque, which will help you maintain healthier gum.
"I cannot floss well. Is there any other tool that is easier to use on my hands?"	Interdental brush or a triangular-shaped wooden toothpick can be used instead of floss. It can be as effective as floss or, in certain cases, even better in removing dental plaque between the teeth. Patients with limited dexterity may find it easier to use an interproximal brush or a triangular-shaped wooden toothpick than floss.
"Should I use an electric or manual toothbrush? If so, which kind?"	The literature suggest that an electric toothbrush is more effective than a manual toothbrush in removing dental plaque. Particularly, an oscillating-rotating electric toothbrush is more effective than other electric toothbrushes in removing dental plaque and reducing gingival bleeding.

tion using a device such as rubber tip stimulator. The gingival stimulation may help maintain adequate blood circulation and produce surface keratinization.[28,29] Care should be taken for patients without periodontitis as a prolonged or forceful gingival stimulation can result in interproximal soft tissue recession.[28]

Table 1 presents the recommended oral hygiene devices and the suggested techniques.

During patient education, clinicians should encourage them to participate actively. This can be achieved by demonstrating the home care techniques to patients using various aids such as a teeth model or a video, letting patients demonstrate their techniques back to the clinicians for calibration, and repeating these processes in several visits.[1,2,30] Furthermore, instead of using the terminology "oral hygiene instruction," clinicians are suggested to use a more active terminology such as "home care therapy." This would help patients understand that what they perform at home is indeed therapeutic in nature and encourage them to take a more active role in the management of their periodontal diseases.[31] Table 2 presents commonly asked questions by patients and the suggested answers, which clinicians can utilize for establishing more effective communication with their patients.

Often, home care therapy is done simultaneously with scaling and root planing in clinical trials or clinical settings.[12] This unfortunately makes it difficult to isolate the magnitude of clinical improvement solely attributed to home care therapy.

In the present case series, all of the noted clinical improvements were strictly from patients' home care therapy without any professional intervention.

Furthermore, completing home care therapy prior to active scaling and root planing can significantly reduce gingival erythema, edema, bleeding, and potentially periodontal pocket depths, all of which can make the professional debridement more effective, easier, and with less side effects.[32-34] The advantage of completing home care therapy prior to scaling and root planing was particularly evident in Case 4.

Conclusion

Home care therapy is an indispensable but often overlooked step in the successful management of periodontal diseases. Ideally, this step should be solidified prior to proceeding with any professional treatment. By motivating patients to participate in the treatment more actively, clinicians can significantly improve the outcome and longevity of their professional interventions.

Disclosure

The authors declare no conflict of interest.

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