International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

Effects of Phase I Periodontal Therapy on Rheumatoid Arthritis: A Clinical Study

[1]Swarga Jyoti Das, [1, 2]Salome Zonunsangihnamte, [3]Chitralekha Baruah

[1]*Department of Periodontics, Regional Dental College, Guwahati, Assam*
[2]*District Hospital, Siaha, Mizoram*
[3]*Department of Medicine, Gauhati Medical College, Guwahati, Assam*

[*]*Corresponding Author: Swarga Jyoti Das*

Date of Submission: 25-12-2020 Date of Acceptance: 10-01-2021

ABSTRACT: ***Background:*** Systemic health has often been closely linked to the state of oral cavity. In the recent past, there has been a greater concern in understanding the association of periodontal disease with many systemic conditions, including rheumatoid arthritis (RA). Since periodontitis and RA are inflammatory in nature and associated with inflammatory cytokine-mediated bone destruction, it is possible that they may have a linking mechanism. Keeping this in mind, the present study was proposed to evaluate the association of periodontal health and RA, and also the effectiveness of phase I periodontal treatment on the severity of RA. ***Materials and methods***: Subjects with RA were selected (n=48) and randomly assigned into 2 groups: A (Control: received no periodontal therapy) (n=25) and B (Test: received oral hygiene instructions, full-mouth scaling and root planing) (n=23). Periodontal health was assessed by using simplified oral hygiene index, gingival index, probing pocket depth and clinical attachment level on day 0 (baseline) and on day 45 and 90. Similarly, disease activity in RA was evaluated using Disease Activity Score 28 (DAS28). ***Results***: A positive and highly significant correlation between DAS28 index and periodontal health was observed. The DAS28 index was reduced in test group in addition to the significant improvement in all the periodontal parameters compared to that of control. ***Conclusion***: We may conclude that poor periodontal health plays an important role on the severity of RA, which may be improved through the promotion of oral health care and awareness program.

KEYWORDS: Periodontitis, Rheumatoid arthritis, inflammatory cytokines, DAS28, Pocket depth.

I. INTRODUCTION

Periodontal disease is an inflammatory disease caused predominantly by Gram-negative, anaerobic, microaerophilic bacteria that colonise the subgingival area, and is modified by environmental and stress-related behavioural factors (e.g. consumption of alcohol, smoking). It results in progressive destruction of the periodontal ligament and alveolar bone, manifested clinically as pocket formation, gingival recession and/or both[1]. Throughout the history of mankind, it has been believed that oral diseases and maladies including periodontal disease may have an effect on the rest of the body [2, 3]. Thus, the concept of linking periodontitis and systemic diseases including rheumatoid arthritis (RA) could be traced back to the beginning of recorded history and medicine.

Rheumatoid arthritis (RA) is an autoimmune disease, characterised by an accumulation and persistence of an inflammatory infiltrate in the synovial membrane that leads to synovitis and subsequently, destruction of the joint architecture resulting in impaired function. It can affect any joint but commonly involves the wrist and fingers [4]. RA is of particular interest since it demonstrates remarkably similar patterns of soft and hard tissue destruction that noted in chronic periodontitis [5].

Though aetiologies of periodontitis and RA are distinctly separate, both are of inflammatory in nature and characterised by an imbalance between proinflammatory and antiinflammatory cytokines. Again, both of them are associated with destruction of bone, mediated by inflammatory cytokines (e.g. IL-1, TNF and PGE2) activated by the host response[6,7,8]. Moreover, detection of periodontal pathogens, namely *P. gingivalis*, *T. forsythia*, and *P. intermedia* and antibodies to these pathogens in the serum and synovial fluid of RA patients suggest a direct association of oral bacteria with etiopathogenesis of RA[9]. It has been shown that

International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

RA patients are at higher risk of developing periodontitis compared to that of the subjects without RA[10,11]. Some studies have even suggested that RA could trigger and/or worsen periodontitis and inversely, periodontitis could add to the inflammatory state and maintain systemic inflammation in RA, thus a possibility of bi-directional relationship between these two conditions exist[12,13]. Schematic representation of the mechanisms by which periodontal inflammation may affect RA is shown in Figure 1.

