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ESTIMATION OF REACTIVE OXYGEN METABOLITE (ROM) LEVELS IN SALIVA AND SERUM IN CHRONIC PERIODONTITIS AND IN PATIENTS WITH AND WITHOUT RHEUMATOID ARTHRITIS- A COMPARATIVE ANALYSIS

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ABSTRACT

Background

Rheumatoid arthritis (RA) and chronic periodontitis (CP) are the most common chronic inflammatory diseases and have similar pathologies. Oxygen metabolism has an important role in the pathogenesis of both CP and RA.

Aims and objective

The aims of the study are

1. To compare the salivary and serum ROM levels in Chronic periodontitis and in patients with and without Rheumatoid arthritis.
2. To clinically correlate the effect of rheumatoid arthritis on the severity and progress of chronic periodontitis.

Materials and methods

The study population consisted of 90 subjects belonging to both the
Sexes were randomly selected. Subjects were divided into three groups. Rheumatoid arthritis group (RA), Chronic Periodontitis (CP) without Rheumatoid arthritis and Rheumatoid arthritis with chronic periodontitis (CP+RA). Saliva and serum were collected in all the three groups to estimate the Reactive oxygen metabolite levels.

Results

There was a significant increase of ROM in the RA + CP group when compared with RA and CP (p value <0.001). But there was no significant differences in salivary ROM levels between RA and CP (p value 0.28) and serum ROM levels between RA and CP (p value 0.178)

Conclusion

The results of our study suggested that a significant oxidative stress occur in Chronic Periodontitis and Rheumatoid arthritis.
The findings also suggest that it might play an important role in the pathogenesis of Periodontitis and Rheumatoid arthritis and the associated tissue damage.

INTRODUCTION

The human body possesses an innate defense mechanism against disease. It is characterized by the ability of certain cells which contain receptors to recognize and in turn impede the invasion of micro-organisms which attack the body. The W.B.C's play a major role in this process and are therefore an integral part of the host immune system. When this active process of host immunization continues without switching off the activities, it results in a chronicity of the inflammatory process. This hazardous event, destroys the normal form and function of parts of the human body causing a slow, and often irreversible damage to the affected host tissues.

The above mentioned condition has often been observed as a common process in both Rheumatoid arthritis and Chronic periodontitis and this is the basis of our current study.

An imbalance in the pro-inflammatory factors like neutrophil elastase & catalase with an alarming growth of Reactive

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oxygen species (ROS), causes an irreversible molecular damage in the gingival tissues as well as the synovial joints in the human body. This in turn results, in an irrevocable damage in the periodontium, manifested as chronic periodontitis[1] as well as the major joints of the body, manifested as Rheumatoid arthritis.

The multifactorial (presence of more than one etiological agent) nature and the production of reactive oxygen species[2] by the activated phagocytes, results in the destruction of the base material in the body, namely the connective tissue.[3]

When more than five joints are affected in the body, there is an expression of this condition in the form of a polyarthritic nature, known as Rheumatoid arthritis (RA).[4] The clinical classification of Rheumatoid arthritis (according to the 2010 ACR), classifies it based upon 9 criteria[5].

The association between Rheumatoid arthritis and Chronic periodontitis, dates back to the early 19th century with Benjamin Rush (a U.S based doctor) being the earliest to have conducted studies to prove the unmistakable link between the two conditions. His observation coincided with William Huntor's "focal infection theory". This subsequently resulted in a random extraction of all infected teeth in the oral cavity which justified the elimination of the 'sepsis' (infection) in other body conditions like rheumatoid arthritis. However, this theory and its practice was later discarded in the 50's for want of a more valid scientific justification[6]. Oxidative stress has been often observed as one of the key factors in rheumatoid arthritis, due to an increased cellular oxygen consumption, the generation of oxygen free radicals and an increased anaerobic glycolysis[7]. The synovial tissue and the synovial fluid is the key area, involved in the damage due to an increased oxidative stress[8]. In chronic periodontitis, it is the junctional epithelium, gingival sulcus and the area of attachment of the periodontal ligament to the cementum, which is largely affected.

Besides the soft tissue damage and the swelling in the bone joints. Other systemic disorders like diabetes mellitus, cardiovascular conditions like atherosclerosis, myocardial infarction and cardiovascular stroke have been observed in patients with chronic periodontitis[9,10,11]. Moreover, a patient with an active condition of rheumatoid arthritis, has been observed to have more than one site in the oral cavity, with an active lesion of chronic periodontitis.

