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Original Research Article

Comparative evaluation of non enzymatic antioxidants, vitamin C and albumin in patients suffering from different grades of periodontitis: a retrospective observational study

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ABSTRACT

Background: Rate of degree of destruction of periodontium may be associated with the oxidative stress due to the imbalance of different serum antioxidants. The objective of this study is to compare the evaluated concentrations of non enzymatic antioxidants like vit-C, albumin levels and total antioxidant status, from the available data of patients with slowly and rapidly progressive periodontitis (RPP).
Methods: This retrospective observational study was confined to a study population, which include; 42 patients with RPP, 42 patients with slowly progressive periodontitis (SPP) and 86 healthy controls, in whom the required data were available. The levels of non enzymatic antioxidants like albumin, vitamin C and total antioxidant capacity (TAC) of patients, suffering from RPP, are compared with SPP and healthy controls.
Results: The values of serum albumin and vitamin-c levels in RPP even remaining within the normal ranges were found to be on the lower side, when compared with healthy controls. The systemic TAC levels were found to be at a lower side in both SPP and RPP, when compared with the healthy control group.
Conclusions: The systemic levels of non enzymatic antioxidants, vit C and albumin, in RPP were found to be on the lower side, even remaining within the normal ranges, when compared with the healthy controls. The TAC in SPP and RPP is also found to be on lower side, consistent with previous studies.

Keywords: Antioxidants, Periodontitis, Serum

INTRODUCTION

The initial stage of periodontal infection is found in about 50-90% of the global population. Periodontal disease is characterized by the gradual inflammatory destruction of tooth-supporting hard and soft tissues, initiated due to infection of specific bacteria within the plaque biofilm. The progression of periodontal disease depends on complex interactions of the host response to the periodontopathic bacteria.[1] Normal cellular metabolic response during inflammatory destruction of periodontium includes generation of reactive oxygen species (ROS), those include oxygen-derived free radicals like superoxide (O_2^-), hydroxyl (OH^-), nitric oxide (NO^-), and non-radical derivatives of oxygen like hydrogen peroxide (H_2O_2) and hypochlorous acid (HOCL). ROS are highly toxic against the invading pathogens, playing the important role in the pathogenic mechanism, associated with phagocytic activity of defense cells during the elaboration of host defense mechanisms. At the same time they are also responsible

to impair a wide variety of intracellular molecules like lipids, proteins and DNAs of the host.[2-6]

ROS are produced in different body metabolic and physiological processes. When harmful oxidative reactions gets triggered in the organism, these ROS are removed via enzymatic and non-enzymatic anti oxidative mechanisms and there occurs an increase in oxidants and decrease in antioxidants, resulting in the shifting of oxidative and anti-oxidative balance in the direction of oxidative stress.[7,8] The harmful reactions of free radicals are prevented by the antioxidant molecules as a total antioxidant response, which is usually measured as their total combined additive antioxidant effects and expressed synonymously in different terminologies such as; TAC, total antioxidant activity (TAA), total antioxidant power (TAOP), total antioxidant status (TAS).[9-13]

The role of lipid peroxidation has been linked to the degree of the inflammation and destruction of the periodontium during progression of periodontitis as per the following findings of different Investigators. The lipid peroxidation levels in gingival crevicular fluid and saliva determine the oxidant activity influencing the damaging effects during the progression of periodontitis. The glutathione (GSH), glutathione peroxidase (GPx) in saliva determine the trend of balance between the oxidant and antioxidant activity influencing the damaging effects of ROS.[14] Increased production of antioxidants is associated with stimulation of salivary flow. The antioxidant potential of saliva does not appear to be compromised in patients with periodontal disease but this may relate to the antioxidant flow from the gingival crevicular fluid. Major aqueous antioxidant component of whole saliva is uric acid, with lesser contributions from ascorbic acid and albumin.[15] Spontaneous release of O_2^- radicals from polymorphonuclear leukocytes (PMN) are found in the gingival crevicular fluid (GCF) of chronic adult periodontitis (CAP) patients. Marked enhancement of O_2^- generation is noticed in GCF with the addition of the exogenous stimuli like phorbol- myristate acetate (PMA) in cases of chronic periodontitis. The protective or destructive effect of PMN in GCF of CAP patients may depend on the variations of the rate of O_2^- formation in respect to the intrinsic antioxidant property of gingival supernatant (GS).[16]