Considering this fact, we may contemplate periodontal therapy directed at elimination of pathogenic organisms and reduction of inflammation may also influence in the severity of RA. However, contradictory observations have been reported in the literature [14,15,16,17,18]. Keeping this in mind, the present study was conducted to evaluate the association of periodontitis and RA and subsequently, the effectiveness of phase I periodontal therapy on the severity of RA.

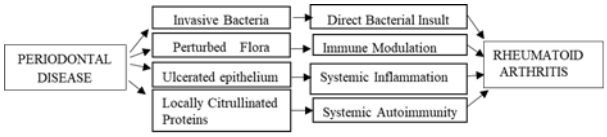

Fig.1: Schematic representation of mechanisms by which periodontal inflammation may affect rheumatoid arthritis.

II. MATERIALS AND METHODS

The study was conducted jointly in the Department of Periodontics and Oral Implantology, Regional Dental College, Guwahati and in the Rheumatology Clinic, Department of Medicine, Gauhati Medical College and Hospital, Guwahati, which is a Tertiary Care Referral Hospital in the entire North Eastern Region. The study was carried out in accordance with the ethical guidelines of the Institutional Research and Ethical Committee.

The subjects (n=48) were randomly selected from the Rheumatology O.P.D., comprising of females (n=40) and males (n=8). The following norms were applied for selection:

Inclusion Criteria:
- RA patients diagnosed by the physician
- 20 - 65 years old subjects
- Subjects having at least 20 teeth excluding last molars

Exclusion Criteria:
- Subjects with any other systemic diseases other than RA
- Smokers and tobacco users
- Pregnant and lactating women
- Subjects who underwent periodontal treatment in the last 6 months
- Subjects on antibiotics in the last 3 months

The entire procedure in details was explained to the subjects and written consent was taken.

The subjects were randomly categorized into two groups:
- Group A (Control) (n=25): did not receive any periodontal therapy
- Group B (Test) (n=23): received phase I periodontal therapy consisting of oral hygiene instructions, full-mouth scaling and root planing.

Thorough medical history and the laboratory investigation of each subject were obtained in detail.

The subjects were diagnosed according to the American College of Rheumatology/European League against Rheumatism (ACR/EULAR) 2010 classification criteria (Kay and Upchurch, 2012)[19], shown in Table 1.

Table 1: ACR/EULAR 2010 Rheumatoid Arthritis Classification Criteria

Points	0	1	2	3	5
Swollen painful joints	≤ 1 (medium to large joints)	2 - 10 (medium to large joints)	1 - 3 small joints	4 - 10 small joints	≥ 11 including small joints
Serology RF & ACPA (Anti-citrullinated protein antibodies)	Negative		One or both weakly positive	One or both strongly positive	
Acute phase CRP & ESR	Normal	One or both elevated			
Duration of symptoms	< 6 weeks	≥ 6 weeks			

DISEASE ACTIVITY SCORE 28 (DAS28)

It is a widely used index of disease activity in RA patients. Number 28 refers to the 28 joints that are examined in this assessment for tenderness and swelling. The joints considered for assessment of tenderness and swelling are proximal interphalangeal joints (PIJ or PIP) (n=10), metacarpophalangeal (MCP) joints (n=10), wrists (n=2), elbows (n=2), shoulders (n=2) and knees (n=2).

'Global assessment of health' (GH) was assessed using a Visual Analogue Scale marking a 10 cm line between very good (score 0) and very poor (Score 10).

International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

DAS28 is calculated using a software programme applying the following formula:

[0.56 x sqrt (tender joint count 28)] + [0.28 x sqrt (Swollen joint count 28)] + [0.70 x (ESR)] + [0.014 x global health]

And it is interpreted as
- High (> 5.1)
- Moderate (3.2 - 5.1)
- Low (< 3.2) and
- Remission (< 2.6)

PERIODONTAL PARAMETERS:

A full mouth periodontal examination was performed in all the subjects on day 0 using the following clinical parameters:

- Simplified Oral Hygiene Index (OHI-S) (Debris and Calculus Index)
- Gingival Index (GI)
- Probing Pocket Depth (PPD)
- Clinical Attachment Level (CAL)

Simplified Oral Hygiene Index (OHI-S) (Greene and Vermilion, 1964)[20]:

This index is used to classify and assess the oral hygiene status. It comprises of two components, the Debris index-Simplified (DI-S) and the Calculus index-Simplified (CI-S). Six tooth surfaces are examined for DI-S and CI-S. Scoring was done on the following teeth and surfaces: buccal surfaces of 16 and 26, labial surfaces of 11 and 31, and lingual surfaces of 36 and 46.