Porphyromonas gingivalis (Pg), a bonafied periodontal pathogen in chronic periodontitis, is markedly increased in an active rheumatoid arthritis patients. This is in tune with a recent study by Wegner *et al*[12]. He presented a unique model wherein a Pg mediated citrullination of bacterial and host proteins generates antigens that in turn trigger an auto-immune response in rheumatoid arthritis. Hitchon *et al*[13] in a genetically predisposed population of native American patients with rheumatoid arthritis and their relatives showed that anti-P.gingivalis antibodies were associated with anticitrullinated protein antibodies, suggesting that immune responses to P.gingivalis may be involved in breaking immune tolerance to citrullinated antigens.

Basic energy for all life activities is provided by oxygen. However, the free energy liberated by free radicals like superoxide and hydroxyl oxide radical causes both tissue injury and tissue damage. Oxidative stress is often due to this imbalance due to an excess reactive oxygen species (ROS) at a cellular level[14]. Similar to a balance between health and disease, there is a normal physiological balance between the oxidants and anti-oxidants at a cellular level. Tissue injury occurs, when there is a tilt of the applecart, favoring an increased production of oxidants and a decrease in the antioxidant level. When the supportive role of an antioxidant in preserving health is diminished, the subsequent destructive role of reactive oxygen species is activated causing an outburst of inflammation; a condition which is common to both rheumatoid arthritis and chronic periodontitis.

This study, to be best of my knowledge is one of the rare studies to compare the salivary and serum ROM levels in south Indian population. The aims of the study are to

1. To compare the salivary and serum ROM levels in patients with rheumatoid arthritis, chronic periodontitis, RA+CP in the south Indian populations
2. To clinically correlate the effect of rheumatoid arthritis on the severity and progress of chronic periodontitis.

MATERIALS AND METHODS

The study group consisted of ninety human volunteers. They were divided into a total of three groups; Group A for chronic periodontitis (CP); Group B for rheumatoid arthritis (RA) and Group C for subjects with a both rheumatoid arthritis (RA)+chronic periodontitis (CP)

The patient pool for chronic periodontitis subjects, was obtained from the Dept. of Periodontics, Thai Moogambigai Dental College. Wherein both clinical and radiographic evaluation was conducted. The inclusion criteria for chronic periodontitis subjects consisted of patients having atleast four teeth with one or more sites, with a probing depth of ≥4mm, CAL of ≥ 4mm and a radiographic evidence of bone loss around atleast four teeth. Patients with rheumatoid arthritis were collected from the out patient pool of the Department of Rheumatology (Kilpauk Medical College & Hospital). The inclusion criterion was fulfilling the American College of Rheumatology classification criteria for RA

Other criteria for inclusion in the study required the volunteers to be free of any systemic disease; having received no periodontal treatment and not taking any medication for the past six months. Bleeding on probing less than ≤ 20% of sites.

Ethical Approval

The Ethical committee for medical and dental research in Kilpauk Medical College and Thai Moogambigai Dental College, Chennai was obtained from them, before proceeding with the study.

Collection of Samples

Collection of Serum

2ml of Blood (3 ml) was collected from the antecubital fossa by venipuncture using a 20-gauge needle with a 2ml syringe and immediately transferred to a laboratory. The blood sample

was allowed to clot at room temperature and after 1hour, serum was separated from blood by centrifugation at 3000 xg for 5 minutes. The extracted serum was immediately transferred to a plastic vial and stored at - 70°c until the time of assay.

Collection of saliva

Draining / Spitting method

The subject is asked to accumulate saliva in the floor of mouth and then spit into a preweighed or graduated test tube.

Laboratory method for detection of ROM

The d-ROMs test developed by world-renowned Italian biochemist (Mauro Carratelli 2001)[15]is a photometric test for measurement of the concentration of hydro peroxides (ROOH) in biological samples. The presence of ROOH in cells indicates oxidative attack of ROS on various organic substrates such as carbohydrates, lipids, amino acids, proteins, or nucleotides.

Test principle

The d-ROMs test uses the principle of Fenton's reaction: by mixing a biological sample with an acidic buffer (Reagent R1), the newly created transition metal ion (iron or copper) catalyzes the breakdown of hydroperoxide, generating new radical species such as hydroxyperoxyl (ROO+) and alkoxyl (RO+).By adding a chromogen (N, N-diethylparaphenylendiamine, Reagent R2) having the ability to donate an electron and change color when oxidized by free radicals, and using photometric reading available with the FRAS 4 dedicated analytical equipment, it becomes possible to quantify the level of hydroperoxides available in the sample.