Defective neutrophil functions, are exhibited in localized juvenile periodontitis or RPP patients, to a variety of environmental and host stimuli. These defects are, chemotaxis depression and defective phagocytosis, while granule release and superoxide production were found to be normal. Both normal responses and defective functions are noticed in the same neutrophil populations, mediated by the same receptor in these patients with aggressive periodontitis or RPP.[17] Under such scenario, the increasing evidence implicating ROS, in the pathogenesis of periodontal tissue destruction due to an imbalance in enzymatic and non-enzymatic degradation mechanisms are reviewed. The identification of an imbalance in the oxidant/antioxidant activity within periodontal pockets, suggests a significant role for ROS in periodontal tissue destruction. The detection of ROS oxidation products, the elevation of iron and copper ions, which catalyze the production of the most ROS, presents the evidence of role of ROS in periodontal destruction.[18]

The two categories of antioxidants with preventive and scavenging actions includes, glutathione peroxidase (GPx), catalase (CAT), superoxide dismutase (SOD), glutathione reductase like enzymes, in the first category and α-tocopherol (vitamin E), ascorbic acid (vitamin C), carotenoids (including retinol-vitamin A), reduced glutathione, uric acid, albumin and polyphenols (flavenoids) like non enzymes in the second category respectively. Changes in intracellular redox state triggers damage to vital molecules of connective tissue.[19-14]

Severe tissue destruction associated with severe aggressive form of periodontitis or RPP may be due to the lower level of plasma antioxidant capacity.[20] Serum antioxidant concentration indicated through total antioxidant level including serum concentration of vitamin c and bilirubin were inversely associated with prevalence of inflammatory destructions during progression of periodontitis.[21] There may be a reduction in probing depth, associated with dietary intake of fruits and vegetables containing vit-c, α-tocopherol, eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA) during healing after non-surgical periodontal therapy.[22]

With this background, the present study was designed for assessing the serum levels of non enzymatic antioxidants, vit-c and albumin with assessment of TAC /status in RPP and SPP for comparing them with their respective healthy control groups.

METHODS

Study type and design

A retrospective observational study design was chosen for this study.

Study place and population

The study was conducted at the department of periodontics of S. C. B. dental college, Cuttack, Odisha, India. Study population and materials included the records of the patients who had visited the department of Periodontics at S. C. B. Dental College, Cuttack, Odisha, India, between July 2013 to July 2018 either for the treatment of their periodontal diseases or only for their routine oral hygiene care and check up.

Study period

Study was conducted between July 2018 to March 2019. Study was initiated with planning and protocol

preparation and finished with statistical analysis for arrival at conclusion.

Selection criteria of patients

All the patients selected having records of similar pattern of living style, food habits and oral hygiene habits. All of them were living under similar geographic and climatic conditions. Records of those patients were selected, which could provide the required data for this study. Records of the patients affected by any other systemic disorders are not included in the study. Similarly records of the patients under any other therapeutic regimen for other diseases, and with the habits of smoking or alcohol are not included in the study.

