The adjunctive use of systemic antioxidant therapy (lycopene) in nonsurgical treatment of chronic periodontitis: A short-term evaluation

Nupur Arora, MDS1/Haritha Avula, MDS2/Jaya Kumar Avula, MDS3

Objective: To evaluate the efficacy of systemic lycopene along with routine scaling and root planing in terms of changes in clinical parameters and levels of circulating tumor OFDSPTJTGBDUPSBMQIB	5/'
TBMJWBSZJOUFSMFVLJOøCFUB	*-
, and uric acid in chronic periodontitis. Method and Materials: Forty-two systemically healthy subjects with chronic periodontitis were included in a randomized, placebo-controlled, parallel design, double-blinded trial. The subjects were randomly distributed between the two treatment groups: test group (n = 21) 8 mg lycopene/day and placebo group (n = 21) along with adjunctive scaling and root planing. Patients were monitored at baseline and at 2 months after therapy. Periodontal parameters, namely plaque index (PI), modified gingival index (MGI), bleeding on probing (BOP), clinical attachment level (CAL) gain, and probing pocket depth (PPD) reduction, were evaluated and peripheral blood samples and whole saliva were obtained at these points of time to measure the levels of IL-1β, TNF-α, and uric acid using commercially available kits. Results: Test group (lycopene) showed better results after therapy compared to the placebo group with reference to PI (P = .004), MGI (P = .002), BOP (P = .021), salivary IL-1β (P = .05), and uric acid levels (P = .02). The CAL gain, PPD reduction and serum TNF-α value were not statistically significant but showed an improvement compared to the placebo group. Conclusion: Further longitudinal studies are required to establish the role of lycopene in the management of chronic periodontitis. (Quintessence Int 2013;44:395–405; doi: 10.3290/j.qi.a29188)

Key words: antioxidants, chronic periodontitis, clinical trial, lycopene

Various models have tried to explain the pathogenesis of periodontal disease and one among them is the role of reactive oxygen species (ROS).1 Tissue damage by ROS has been attributed to various mechanisms including DNA damage, lipid peroxidation, protein damage, oxidation of important enzymes, and stimulation of pro-inflammatory cytokine release,2 and therefore any imbalance between oxidative stress induced by ROS and the concentrations of the antioxidant may result in tissue damage.3

The role of dietary antioxidants in disease prevention has received much attention in recent years. Research has shown that diets rich in antioxidant micronutrients are anti-inflammatory.4 It has been suggested that boosting antioxidant micronutrient status of chronic periodontitis patients may have adjunctive therapeutic benefit.5 Several studies have demonstrated that patients with periodontitis have significantly lower serum antioxidant levels compared to healthy controls.6,8 While nonsurgical periodontal therapy by itself can reverse this trend and partly restore antioxidant levels,9,10 such therapy alone may not have the desired benefit. It has been suggested that phytonutritional interventions in the form of antioxidant supplementation may have a beneficial effect through direct scavenging of ROS as well as modulation of redox-sensitive proinflammatory gene transcription factors.11 Lycopene is one such dietary antioxidant providing defense against oxidation. It

1Senior Lecturer, Department of Periodontics, Sri Sai College of Dental Surgery, Vikarabad, Andhra Pradesh, India.
2Professor, Department of Periodontics, Sri Sai College of Dental Surgery, Vikarabad, Andhra Pradesh, India.
3Professor, Department of Periodontics, Sri Sai College of Dental Surgery, Vikarabad, Andhra Pradesh, India.

Correspondence: Dr Nupur Arora, Department of Periodontics, Sri Sai College of Dental Surgery, Vikarabad- 501101, Andhra Pradesh, India. Email: drnupurarora@gmail.com
gives tomato its bright red color. It is a highly unsaturated hydrocarbon containing 11 conjugated and 2 unconjugated double bonds.12 The singlet oxygen quenching capacity is 2 to 10 times higher than that offered by other naturally occurring antioxidants.12 Lycopene is also known to reverse the DNA damage induced by H2O2.13 Serum and tissue lycopene levels are inversely related to chronic disease risk.14 Apparently, lycopene reduces risk of cardiovascular disease,15,17 cancer,18 osteoporosis,19 and in some cases, even male infertility.20

Using data provided by the Third National Health and Nutrition Examination Survey (NHANES III), Wood and Johnson21 concluded that “a relationship exists between periodontitis and congestive heart failure risk, and high monthly tomato consumption appears to affect this relationship in a positive direction in periodontitis subjects.”21 An additional beneficial effect from continued dietary supplementation of lycopene along with scaling and root planing (SRP) in chronic periodontitis patients may confer a decreased risk of coronary heart disease.