Statistical Analysis

All statistical analyses were performed using a software program (Spss 15 version). Comparison of ROM levels in serum and saliva in patient with Rheumatoid arthritis and Chronic periodontiotis, Rheumatoid arthritis only and Chronic Periodontitis only were analyzed using Kruskal wallis test and post hoc comparison between groups were done using Mann Whitney U test and bonferroni correction

RESULTS

The demographic data of mean age, salivary ROM levels, serum ROM levels, probing depth, clinical attachment level are given in Table 1.

The comparison of mean saliva ROM values amongst three groups namely RA group, CP group and a RA+CP were conducted using a kruskal wallis test and a P value of < 0.001 was attained. These results were found to be clinically significant are given in Table 2

The results of this table was achieved comparing salivary ROM levels within three groups. The comparison of salivary ROM levels between the RA group and CP group was found to be clinically insignificant, at a P value of 0.28; whereas the comparison of salivary ROM levels between the other two groups, namely RA and RA+CP and CP and RA+CP group was found to be clinically significant with a P value of < 0.001.given in Table 2a

Comparison of mean serum ROM values was made among three groups using a kruskal wallis test. The results were found to be statistically significant (P value <0.001) were given in Table 3

The results of the serum ROM levels between two groups namely RA vs RA+CPand CP vs RA+CP were found to be statistically significant. Whereas when comparing the levels of serum ROM of RA with the serum ROM of CP the result was not statistically significant (P= 0.178) given in Table 3a

DISCUSSION

The relationship between RA and CP has been confounded by innumerable studies, done in the past[16,17,18,19]. However at a cellular level, there have been very few studies, concerning their status in relation to anti-oxidants and oxidants. The endorgenous production of ROS (Reactive oxygen species) in RA is still unclear.

Table 1 Demographic data

		RA	CP+RA	CP		
Gender	Male	12	20	12		
	Female	18	10	18		
		N	Mean	Std. Deviation	Minimum	Maximum
Age	RA	30	49.50	5.39955	39.00	59.00
	CP+RA	30	46.90	5.57921	38.00	56.00
	CP	30	43.00	5.81911	30.00	56.00
	Total	90	46.46	6.15566	30.00	59.00
Serum_ROM	RA	30	346.60	92.58867	200.00	520.00
	CP+RA	30	417.23	230.19491	25.40	950.00
	CP	30	238.49	66.84273	18.00	356.75
	Total	90	334.11	164.25322	18.00	950.00
Saliva_ROM	RA	30	390.30	101.06063	245.00	562.00
	RA+CP	30	462.07	249.07909	30.50	978.00
	CP	30	238.29	77.65890	20.00	355.75
	Total	90	363.55	185.22911	20.00	978.00
PD	RA+CP	30	4.33	1.29544	3.00	8.00
	RA	30	4.13	1.40770	2.00	8.00
	CP	29	1.65	.48373	1.00	2.00
	Total	89	3.39	1.66254	1.00	8.00
CAL	RA+CP	30	7.06	1.74066	4.00	12.00
	RA	30	6.53	1.92503	1.00	10.00
	CP	29	.13	.35093	.00	1.00
	Total	89	4.62	3.48823	.00	12.00

Table 2 Comparison of Mean Saliva Rom values among three groups using Kruskal wallis test

Groups	N	Mean	Std. Deviation	95% Confidence Interval for Mean		Minimum	Maximum	P value
				Lower Bound	Upper Bound			
RA	30	390.30	101.06	352.5633	428.0367	245.00	562.00	
CP+RA	30	462.07	249.07	369.0590	555.0743	30.50	978.00	<0.001
CP	30	238.29	77.65	209.2934	267.2900	20.00	355.75	
Total	90	363.55	185.22	324.7573	402.3483	20.00	978.00	

Table 2a Post hoc comparison using Mann Whitney U test and bonferroni correction

	Grp	N	Mean	Std. Deviation	P value
Saliva_ROM	RA	30	390.30	101.06	0.28
	CP	30	462.07	249.07	
Saliva_ROM	RA+CP	30	390.30	101.06	
	CP	30	238.29	77.65	<0.001
Saliva_ROM	CP+RA	30	462.07	249.07	<0.001
	RA	30	238.29	77.65	

P<0.016 is considered significant (Bonferroni Correction)

There was a total of 46 females and 44 males in the total group of 90 human volunteers. A slight tilt in the ratio of females: males, was probably because of the prevalence of RA being more evident in females[22].

The results of the present study shows independently both CP and RA may have an effect of increasing oxidative stress, Which is evident by the ROM and very significant in RA+CP patients.