Study procedure

A total number of case history records of 168 patients were selected for this retrospective observational study. The analysis was confined to the population, in whom the required data of periodontal clinical parameters and biochemical parameters were available from their case history records. The study population was divided into 3 groups. Out of which, 42 patients suffering from SPP, including, 30 males and 12 females, ages between 36 to 62 years, were included as group 1. 42 patients suffering from RPP, including 28 females and14 males, ages between 18 to 28 years, were included as group 2. Healthy controls comprised of 84 subjects including 44 males, 40 females having age between 18 to 62 years included as group 3. The control group again subdivided into 2 subgroups. Control sub group 1 included the patients between 30 to 62 years comprising of 42 patients to be compared with SPP. Control subgroup 2 included the patients between 18 to 30 years comprising of 42 patients to be compared with RPP. The demographic data of patients displayed in Table 1. The patients were evaluated for their periodontal examinations with clinical and radiographic findings. The diagnosis of the slowly and RPP was made on the basis of these findings and on the basis of history of the disease progression. Normal patients without any such diseases, who had come for only oral prophylaxis were included in the groups of healthy controls. The criteria for diagnosis were followed as per the new classification and case definition, for staging and grading of periodontitis, of world workshop of periodontology 2017. The patients with SPP included the diagnosed conditions with grade A and grade B at stage 2 and stage 3 with high plaque score, whereas the patients with RPP included the diagnosed condition at grade C from stage 2 to stage 4, with less plaque score from expected value.

Periodontal clinical parameters included, clinical attachment level (CAL), gingival bleeding index (GBI) and plaque index (PI). Clinical attachment levels were included of 6 sites of teeth. Mean values of base line clinical parameters for different groups were shown in Table 2.

Biochemical parameters included TAC/status. Serum levels on TAC data were collected from pathological investigation reports of patients from different groups for this study. TAC was determined by the routinely used method in the attached hospital set up. The method was developed by Erel.[23] This is one colorimetric direct method for measuring total antioxidant status, which is rapid, easy, stable, reliable, sensitive and inexpensive.[23] Vitamin-C assay data collected from pathological investigation report of patients of different group for this study was done by the method developed by Line et al which is a simple, reliable method used for routine plasma vitamin C analysis in the attached hospital set up, where this study was conducted.[24] This is a validated and reproducible method for accurate measurement of the concentration of vitamin c in blood. Serum albumin assay data were collected for all patients from their routine pathological investigation report. The said assay of serum albumin was done by the colorimetric method.[26]

Statistical analysis

Bio chemical parameters were subjected to statistical analysis using IBM SPSS statistics 21 core system package for group's comparison analysis for test of significance. Microsoft word excel used for presentation of data and graphical icon.

RESULTS

Demographic characters for all the groups are listed in Table 1. All the mean values of clinical parameters were found to be on higher side in the SPP and RRP groups when compared with their respective control sub groups (Table 2). The mean PI (plaque index) value is only found to be less in group II when compared with group I (Table 2). The graphical presentation of CAL, GBI and PI are presented for different groups in Figure 4-6 respectively.

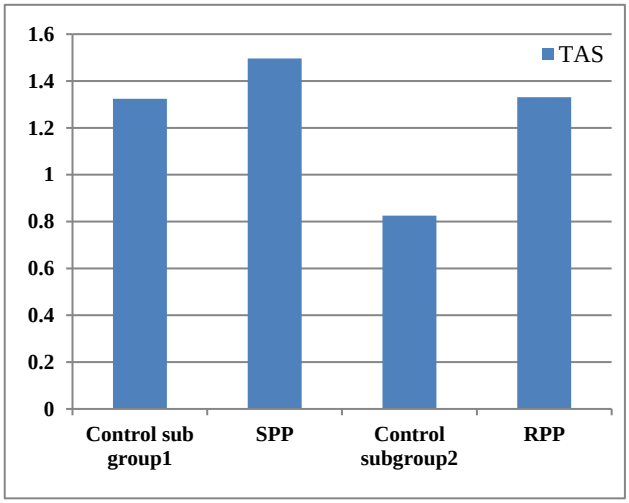

Figure 1: Comparison of total antioxidant status/ capacity, between control subgroup-1 and SPP, control subgroup-2 and RPP.