The aim of this preliminary, short-term investigative study was to ascertain whether or not daily dietary supplementation for two months of commercially available lycopene, adjunctively used with mechanical nonsurgical periodontal therapy in the form of SRP, improved the clinical and immunological parameters in chronic periodontitis. It is proposed to evaluate the effect of clinical intervention by assessment of clinical parameters and levels of proinflammatory cytokines (interleukin 1 beta [IL-1β] and tumor necrosis factor alpha [TNF-α]). Uric acid has an important role as an oxidative stress marker and it is proposed to estimate the salivary uric acid levels before and after such an intervention.

**METHOD AND MATERIALS**

The present study was a randomized, placebo-controlled, two-arm, double-blinded trial conducted during April 2010 to October 2010. The protocol was approved by the Institutional Ethical Committee of Sri Sai College of Dental Surgery, Vikarabad, Andhra Pradesh, India. The study was performed in accordance with the Helsinki declaration of 1975, as revised in 2000, and adhered to the guidelines of Good Clinical Practice.

**Subject population**

The study population consisted of 46 chronic periodontitis patients (23 men and 23 women; age range 25 to 52 years) attending the Department of Periodontics, Sri Sai College of Dental Surgery. Sample size was not based on formal power calculation due to the absence of available reference data at the start of the study.

Chronic periodontitis was defined as presence of at least 4 teeth with probing pocket depth (PPD) > 4 mm, clinical attachment loss (CAL) ≥ 2 mm, and radiographic evidence of alveolar bone loss.22 Participants were enrolled for the study if they had ≥ 12 scorable teeth (not including third molars and teeth with orthodontic appliances, fixed dental prostheses, crowns, or implants).

Medical history of the subjects was ascertained at the time of recruitment through a questionnaire. Among the exclusion factors were systemic illnesses and chronic inflammatory conditions like diabetes mellitus, hypertension, rheumatoid arthritis, obesity, and chronic obstructive pulmonary disease. In addition pregnant/lactating subjects or women who intended to become pregnant during the study period were not included. Also, smokers, subjects on antibiotics, anti-inflammatory medications, or over-the-counter antioxidants such as vitamin C, vitamin E, or β-carotene within the previous 3 months, and those with a history of periodontal treatment during 3 months prior to the study recruitment were excluded.

Participants who fulfilled the above criteria were enrolled in the study after informed consent. Patients presenting with any other emergency dental needs were attended to before the commencement of the study. Comprehensive dental or further periodontal care was deferred until completion of the study. Patients were requested to refrain from consumption of any other foods, fruits, or vegetables that are known to be rich in antioxidants, as listed in their instruction manual.
Examiner calibration
Various parameters were clinically assessed by a calibrated periodontist (NA) at baseline and at the end of the study period (2 months). Examiner calibration was conducted in six subjects with clinical conditions similar to study subjects. It involved PPD and CAL measurements (two examinations, with an interval of 48 hours). Calibration was judged reproducible when measurements at baseline and at 48 hours were within 1 mm, 95% of the time.

Outcome measures
The primary outcome measure was mean reduction in salivary IL-1β levels 2 months post-therapy. Secondary outcome measures were reduction in bleeding on probing, Plaque Index (PI), Modified Gingival Index (MGI), serum TNF-α levels, mean reductions in PPD, and mean increase in CAL and salivary uric acid levels 2 months post-therapy.

Full mouth PPD and CAL were assessed using a UNC-15 probe (Hu-Friedy) at 6 sites around the teeth. Radiographic examination of teeth to confirm the diagnosis of chronic periodontitis was done using conventional intraoral periapical radiographs.

Whole unstimulated saliva (3 ml) was collected 2 hours after breakfast from all the subjects in sterile containers. The saliva samples were centrifuged at 3,000 rpm for 5 minutes. The supernatant fraction was then aliquoted into storage vials and stored at -70 °C until analyzed.

Venous blood was collected after overnight fasting using a 20-gauge needle with 5-ml syringes. Approximately 3 ml of blood was collected from the antecubital fossa by venipuncture into a noncoated vacutainer. Collected samples were allowed to clot at room temperature; serum was separated from blood by centrifuging at 1,000 x g for 15 minutes and immediately transferred to a plastic vial and stored at -70 °C until assayed.