Table3 Comparison of Mean Serum Rom values among three groups using Kruskal wallis test

Groups	N	Mean	Std. Deviation	95% Confidence Interval for Mean		Minimum	Maximum	P value
				Lower Bound	Upper Bound			
RA	30	346.60	92.58	312.0268	381.1732	200.00	520.00	
CP+RA	30	417.23	230.19	331.2738	503.1862	25.40	950.00	<0.001
CP	30	238.49	66.84	213.5272	263.4462	18.00	356.75	
Total	90	334.11	164.25	299.7034	368.5077	18.00	950.00	

Table3a Posthoc multiple comparisons using Mann Whitney U test and Bonferroni correction

	Grp	N	Mean	Std. Deviation	P value
Serum_ROM	RA	30	346.60	92.58867	0.178
	CP	30	417.23	230.19491	
Serum_ROM	RA+CP	30	3.4660E2	92.58867	<0.001
	RA	30	2.3849E2	66.84273	
Serum_ROM	CP+RA	30	417.23	230.19491	<0.001
	CP	30	238.49	66.84273	

P<0.016 is considered significant (Bonferroni Correction)

Recent studies by uma *et al*[23] has shown that there is an increase in ROM levels in CP by comparing with GCF, saliva and plasma.

This study shows increased ROM levels in all three groups, but significantly more in RA+CP group. A study by ufuk sezer *et al*[24] have shown increased OSI in RA & CP, with higher OSI in CP+RA.Our study is smaller to sezer *et al* in measuring in oxidative stress, but we have done using ROM.

The salivary and serum levels were not statistically significant in two groups in the study. This could be due to the small sample size used in the study. These data also agreed with the study by Esen *et al*[25].

The pathogenesis of both RA and CP hold many similarities[26]. An underlying exaggeration in the inflammatory response in both RA and CP, has been observed as a common factor by Mercado *et al*[27]. In a study by Pischon *et al*[28] it was observed that there is a distinct association between a deteriorating oral hygiene with the progression of attachment loss of CP and the manuel disability caused by RA.

Both these groups in the above study showed a statistically significant P value (<0.001) when compared to healthy controls, who were free of both RA and CP. A significant increase in salivary and serum samples of RA patients, could be due to a combination of environmental and genetic factors causing an exaggerated host response in the RA inflicted subjects.

Ozturk *et al*[29] suggested in his study, that the ROM serum and salivary levels were increased in RApatients due to an increase in oxidative stress levels. Several studies independently claim an association between CP and oxidative stress levels.

In our study when compared with the healthy group both the salivary and serum ROM levels showed a statistical difference. There was no statistically significant difference among the two

The SOD concentration present in our body fluids act as a lubricants and in a way prevents inflammatory changes in our joints. During inflammatory conditions like RA certain pro inflammatory molecules like PGE2, cytokines, ROS, NO reduce the levels of SOD in the joint fluids there by aggravating the pathology associated with degenerative joints. The antioxidant levels in the synovial fluids are also reduced drastically in RA due to release of ROS in the inflammatory site[20]. Similar conditions occur in the DNA, collagen and cartilaginous structure. Cerhan JR *et al*[21] have reported similar studies which increases oxidative stress levels in RA. Although many studiers have been conducted with above parameter our study is a rare one evaluating salivary and serum ROM levels in both CP and RA patients.

The imbalance between oxidants and antioxidants leads to the destruction of the periodontium. It has been proved that the bacterial species in subgingival plaque and the PMN are responsible for the changes in the PDL status. Increase in the levels of ROS lead to the destruction of periodontal tissue leading to periodontitis. Females are three times more affected with RA than males with a prevalence of 0.5% to 1% in the American and European population. In our study there was a total of 18 females and 12 males in RA group and in the CP group.

groups in terms of ROM values. To the best of our knowledge, this is the first report to consider the prevalence of ROM in rheumatoid arthritis and chronic periodontitis.

Findings of the present study clearly demonstrate that presence of the local inflammatory condition acted as a principle factor rather than the systemic (serum) inflammatory condition. Moreover, RA seems to affect neither serum nor salivary ROM values in the presence of CP. Both saliva and serum samples provide a measurement of the ROM concentration at a single point in time. Therefore they can be used to test chronic changes, but subjects are to major physiological daily fluctuations, making the assessment of overall long term systemic ROM exposure is difficult. Hence single measurement is cannot reflect the integral of systemic exposure. Salivary ROM concentrations correlate well with serum concentrations. In contrast to serum ROM, salivary ROM reflects free ROM and is collected in a less invasive method.

Further studies with a large sample size are needed with a different design and larger patient groups to evaluate the relationship between RA and periodontal disease and the impact of oxidative stress on the diseases pathogenesis.

CONCLUSION

Increased levels of ROM in RA+CP, underlines the role of oxidative stress in pathogenesis of both the disease. The understanding of the interplay between the two, will be helpful in development of novel therapeutic strategies for RA.

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