Debris Index - Simplified (DI-S):

The mouth was examined first for debris (i.e., DI-S). The surface area covered by debris was estimated by running the side of the No. 5 explorer along the tooth surface to be examined. The occlusal or incisal extent of the debris was determined and recorded as follow:

- 0 = No debris or stain present.
- 1 = Soft debris covering not >1/3 of the tooth surface, and/or the presence of extrinsic stains without other debris regardless of the surface area covered.
- 2 = Soft debris covering >1/3, but not >2/3 of the exposed tooth surface.
- 3 =Soft debris covering >2/3 of the exposed tooth surface.

DI-S of a subject is obtained by dividing the sum of the debris scores of designated surfaces of index teeth by the number of surfaces evaluated.

Calculus Index - Simplified (CI-S):

The CI-S was measured by using an explorer to estimate the surface area covered by supragingival calculus and probed for subgingival calculus. The following criteria were used:

- 0 = No calculus present.
- 1 = Supragingival calculus covering not > 1/3 of the exposed tooth surface.
- 2 = Supragingival calculus covering > 1/3 but not > 2/3 of the exposed tooth surface and/or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth.
- 3 = Supragingival calculus covering > 2/3 of the exposed tooth surface and/or the presence of a continuous band of subgingival calculus around the cervical portion of the tooth.

The calculus score for each surface was added and then divided by the number of surfaces examined to obtain the CI-S.

The DI-S and CI-S were interpreted as follows:
- Good = 0.0 - 0.6
- Fair = 0.7 - 1.8
- Poor = 1.9 - 3.0

OHI-S for each subject was calculated by totalling of DI-S and CI-S and interpreted as:
- Good = 0.0 - 1.2
- Fair = 1.3 - 3.0
- Poor = 3.1 - 6.0

Gingival index (GI) (Loe and Silness, 1963)[21]:

The gingival health status was assessed using a mouth mirror and a periodontal probe on a numerical scale. The following criteria were used:

- 0 = Normal gingiva
- 1=Mild inflammation: slight change in colour and slight oedema; no bleeding on probing.
- 2=Moderate inflammation: redness, oedema and glazing; bleeding on probing.
- 3=Severe inflammation: marked redness and oedema; ulceration; tendency to spontaneous bleeding.

The Gingival score for a tooth was obtained by dividing the sum of scores obtained at four areas by four. Then scores of each tooth are added and divided by the number of teeth examined to acquire gingival index scores for an individual.

International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

Probing Pocket Depth (PPD):

Probing Pocket depth (PPD) was measured using the UNC -15 periodontal probe. The working end of this probe is 15 mm long with markings at each millimeter and colour coding at 5^{th}, 10^{th} and 15^{th} mm. The probe was inserted with a firm, gentle pressure (0.75 N) to the bottom of the pocket aligning the shank with the long axis of the tooth surface to be probed. PPD was measured from gingival margin to base of the pocket in mm, at four specific points in relation to a tooth: distofacial and mesiofacial line angles, middle of facial and lingual surfaces.

PPD of each tooth was obtained by dividing the sum of depth obtained at four areas by four. Then pocket depth of each tooth was added and divided by the number of teeth examined to acquire pocket depth for an individual subject.

Clinical attachment level (CAL):

Clinical attachment level (CAL) was measured using an 'Occlusal Stent'. It was measured from a fixed reference point to the base of the pocket. Here, the coronal border of the stent was considered as a fixed reference point. It was measured in mm using UNC -15, keeping the probe on vertical grooves prepared on the occlusal stent as a reference point to avoid clinical variations at different time points of measurement[22]. CAL for each subject was determined by adding all the individual scores and then dividing this by the total number of surfaces recorded.

The patients were re-evaluated on day 45 and 90 for periodontal health and disease activity in RA using periodontal parameters and DAS28, respectively.

The data were collected and analysed statistically using SPSS (SPSS Inc, Chicago) version 17.0 software packages. Pearson's correlation coefficient was applied to assess the relationship between the measurements. Independent 't' tests was used to evaluate whether the difference in the means between two independent variables at each level is statistically significant or not. