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Original Article

Dentistry Section

Estimation of Pentraxin 3 and *Porphyromonas Gingivalis* Levels in Patients with Rheumatoid Arthritis and Periodontitis- An Observational Study

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ABSTRACT

Introduction: Periodontal diseases are inflammatory in nature involving interplay between the bacterial plaque and the micro-organisms, with the response of the host playing a pivotal role in either attenuating or eliminating the disease. Rheumatoid Arthritis (RA) is also a chronic inflammatory disease which shares common risk factors with periodontitis. *Porphyromonas Gingivalis* and Pentraxin 3 (an acute inflammatory protein) have been observed to be associated with both the diseases.

Aim: This study was done to determine if there was any association between *Pophyromonas Gingivalis* and Pentraxin 3 levels in patients with RA and Chronic Periodontitis in comparison with Healthy controls.

Materials and Methods: This observational study was conducted on 90 subjects (42 Males and 48 Females) aged between 30-60 years. The subjects were selected from the out patient ward of a tertiary referral care hospital. The selected subjects were equally divided into three groups. Group I: Comprising of 30 patients with RA and chronic periodontitis who were in turn, subdivided into Group I- A (n=15) –Patients just diagnosed with RA and Group I- B (n=15) Patients under medications for more than three months. Group II: Patients with Chronic Periodontitis (n=30) and Group III: Healthy Controls (n=30) Intergroup comparison for continuous data was done by One-way analysis of variance test followed by Bonferroni's post-hoc test. Intragroup comparison for continuous data was done by paired t-test.

Results: Intergroup comparison between Group I and Group II did not show any statistical difference pertaining to the clinical parameters except for the Gingival Index (GI) which was found to be higher in Group II (Mean= 2.07) when compared to Group I (Mean=1.59). Intragroup comparison between Group IA and Group IB was statistically significant for GI, Plaque Index (PI), and Clinical Attachment Levels (CAL). Intragroup comparison between Group I-A and Group I-B showed that both *Porphyromonas gingivalis* and Pentraxin 3 Levels were higher in Group I- A.

Conclusion: In this study, it was observed that there was a positive association between *Porphyromonas gingivalis* and Pentraxin 3 levels in patients with rheumatoid arthritis and periodontitis.

Keywords: Clinical parameters, Polymerase chain reaction, Quantikine enzyme linked immunosorbent assay

INTRODUCTION

Periodontitis and RA are chronic inflammatory diseases which share common risk factors. In the past few years, a lot of work has been done to undermine the relationship between both the diseases. Alteration in the protein arginine has been implicated as a major cause for RA. As the structure of the protein changes due to citrullination the host recognises them as foreign bodies and produces antibodies. Enzymes produced by *Porphyromonas Gingivalis (P.Gingivalis)* also have the propensity to cause citrullination of the host proteins, thus evoking speculations about the role of this organism in RA and periodontitis [1,2].

As both the diseases are inflammatory in nature many studies have been done assessing inflammatory markers like TNFα, RA Factor, IL-1,IL-6 and their contribution in the aetiology of both the diseases [3]. Pentraxin 3 is one such marker which has been asessed in recent times. It belongs to the family of acute phase proteins. As there is ample evidence that there is a link between RA and periodontitis, this study is an attempt to understand the effects of *Porphyromonas Gingivalis (P.Gingivalis)* [4,5] and Pentraxin 3 in patients affected with both conditions. The hypothesis to be studied was if there was a correlation between RA and periodontitis and also to ascertain if Patients under medication for RA more than three months showed any changes in their microbiologic, Immunologic and periodontal parameters when compared to others.

MATERIALS AND METHODS

This observational study was undertaken in the Department of Periodontics of Panineeya Mahavidhyalaya Institute of Dental Sciences, in collaboration with the Department of Rheumatology of Ozone Hospitals in Hyderabad, India. The guidelines of Helsinki declaration of 1975 as revised in 2000, were strictly followed while conducting the study. This study was approved by the Institutional Review Board and registered in clinical trials registry (NCT 02503046). A written informed consent form was signed by each patient before the start of the study. The total sample size was 90 which included both male and female subjects. They were further equally divided (30 Subjects each) into three groups and were aged between 30-60 years.

Inclusion Criteria

Group I included patients who were diagnosed with RA with chronic periodontitis and willing to participate in the study (n=30). They were further subdivided into Group IA: Just diagnosed RA patients with chronic periodontitis (n=15) and Group IB: RA patients with chronic periodontitis who were under medication for more than three months (n=15). Group II patients included those with chronic periodontitis only (n=30), whereas Group III comprised of healthy subjects taken as controls (n=30)

Exclusion Criteria

Patients with systemic diseases other than RA, smokers, pregnant and lactating women and those who had undergone periodontal treatment within the last three months.