Nature of supplement and placebo
The commercially available lycopene used in the study was in the form of 2-mg softgels (LycoRed, Jagsonpal Pharmaceuticals).

Test drug consisted of lycopene 2,000 μg, vitamin A 2,500 IU, β-tocopherol acetate 10 IU, vitamin C 50 mg, zinc sulfate monohydrate 27.45 mg, selenium dioxide 70 μg, soybean oil, bees wax, hydrogenated castor oil, butylated hydroxytoluene, butylated hydroxyanisole, soy lecithin, citric acid, sodium citrate, and simethicone. The placebo comprised identical looking softgels, containing pharmaceutically ineffective fillers.

Study procedure
Randomization was performed by the statistician (Dr Vishnu Vardhan Rao) who was independent of the clinical study team. A computer sequence of numbers was generated and 46 patients were allotted to two groups, ie the lycopene (test) group and the placebo group. All the subjects and the investigator were blinded to the nature of allotment, ensuring double blindness.

The computer-generated code was maintained by one of the investigators (HA). Uncoding was not done until all data had been collected. Recording of clinical parameters, collection of salivary samples and delivery of oral hygiene instructions to the patients was scheduled in the week prior to the baseline visit. In these sessions, explanations were offered about brushing techniques, and interdental cleaning with dental floss and interdental toothbrushes. At the baseline visit, patients were asked to fast overnight and blood samples were collected. Full mouth SRP with hand (Hu-Friedy) and ultrasonic (Electro Medical Systems) instruments was performed by one of the investigators (NA) at this point of time. The patient was then given a sealed bottle containing either the test drug or placebo, which were identical in appearance.

Patients were asked to take two test/placebo softgels twice a day (4 capsules per day) for 15 days. Every 15 days, patients were recalled and questioned about compliance, and oral hygiene instruction was reinforced. At every such visit, the test/placebo softgels were replenished until the completion of the study.

The bottle of drugs was physically checked to ascertain drug compliance. The patients were also instructed to maintain a diary of their daily intake of the softgels pro-
vided and also to chronicle any adverse events following the intake of the provided medication.

**Estimation of immunological parameters**

The salivary IL-1β and serum TNF-α were assayed pre- and postintervention employing an enzyme-linked immunoabsorbent assay (ELISA) kit (Transasia Bio-Medicals) according to manufacturer’s instructions. The assay employed the quantitative sandwich enzyme immunoassay technique. A streptavidin-coated plate was incubated with a biotinylated monoclonal antibody specific for IL-1β and TNF-α respectively. Pretreated standards and samples were added to the wells. Any IL-1β and TNF-α present was bound by the immobilized biotinylated antibody. After washing away the unbound substances, an enzyme-linked conjugate specific for IL-1β/TNF-α was added to the wells. After a wash to remove any unbound conjugate, a substrate solution was added to the wells, and color developed in proportion to the amount of IL-1β/TNF-α bound. The reaction was stopped by 2 mol/l sulfuric acid, and the absorbance was measured using 450 nm as the primary wavelength. Concentrations of IL-1β/TNF-α in the tested samples were estimated using the reference calibrated standard curve.

The principle for the determination of salivary uric acid levels was introduced by Trivedi et al and Kabasakalian et al with a modified Trinder peroxidase method using 2,4,6-tribromo-3 hydroxy benzoic acid (TBHB). In this assay, uric acid was transformed from uricase into allantoin and hydrogen peroxide, which, under catalytic influence of peroxidase, oxidized the chromogen (4-aminoantipyrine), to form a red compound with an intensity of color proportional to the amount of uric acid present in the sample.

**Statistical analysis**

The treatment allocation was unblinded after the completion of the clinical trial and prior to statistical analysis. SPSS 15.0 for Windows (SPSS) was used for the data analysis. Clinical data pertaining to baseline and posttreatment analysis of various parameters were expressed as mean ± standard deviation (SD). Intragroup and intergroup differences were analyzed using paired t test and the Student’s independent t test respectively. As there was a statistical significance difference in the baseline PPD values between the groups, analysis of covariance (ANCOVA) was performed taking the baseline PPD value as a cofactor.

**RESULTS**

The study incorporated an active intervention program for 2 months with either lycopene (8 mg/day) or placebo. Two patients each in the test and placebo groups were lost to follow-up and finally data on 21 patients in each group were available for analysis (Fig 1). The reasons for the withdrawal from the study were that the patients either could not afford time due to their job schedules, or had difficulty in keeping appointments due to domestic compulsions. The two groups evenly matched with reference to age and sex; 11 men and 10 women constituted the test group (mean age 35.40 years) and 10 men and 11 women constituted the control group (mean age 36.0 years).