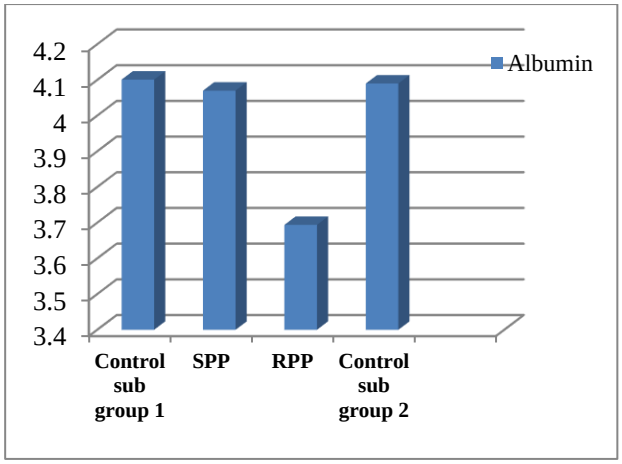

Figure 2: Comparison of serum albumin level between control subgroup-1, SPP, control subgroup-2 and RPP.

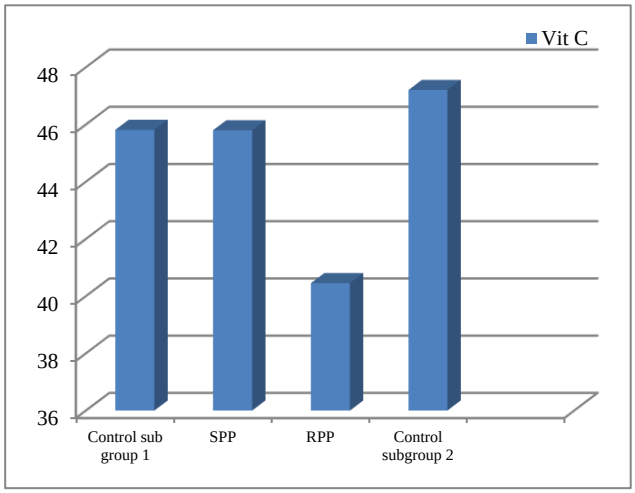

Figure 3: Comparison of vitamin-C level between, control subgroup-1 and SPP, control subgroup-2 and RPP.

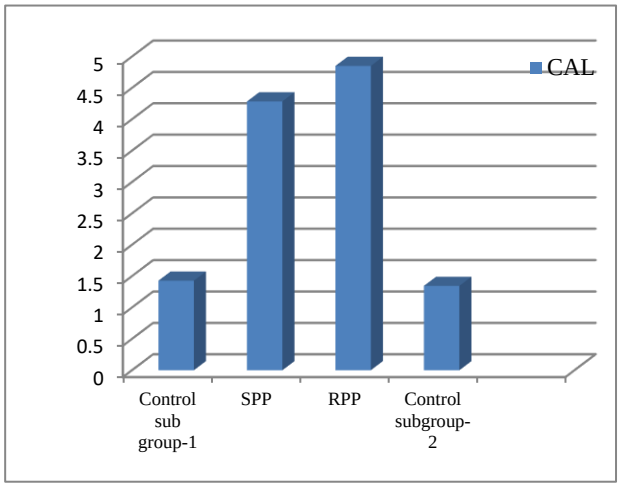

Figure 4: Comparison of clinical attachment level between control subgroup-1, SPP, RPP, and control subgroup-2.

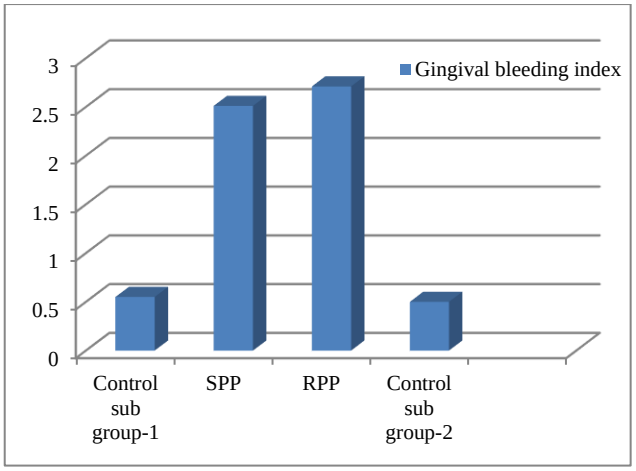

Figure 5: Comparison of gingival bleeding index between control subgroup-1, SPP, RPP, and control subgroup-2.

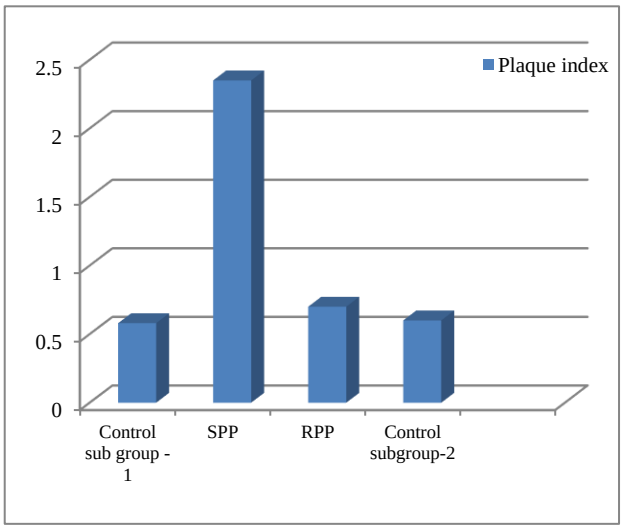

Figure 6: Comparison of plaque index between control subgroup-1, SPP, RPP and control subgroup-2.

Biochemical findings

Biochemical parameters of different groups are displayed in Table 3. Groups comparison statistics of biochemical parameters presented in Table 4. Groups comparison analysis for test of significance presented in Table 5.

Contrgrt1: This designated comparison, in the Table 4 and 5 shows, Statistical analysis comparing the TAC /status score of SPP with the control sub group 1, where the difference was found to be statistically significant.

Contrgrt2: This designated comparison, in the Table 4 and 5 shows, statistical analysis comparing TAC /status score of RPP with the control sub group 2, where the difference was found to be statistically significant.

Contgrpc1: This designated comparison, in the Table 4 and 5 shows, Statistical analysis comparing serum vit-C score of SPP with the control sub group 1, where the difference could not be considered significant.

Contgrpc2: This designated comparison in the Table 4 and 5 shows, Statistical analysis comparing serum vit-C score of RPP with the control sub group 2, where the difference could be considered as significant.

Contgralb1: This designated comparison in the Table 4 and 5 shows, Statistical analysis comparing serum albumin level between SPP with the control sub group 1, where the difference could not be considered as significant.

Contgralb 2: This designated comparison in the Table 4 and 5 shows, Statistical analysis comparing serum albumin level between RPP with the control sub group 2, where the difference could be considered as significant.

Table 1: Demographic characteristics of different study groups.

Characteristics		Control group				SPP (Gr-l), (n=42)		RPP (Gr-ll), (n=42)	
		Control sub gr-1, (n=42)		Control sub gr-2, (n=42)					
Sex	Males (M)	28		18		30		14	
	Females (F)	16		22		12		28	
Age (in years)		M	F	M	F	M	F	M	F
18-28				18	22			14	28
29-39		4	6			5	3		
40-50		18	8			16	7		
51-62		6	2			9	2		

Table 2: Comparison of clinical parameters in different study groups at base line (cross sectional analysis at base line).

Parameters	Groups			
	Control group, (n=84)		SPP (Gr-l), (n=42)	RPP (Gr-ll), (n=42)
	Control sub gr-1,	Control sub gr-2,		
	1.42±0.09	1.34±0.21	4.28±0.23	4.85±0.21
GI	0.55±0.07	0.50±0.07	2.51±0.08	2.71±0.13
PI	0.58±0.13	0.60±0.11	2.35±0.31	0.70±0.14

Table 3: Bio chemical parameters of control groups and SPP, RPP patients.