Clinical Parameters

Samples that satisfied periodontal disease as *a priori* according to the definition of Machtei EE et al., (presence of clinical attachment loss ≥ 6 mm on ≥ 2 teeth and one or more sites with probing depths ≥ 5 mm and presence of 20 teeth) [6] and the 1987 American College of Rheumatology (ACR) classification criteria for RA [7] {morning stiffness, at least three areas of swollen joints, arthritis of hand joints, symmetric arthritis, rheumatoid nodules, serum rheumatoid factor +ve} where clinically assessed using a William's probe. The indices assessed were the PI [8], GI [8], Pocket Depth (PPD) and CAL.

Microbiological Analysis

Subgingival plaque was collected from sites with probing depths >5 mm, following removal of visible supragingival plaque, using sterile Gracey curettes. Each plaque sample was placed in an Eppendorf tube containing 150 µl of sterile TE buffer (10 mM Tris–HCl, 1 mM EDTA, pH 7.6). Then 100 µl of 0.5 M NaOH was added to the plaque pellet and the bacterial suspension was stored at -20°C pending further processing, DNA extraction was done by Modified Proteinase-K method [9] and the level of *P.Gingivalis* was estimated using Polymerase Chain reaction set up.

PCR Procedure

The Following set of PCR primers were used which was specific to *P.Gingivalis.* Amplicon size 404 bp. Forward primer: AGG CAG CTT GCC ATA CTG CG. Reverse primer: ACT GTT AGC AAC TAC CGA TGT. Samples were kept at 4°C following PCR. Amplified products were subjected to electrophoresis through 2% Agarose gel containing 1x TAE Buffer (Tris Base-12.1gm, Glacial acetic acid-2.850 ml and 0.5 M EDTA- 5 ml) [Table/Fig-1] [10]. The reagents used for this study included, PCR Master mix: Ampliqon, Oligonucleotide Primers- Bioserve India Pvt. Ltd., Agarose powder-Puregene, Genetix, and the equipment made use of included: Thermal cycler: Applied Biosystems, USA, Electrophoresis apparatus: Bio bee Tech, Bangalore, Gel Documentation system: Major Science, USA.

[Table/Fig-1]: Band identification for *P.Gingivalis* by PCR; **[Table/Fig-2]** Quantikine ELISA Kit for Pentraxin3 estimation.

Immunological Analysis

A 2 ml of blood sample from each subject was collected in Ethylene Diamine Tetra-acetic Acid (EDTA) containing vials and plasma was separated from samples stored at -20°C, to evaluate pentraxin 3 by Quantikine ELISA method [Table/Fig-2] [11].

Primary and Secondary Outcome Measures: The primary outcome measures asessed were the Serum PTX3 levels (pg/ml) and sub-gingival *P.Gingivalis* levels (Cfu/ml). The secondary outcome measures assessed were the clinical parameters (i.e.,) GI, PI, PD and CAL.

STATISTICAL ANALYSIS

To get a mean difference of 0.43 in Pentraxin 3 levels before and after medication in Group I with power at 80% and level of significance 5%, it was seen that a minimum of 30 samples per group would be sufficient. Data was analysed by SPSS version 16.0; SPSS Inc., (Chicago, IL, USA). The comparison between the three groups (Intergroup) for continuous data (Clinical, Microbiological and Biochemical parameters) was done by One-way analysis of variance test followed by Bonferroni's post-hoc test. The comparison between pre and post medication (Group I- Intragroup) for continuous data (Clinical, Microbiological and Biochemical parameters) was done by paired t-test. All p-values less than 0.05 were considered statistically significant.

RESULTS

In this study, the intergroup comparison between Group I (RA with chronic periodontitis) and Group II (chronic periodontitis patients only) showed that the clinical parameters like PI, PD, CAL were not significantly different. Higher values have been observed in relation to GI in Group II patients when compared with Group I [Table/Fig-3]. However, Intragroup comparison of Group I patients who were equally divided into Group IA (Early diagnosed RA) and Group IB (RA patients under medication for more than three months) showed that in group IB lower values were observed in relation to PI, GI, and CALs. However, there was not much difference in the Probing depth within the two groups [Table/Fig-4].

Microbiological Assessment

The intergroup comparison for *P. Ginigvalis* was not significantly different in Group I and Group II patients, though when each of the experimental groups were compared to the control group (Group III) the values of *P.Gingivalis* was statistically significant ($p<0.001$) [Table/Fig-3]. However, an intragroup comparison between Group I A (mean 3.43 and SD 1.78) and Group I B (mean 2.49 and SD 0.67) for *P.Gingivalis* yielded a p-value 0.048 which was found to be statistically significant [Table/Fig-5].