**Adherence/safety outcomes**

The supplement diaries and capsule counts indicated that protocol adherence had been equivalent in both the groups. No adverse events or discontinuations owing to adverse events were reported in either treatment group during this 2-month interventional trial.

**Baseline data**

A study of baseline data indicates that both the groups showed similar oral hygiene and periodontal disease status at the time of recruitment, except that PPD was marginally higher in the placebo group and this discrepancy was statistically remedied (Table 1).

**Post-intervention data**

Both the groups showed highly significant improvements in all the parameters, confirming the pivotal role of SRP in periodontal therapy.
On intergroup comparison (Table 2), there was a significant improvement in the primary outcome in the test group compared to placebo group. Salivary IL-1β levels showed a reduction from 864.38 ± 130.09 pg/ml to 426.67 ± 108.50 pg/ml with a mean reduction of 437.71 ± 84.59 pg/ml in the test group. The corresponding reduction in the placebo group was 377.62 ± 111.03 pg/ml. This improvement in IL-1β levels was statistically significant.

Among the secondary outcomes, bleeding on probing showed a mean reduction of 1.12 ± 0.138 compared to 1.02 ± 0.144 in the placebo group, and this difference between the groups was statistically significant.

PI showed a mean reduction of 1.24 ± 0.167 in test and 1.09 ± 0.137 in placebo group, and for MGI the mean reduction was 1.49 ± 0.163 in the test and
In the placebo group, which was statistically significant (Figs 2 and 3).

Mean PPD reduction was 1.90 ± 0.625 mm in the test group and an identical improvement of 1.90 ± 0.301 mm was observed in the placebo group; this was not statistically significant.

Mean CAL gain was 1.62 ± 0.498 mm in the test group and 1.33 ± 0.483 mm in the placebo group, which indicated a marginal improvement although not statistically significant. Mean serum TNF-α reduction from 3.57 ± 0.476 to 2.24 ± 0.406 pg/ml in the test group and 3.55 ± 0.467 to 2.28 ± 0.424 pg/ml in the placebo group was statistically insignificant.

Salivary uric acid levels, which are indicative of efficacy of antioxidant intervention, showed statistically significant improvement in the test group (-1.23 ± 0.295 mg/dl) compared to the placebo group (-0.98 ± 0.376 mg/dl).

**DISCUSSION**

Excessive production of ROS resulting in oxidative stress has been implicated in the pathogenesis of many human diseases including periodontitis. The highly toxic and destructive nature of ROS may represent an important pathogenic mechanism for tissue damage that is seen in periodontitis. Various researchers have suggested antioxidant therapy as a possible strategy to combat periodontal disease.

Lycopene (β,γ-carotene), the most abundant carotenoid in tomatoes, is an

**Table 1** Baseline parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Test group (mean ± SD)</th>
<th>Placebo group (mean ± SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PI</td>
<td>1.98 ± 0.124</td>
<td>1.92 ± 0.116</td>
<td>NS</td>
</tr>
<tr>
<td>MGI</td>
<td>2.24 ± 0.114</td>
<td>2.22 ± 0.131</td>
<td>NS</td>
</tr>
<tr>
<td>BOP</td>
<td>1.74 ± 0.156</td>
<td>1.77 ± 0.153</td>
<td>NS</td>
</tr>
<tr>
<td>Mean PPD (mm)</td>
<td>7.00 ± 0.949</td>
<td>7.67 ± 0.577</td>
<td>.009*</td>
</tr>
<tr>
<td>Mean CAL (mm)</td>
<td>6.48 ± 0.928</td>
<td>6.48 ± 0.602</td>
<td>NS</td>
</tr>
<tr>
<td>IL-1β (pg/ml)</td>
<td>864.38 ± 130.09</td>
<td>869.05 ± 88.31</td>
<td>NS</td>
</tr>
<tr>
<td>TNF-α (pg/ml)</td>
<td>3.57 ± 0.476</td>
<td>3.55 ± 0.467</td>
<td>NS</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>3.65 ± 0.352</td>
<td>3.75 ± 0.481</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Statistically significant (P ≤ .05); NS, nonsignificant; SD, standard deviation.

**Table 2** Intergroup evaluation (posttreatment)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Test group (mean difference ± SD)</th>
<th>Placebo group (mean difference ± SD)</th>
<th>P value</th>
<th>F value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PI</td>
<td>1.24 ± 0.167</td>
<td>1.09 ± 0.137</td>
<td>.004*</td>
<td></td>
</tr>
<tr>
<td>MGI</td>
<td>1.49 ± 0.163</td>
<td>1.34 ± 0.126</td>
<td>.02*</td>
<td></td>
</tr>
<tr>
<td>BOP</td>
<td>1.12 ± 0.138</td>
<td>1.02 ± 0.144</td>
<td>.021*</td>
<td></td>
</tr>
<tr>
<td>PPD reduction (mm)</td>
<td>1.90 ± 0.625</td>
<td>1.90 ± 0.301</td>
<td>NS</td>
<td>8.069 (NS)</td>
</tr>
<tr>
<td>CAL gain (mm)</td>
<td>1.62 ± 0.498</td>
<td>1.33 ± 0.483</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Salivary IL-1β (pg/ml)</td>
<td>437.71 ± 84.59</td>
<td>377.62 ± 111.03</td>
<td>.05*</td>
<td></td>
</tr>
<tr>
<td>Serum TNF-α (pg/ml)</td>
<td>1.32 ± 0.234</td>
<td>1.26 ± 0.390</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Salivary uric acid (mg/dl)</td>
<td>-1.23 ± 0.295</td>
<td>-0.98 ± 0.376</td>
<td>.02*</td>
<td></td>
</tr>
</tbody>
</table>

*Statistically significant (P ≤ .05); F value, analysis of variance; NS, nonsignificant; SD, standard deviation.
effective natural antioxidant. It is also reported to positively affect the immune system by anti-apoptotic effect, and enhancement of phagocytic and bacterial killing activity of neutrophilic cells. Lycopene stands as the most potent antioxidant among various carotenoids and its antioxidant capacity is relatively high compared with other carotenoids like α-tocopherol, α-carotene, β-carotene, and lutein. However, it is pertinent to state that these carotenoids and other plant compounds have developed sets of interacting elements and this complexity of arrangement limits their individual identity.

There is controversy in the literature on the health benefits of lycopene alone and the majority of the studies have been conducted using tomato-based foods or tomato extracts but not with pure lycopene. Fuhrman et al reported tomato oleoresin exhibited around a five-fold superior capacity to inhibit low-density lipoprotein (LDL) oxidation in comparison to pure lycopene and this was proved later in vivo.

Measuring the scavenging capacity of lycopene, vitamin C, vitamin E, and β-carotene alone and in different combinations, Liu et al reported that the combination of them was substantially superior to the sum of the individual effects. There might also be lycopene-carotenoid interaction in biological systems. The combination of lycopene and other carotenoids was reported to be most effective towards ROS-mediated injury.

Pure lycopene supplementation alone in humans did not modify LDL susceptibility to oxidative and plasma malondialdehyde (MDA) and hydroxynonenal (HNE) levels, and also there was no effect on urinary F2-isoprostane levels. It was suggested that the combined carotenoids rather than purified lycopene may be beneficial in protecting against lipid oxidation.

Nonetheless, it is also a known fact that of all the ingredients, lycopene is the most potent antioxidant, with singlet oxygen quenching ability twice as high as that of β-carotene and 10 times higher than that of...
α-tocopherol.\textsuperscript{14} Based on this information a commercial preparation of lycopene with added phytonutrients was chosen for the present study.

The aim of the present study was to evaluate the efficacy of systemically administered lycopene as an adjunct to SRP in terms of changes in the levels of selected biomarkers over 2 months. A short-term evaluation period of 2 months was selected for the present study as adopted in other interventional studies assessing the efficacy of various adjunctive treatment modalities to initial nonsurgical periodontal therapy (NSPT).\textsuperscript{38-40} It has also been reported that the greatest change in probing depth reduction and gain in clinical attachment post-SRP occurs within 1 to 3 months.\textsuperscript{41} Evaluation of the results of the present study after 2 months of intervention appears rational, although a longer follow-up could have resulted in more definitive results.

The improvement in gingival status in the present study is highly significant in both groups following therapy; this is expected, as SRP is one of the standard means of controlling inflammation.\textsuperscript{42} The improvement in the test group was statistically significant ($P = .002$) compared to that in the placebo group (Table 2). Supplementation with lycopene seems to have augmented the healing sequence of inflamed gingival tissues.

Both the groups showed improvement in bleeding on probing. However, this was better in the test group compared to the placebo group (Table 2). This improvement in bleeding on probing is a natural corollary of healing tissues and reflects the contribution of lycopene in complementing the role of SRP.