Variables	Control sub group-1, (n=42)	SPP group-1, (n=42)	Control sub group-2, (n=42)	RPP group-2, (n=42)
Vit-C (µmol/L)	45.81±3.87	45.798±3.9765	47.20±2.569042	40.45±3.499
TAC (F=0.11 mmol trolox equivalent)	1.49±0.29	1.32±0.05	1.33±0.18	0.213±0.069
Albumin (gm/dl)	4.10±0.245	4.079±0.27	4.09±0.36	3.69±.07

Table 4: Groups comparison statistics.

Groups		N	Mean	SD	Std. error mean
Contrgrt1	1.00	42	1.3240	0.05379	0.00830
	2.00	42	1.4964	0.29183	0.04503
Contrgrt2	1.00	42	1.3314	0.18059	0.02787
	2.00	42	0.213	0.069	0.01080
Contgrpc1	1.00	42	45.812	3.8743	0.5978
	2.00	42	45.798	3.97	0.6210
Contgrpc2	1.00	42	47.205	2.56	0.4012
	2.00	42	40.452	3.49	0.5465
Contgralb1	1.00	42	4.1019	0.24819	0.03830
	2.00	42	4.0790	0.27379	0.04225
Contgralb2	1.00	42	4.0905	0.36590	0.05646
	2.00	42	3.6948	0.07313	0.01128

Table 5: Groups comparison analysis for test of significance.

Variables	T test for equality of means						
	T	Df	Sig. (2 tailed)	Mean difference	Std. error difference	95% CI difference	
						Lower	Upper
Contrgrt1	-3.765	82	0.005	-0.17238	0.04579	-0.26347	-0.08129
	-3.765	43.782	0.005	-0.17238	0.04579	-0.26468	-0.08009
Contrgrt2	37.429	82	0.005	1.11857	0.02989	1.05912	1.17802
	37.429	53.046	0.005	1.11857	0.02989	1.05863	1.17851
Contgrpc1	0.017	82	0.987	0.0143	0.8620	-1.7005	1.7291
	0.017	81.881	0.987	0.0143	0.8620	-1.7006	1.7291
Contgrpc2	9.960	82	0.005	6.7524	0.6780	5.4037	8.1011
	9.960	75.248	0.005	6.7524	0.6780	5.4019	8.1029
Contgralb1	0.401	82	0.690	0.02286	0.05702	-0.09057	0.13629
	0.401	81.222	0.690	0.02286	0.05702	-0.09059	0.13631
Contgralb2	6.873	82	0.005	0.39571	0.05758	0.28118	0.51025
	6.873	44.270	0.005	0.39571	0.05758	0.27970	0.51173

Summary of the results

The values of serum TAC were found to be at a lower level in both SPP and RPP, when compared with their healthy controls. The values of serum vitamin C and albumin levels in RPP even remaining within the normal ranges were found to be on the lower side when compared with their healthy controls, where as those values in SPP are not significantly different when compared with their healthy controls.

DISCUSSION

The "disturbance of homeostatic balance", between the protecting antioxidant defense system of supporting tissues of periodontium, the proteolytic enzymes, their inhibitors and ROS, is believed to be the specific cause of variable rate of tissue destruction during the progression of periodontitis. Free radical-induced tissue damage that involves pathogenic processes in inflammatory diseases is similar to the pattern of tissue destruction in periodontal diseases.[4]