Biochemical Assessment

When an intergroup comparison was done for Pentraxin 3 levels between Group I and Group II, the values were not statistically significant, [Table/Fig-3] however when a comparison was made within Group I it was observed that in patients belonging to Group I B (RA patients under medication), the levels were much lower (mean 1.31 and SD 0.31) when compared to Pentraxin 3 levels in patients from Group I A (mean 1.74 and SD 0.42) [Table/Fig-6].

DISCUSSION

Periodontitis is a globally rampant disease causing the destruction of connective tissue and bone with loss of teeth as the final outcome. This disease involves an interplay between plaque, microorganisms and the host response [12-14].

RA is a chronic inflammatory disease which not only affects the joints but also damages other organs like the eyes, lungs, skin, blood vessels etc. Patients afflicted with this disease often complain of pain, stiffness, swelling and limited movement and function of the joints [4].

Periodontitis has been linked to many systemic diseases like Diabetes Mellitus [15], Coronary Artery Disease [16], Chronic Obstructive Pulmonary Disease [17], Preterm birth [18] and RA [19] to name a few. Both periodontitis and RA are chronic inflammatory diseases which share many common risk factors. In both diseases, the Cytokines IL-1, IL-6, and TNFα are expressed more with deleterious effects on the host [3].

In this study, the intergroup comparison between Group I (RA with chronic periodontitis) and Group II (chronic periodontitis patients only) showed that the clinical parameters like PI, PD, CALs were not significantly different. Higher values were observed in relation to GI in Group II patients when compared with Group I [Table/Fig-3]. However, Intragroup comparison of Group I patients who were equally divided into Group IA (Early diagnosed RA) and Group IB (RA Patients under medication for more than three months) showed that in Group IB lower values were observed in relation to PI, GI, and CALs. However, there was not much difference in the PD within the two groups [Table/Fig-4].

Parameter	Groups	N	Mean	SD	p-value	(I) Groups	(J) Groups	Mean Difference (I-J)	p-value
PI	Group I	30	1.86	0.63	< 0.0001	Group I	Group II	0.056	1.000
	Group II	30	1.80	0.46			Group III	0.825	< 0.0001
	Group III	30	1.03	0.48		Group II	Group III	0.769	< 0.0001
GI	Group I	30	1.59	0.50	< 0.0001	Group I	Group II	0.483	< 0.0001
	Group II	30	2.07	0.39			Group III	1.037	< 0.0001
	Group III	30	0.55	0.29		Group II	Group III	1.520	< 0.0001
PD	Group I	30	6.70	1.12	< 0.0001	Group I	Group II	0.200	1.000
	Group II	30	6.90	1.27			Group III	3.867	< 0.0001
	Group III	30	2.83	0.75		Group II	Group III	4.067	< 0.0001
CAL	Group I	30	6.17	1.15	< 0.0001	Group I	Group II	0.200	1.000
	Group II	30	6.37	1.56			Group III	4.700	< 0.0001
	Group III	30	1.47	1.20		Group II	Group III	4.900	< 0.0001
Pentraxin 3	Group I	30	3.60	1.66	< 0.0001	Group I	Group II	0.503	0.523
	Group II	30	3.09	1.82			Group III	3.597	< 0.0001
	Group III	30	0.00	0.00		Group II	Group III	3.093	<0.0001
P.gingivalis (CFU/ml)	Group I	30	2.96	1.40	< 0.0001	Group I	Group II	0.51	0.166
	Group II	30	2.45	1.07			Group III	2.96	< 0.0001
	Group III	30	0.00	0.00		Group II	Group III	2.45	<0.0001

[Table/Fig-3]: Comparison of clinical, biochemical and microbiological parameters between groups.
Clinical parameters- PI-Plaque index, GI-Gingival index, PD- Probing depth, CAL -Clinical attachment levels.
†SD- Standard deviation; ‡N-Number of patients.
One-way analysis of variance test, followed by Bonferroni's test

Parameters	Groups	N	Mean	SD	p-value
PI	Group I A	15	2.33	0.33	0.001
	Group I B	15	1.38	0.48	
GI	Group I A	15	1.91	0.34	0.002
	Group I B	15	1.26	0.42	
PD	Group I A	15	6.87	1.19	0.403
	Group I B	15	6.53	1.06	
CAL	Group I A	15	6.53	1.06	0.022
	Group I B	15	5.80	1.15	

[Table/Fig-4]: Intragroup comparison (Group I) of clinical parameters. Paired t-test. used for statistical analysis.
* Clinical parameters- PI-Plaque index, GI-Gingival index, PD- Probing depth, CAL-Clinical attachment levels
† Group I A – Just diagnosed patients with RA and Group I B – RA patients on medication for more than three months
†SD- Standard deviation
§ N-Number of patients