There was an improvement in the mean PPDs in both the groups compared to baseline levels, but the difference between the groups was not statistically significant. Improvement in BOP and lack of statistical significance of differences in probing depths are at variance with the results of a recent study where fruit/vegetable or fruit/vegetable/berry concentrate was used as an adjunct to mechanical NSPT.\textsuperscript{11} The authors showed an additional improvement in PPD in the supplementation group compared to the placebo. However, comparison can only be made to a limited extent as a different product was used and the follow-up was at 2, 5, and 8 months. Their study further reported that the initial greater pocket depth reduction in the fruit/vegetable group was not sustained at 8 months post-therapy.

In the present study, CAL gain, which is a true and predictable parameter of tissue repair, did not show significant improvement in the test group. The study by Chapple et al\textsuperscript{11} did show a significant CAL gain but the differences in improvement within supplement groups compared to the placebo group did not show statistical significance.

Apart from examining the clinical parameters, the present study has introduced additional refinements, ie assessment of inflammatory markers (IL-1β, TNF-α) and uric acid, which is considered an indicator of antioxidant status.

Salivary IL-1β levels have been suggested as one of the biomarkers of periodontal disease. Miller et al\textsuperscript{43} reported mean salivary levels of IL-1β that were 3.5 times higher in periodontitis cases compared to non-periodontitis subjects. Salivary IL-1β levels in both test and placebo groups in the present study showed reduction from baseline values but the test group showed a statistically significant reduction ($P = .05$) compared to controls (Table 2). The significance of salivary IL-1β levels in chronic periodontitis and their reduction after phase I therapy has also been reported in another study.\textsuperscript{44} The choice of salivary IL-1β in the present study seems to be justified in view of the above.

With reference to the serum levels of TNF-α following lycopene and related supplementation, Riso et al\textsuperscript{45} reported a 34% decrease in TNF-α production in healthy volunteers after consumption of a tomato-based drink over a period of 26 days. The present study, however, did not reveal a significant reduction in TNF-α levels following lycopene supplementation. It is pertinent to note here that the study by Riso et al\textsuperscript{45} involved healthy volunteers whereas the present study was done on chronic periodontitis patients. This variation in subject population would have influenced the TNF-α levels to a certain extent.
Salivary uric acid levels rather than the estimation of total antioxidant power (TAP) were evaluated in the present study. Uric acid has an important role as an oxidative stress marker and has a potential therapeutic role as an antioxidant. Investigation of individual antioxidant is more often done than TAP. Urate constitutes up to 65% of total radical-trapping antioxidant parameter (TRAP) and could be relied upon as an indicator of antioxidant activity. Comparable ease and freedom from variation, and uninfluenced by salivary flow rate, uric acid lends itself as a dependable marker of antioxidant activity. Accordingly, in the present study, the response to antioxidant therapy was correlated to the salivary uric acid levels; the uric acid levels pre- and postantioxidant therapy showed a statistically significant improvement in the test group compared to placebo group (Table 2).

Understanding and objectively assessing the role of clinical and inflammatory markers in lycopene’s modulation of the immune system might positively influence decisions as far as dietary supplementation of antioxidants in the management of chronic periodontitis is concerned.

The present study being a preliminary investigation has certain limitations like small sample size and a short duration of observation/supplementation. Further long-term interventional studies are needed if lycopene supplementation can be considered as an adjunct to NSPT.

CONCLUSION

The results of the present study indicate that SRP augmented with concomitant use of antioxidant supplementation resulted in improved periodontal parameters PI, MGI, and BOP, but not the other parameters like mean PPD reduction and CAL gain. Salivary uric acid levels, which were initially low in subjects with chronic periodontitis, increased to statistically significant levels and this coupled with decreased levels of salivary IL-1β encourages a rational use of such supplementation in the management of periodontal disease. Further longitudinal studies are required to establish the role of lycopene supplementation in the management of chronic periodontitis.

ACKNOWLEDGMENTS

The authors thank Jagsonpal Pharmaceuticals, especially Miss Deepika Chabbra for her constant help in providing the test and placebo medicines and investigative kits. The authors are grateful to Dr Shanthi Naidu, Head, Department of Laboratory Medicine, Care Hospital, Banjara Hills, Hyderabad, Andhra Pradesh, for technical assistance and support in the ELISA procedure. The authors also thank Dr Vishnu Vardhan Rao for statistical assistance.

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