The range of endogenous and exogenous anti-oxidants present in the human body for countering the free radicals is wide and extensive. They are divided into 3 groups such as; anti-oxidant enzymes, chain breaking antioxidants, and transition metal binding proteins. Chain breaking antioxidants are divided again as, lipid phase chain breaking antioxidants and aqueous phase chain breaking antioxidants. Among aqueous phase chain breaking anti-oxidants the vitamin C and sulphydryl groups present on plasma proteins are important antioxidants, directly scavenge radicals, present in the aqueous compartment.[7,8] Vitamin C (ascorbate) is one of the most important anti-oxidant. It acts as an essential cofactor for several enzymes for catalysing hydroxylation reactions. It also acts as a cofactor for prolyl and lysyl oxidases for helping in the synthesis of collagen. Vitamin C also act as a scavenger for radicals like, superoxide, hypochlorous acid, hydrogen peroxide, aqueous peroxyl radicals, the hydroxyl radical and singlet oxygen. Plasma proteins can function as chain breaking antioxidants because of their sulphydryl groups, by the way of donating an electron to neutralize a free radical. Major portion to plasma sulphydryl groups is contributed through albumin. Albumin has also several other antioxidant properties, having 17 disulphide bridges with a single remaining cysteine residue, responsible for neutralize of peroxyl radicals. This property is important in the context of the role of albumin as a carrier of free fatty acid. The role of albumin in binding copper ion helps in inhibition of lipid peroxidation and hydroxyl radical formation. Albumin also acts as a scavenger of hypochlorous acid, the product of phagocytosis.[8] Different authors have suggested different methods for assessment of power of antioxidant system for countering the harmful effects of ROS. Total antioxidant response is expressed by various authors through different synonymous terminologies like; TAC, TAA, TAOP, TAS.[9-13]

The lipid peroxidation levels in serum, gingival crevicular fluid and saliva determines the trend of balance between the oxidant and antioxidant activity influencing the damaging effects of ROS. Spontaneous release of O_2^- radicals from polymorphonuclear leukocytes (PMN) are found in the gingival crevicular fluid (GCF) of chronic adult periodontitis (CAP) patients. The PMN in GCF of CAP patients can elaborate its protective or destructive effect depending on the variations of the rate of $O2^-$ formation in respect to the intrinsic antioxidant property. Defective neutrophil functions to a variety of environmental and host stimuli are exhibited in LJP patients. The substantial role of free radicals or ROS in periodontal destruction is clearly defined, but research conducted in this area is very little. Normal superoxide formation are exhibited in patients suffering from aggressive periodontitis.[14-16]

The review on the studies of the pathogenesis of periodontal tissue destruction due to an imbalance in enzymatic and non-enzymatic degradation mechanisms revealed that, the imbalance in the oxidant/antioxidant activity within periodontal pockets, emphasizes the role of "ROS" in the pathogenesis of periodontal tissue destruction".[18] Changes in Intracellular redox state, trigger gene transcription events, resulting in tissue damage, that occurs secondary to the induction of pro-inflammatory state. Vital and ubiquitous transcription factors such as nuclear factors-KB and activating protein-1 are redox sensitive, so upward shift in the pro oxidant and anti-oxidant ratio intracellularly causes direct damage to the vital molecules and structures of connective tissues.[19] Massive tissue destruction associated with severe periodontitis, especially in aggressive forms may be due to the reduction in antioxidant capacity of the patients.[20] Inflammatory destruction during progression of periodontitis is inversely associated with serum antioxidant concentration.[21] Application of dietary strategies to optimize healing after periodontal procedures has been suggested after analysis of observations, on the influence of dietary intake of fruits and vegetables with vitamin C, β-carotene, eicosapentaenoic acid, docosahexaenoic acid and α-tocopherol, in reduction of pocket depth.[22]

One of the major intravascular antioxidants is serum albumin. While initiating the discussion about the antioxidant role of the albumin, the role of albumin in creating oxidative stress in the pathophysiology of IgA nephropathy cannot be ignored. One retrospective cohort study was done to access the possibility of the association between serum albumin and the progression of IgA nephropathy.[25] Albumin has various physiological functions, like, regulation of osmotic pressure, and transport of drugs, fatty acids, and metals. Serum albumin can be measured by the colorimetric method, the said method was followed in this study.[26]

Ascorbate, albumin and urate have been documented to be the chief non enzymatic antioxidants of the plasma. These antioxidants also display sensitivity to dietary intake, whereas, the effect of dietary influence upon salivary antioxidant status is not clear. Urate has been documented to be the major contributor of the salivary antioxidants and albumin and ascorbate were considered as the minor contributors. There have been attempts for exploring the possible nutritional strategy for improving the antioxidants status for treatment of periodontitis.[27]