Groups	N	Mean	SD	SEM	Minimum	Maximum	p-value
Group I A	15	3.43 CFU/ml	1.78	0.459	1.1	7.3	0.048
Group I B	15	2.49 CFU/ml	0.67	0.173	1.6	3.5	

[Table /Fig-5]: Intra-group comparison of *P. gingivalis* (CFU/ml).
Paired t-test used for statistical analysis
* CFU/ml- Colony forming units per millilitre
† Group IA (RA patients just diagnosed)
‡ Group IB (RA patients under medication for more than three months

Groups	N	Mean	SD	SEM	Minimum	Maximum	p-value
Group I A	15	1.74 pg/ml	0.42	0.110	1.005	2.531	0.004
Group I B	15	1.31 pg/ml	0.31	0.081	1.023	1.902	

[Table/Fig-6]: Intra-group comparison of serum Pentraxin 3 (pg/ml).
Paired t-test used for statistical analysis
* pg/ml-picograms per millilitre
† N- Number of patients
SD- Standard deviation
‡ SEM- Standard error of mean
§ Group IA- Just diagnosed patients with RA, Group I B-RA patients on medication for more than three months

High levels of antibodies to oral bacteria in the serum and synovial fluid of patients with RA and periodontitis was observed by Ogrendik M. Raised titres of *P.Gingivalis* and T.Forsythtia were detected in the synovial fluid of patients with RA [20].

Another study threw light on the capability of *P.Gingivalis* to produce an enzyme capable of modifying specific proteins. The bacterial enzyme catalyses a protein and structurally modifies it which is recognised by the host as a foreign body. The body thus mobilizes an autoimmune response to these proteins, which culminates clinically in the joint destruction typically seen in RA-susceptible individuals [21].

In this study, intergroup comparison between Group I and Group II showed that the *P.Gingivalis* levels were not statistically significant between the groups. However, when an intragroup comparison was made between Group IA and Group IB, it was observed that the *P.Gingivalis* levels were significantly less in Group IB, which could be attributed to the patients taking medication [Table/Fig-5].

Several studies have been performed assessing the role of salivary biomarkers in healthy and disease. Accordingly, it has been observed that levels of IL-6, MMP'S 8 and 9, TNFα, Receptor Activator Of Nuclear Kappa B Ligand (RANKL) are elevated in the saliva and serum of patients with periodontitis and RA [1]. Pentraxin 3 has been researched a lot recently. The level of this biomarker has been observed to be elevated in inflammatory conditions. Fatty liver [22], Systemic lupus erythmatosis [23], Retinal disorders due to diabetes [24], are but a few of the innumerable conditions wherein the levels of this protein are increased.

Researchers observed that in patients with atherosclerosis and RA the serum levels of both Pentraxin 3 and IL-6 were elevated. They concluded that critical management of RA patients with elevated levels of sPTX3 and sIL 6 is very essential to prevent cardiovascular complications [25].

In another study, it was observed that female patients with RA have increased concentrations of Pentraxin 3 compared with control subjects. Pentraxin 3 was significantly associated with radiographic progression of joint damage but not with carotid atherosclerosis in RA [26].

In yet another study, it was observed that when the disease progresses from gingivitis to periodontitis pentraxin 3 levels increases in GCF and plasma, and that plasma pentraxin 3 levels are not significant when compared to GCF pentraxin 3 levels [27]. A group of researchers have done a study in generalized

aggressive periodontitis and generalized chronic periodontitis patients to estimate pentraxin 3 levels in gingival tissues. In their study, generalized aggressive periodontitis patients showed higher pentraxin 3 levels when compared to generalized chronic periodontitis and healthy group [28].

This study was in accordance with the previous studies in relation to increased levels of Pentraxin 3 in diseased conditions. But an intra group comparison between Group IA and Group IB, showed that the levels of Pentraxin 3 were reduced in group IB, which could be attributed to these patients taking anti rheumatoid medication [Table/Fig-6].

LIMITATION

This study was an observational study, perhaps the role of *Porphyromonas gingivalis* and Pentraxin 3 as diagnostic markers could have been validated better if the patients were treated by nonsurgical periodontal therapy (scaling and root planning). This study though observational, has shown that in patients treated for RA (Group IB) the levels of both *Porphyromonas gingivalis* and Pentraxin 3 were reduced when compared to the other experimental groups (Group IA and Group II). Perhaps these two markers could be used as diagnostic tools for both the diseases.

CONCLUSION

This study has shown that both *Porphyromonas gingivalis* and Pentraxin 3 levels were found to be increased in patients with RA and periodontitis. However, the levels of both these markers decreased after treatment in Group I B. This observation throws light on the possibility that these markers could be used as diagnostic tools to assess both diseases, but more longitudinal and interventional studies should be conducted to validate their role.

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