Investigations on total oxidant status revealed that patients with chronic periodontitis were having increased lipid peroxidation levels and oxidative stress both at systemic level and at local sites.[28] On evaluation of intracellular production and extracellular release of ROS by PMN stimulated by fusobacteria, it was found that local microbes might stimulate PMN to release ROS bringing inflammatory destruction.[29] On examination of the oxidative burst of polymorphonuclear leukocytes (PMNL) in the peripheral blood from the patients with various types of periodontal diseases including localized juvenile (LJP), generalized juvenile (GJP) and adult periodontitis, it was found that, the inflammatory status of periodontal disease may be the reflection, of the capacity of PMNL, of peripheral blood, in mounting oxidative burst.[30]

Improvement in initial periodontal pocket depth reductions may be found with intake of adjunctive juice powder concentrates in nutritionally replete patients, during non-surgical debridement maintainance.[31] Association of chronic periodontitis with lower level of plasma TAC is found to be significant and oxidative stress is relieved after non-surgical periodontal therapy without any significant effect by adjunctive vit-C supplement.[32] Investigation, on the effect of fish oil with dietary ω-3 fatty acids (FAs), docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) on periodontal health reflected that, there may be an inverse, independent relation of dietary docosahexaenoic acid DHA intake to the progression of periodontal disease in older people.[33] Lower intake of vitamin C showed significant risk of periodontal disease in current and former smokers. A relationship between reduced dietary vitamin C and increased risk for periodontal disease for the overall population was found.[34]

When the relationship between periodontal disease is correlated with general health status in community-dwelling elderly patients, assessing serum albumin concentration as the criterion for maintenance of nutritional status, it was found that, there is an inverse relation between the serum albumin concentration and periodontal disease, which indicates that the role of serum albumin cannot be ignored for control of periodontal disease. Among the risk predictors of periodontal disease, serum albumin was also found to be one of the risk predictors for disease progression in elderly non smokers.[35,36]

With this background, data for TAC, vitamin and serum albumin assay for all patients were obtained from their routine pathological investigation reports, and subjected to statistical analysis in this study. TAC was determined by the use of novel automated method, developed by Erel.[23] This is one colorimetric direct method for measuring total antioxidant status, which is a rapid, easy, routinely used in the attached hospital set up, where this study was conducted. Plasma vitamin C and serum albumin concentrations were determined by simple and reliable methods, routinely used in the attached hospital setup.[24,26] The levels of non enzymatic antioxidants, albumin, vitamin C and TAC of patients, suffering from RPP and SPP are compared with their respective healthy controls through statistical analysis.

In this study, values of serum TAC were found to be at a lower level in both SPP and RPP, when compared with their respective healthy controls (Table 4 and 5). The

values of levels of vitamin C and albumin in RPP even remaining within the normal ranges were found to be on the lower side when compared with their healthy controls (Table 4 and 5). Values of vitamin C and albumin levels in SPP are not significantly different when compared with their healthy controls (Table 4and 5). Graphical comparison of TAC / status, between control subgroup-1 and SPP, and between control subgroup-2 and RPP presented in Figure 1. Graphical comparison of serum albumin levels between control subgroup-1 and SPP and between control subgroup-2 and RPP presented in Figure 2. Graphical comparison of vitamin-C levels between control subgroup-1 and SPP, and between control subgroup-2 and RPP presented in Figure 3.

CONCLUSION

Reduction in antioxidant status and increase of oxidative stress may have a role in playing as a risk factor in the pathogenesis of SPP and RPP. The systemic levels of non enzymatic antioxidants, vit C and albumin, in RPP were found to be on the lower side, even remaining within the normal ranges, when compared with the healthy controls. The role of non-enzyamatic antioxidants like albumin and vit C in the pathogenesis of RPP needs further research in this field.

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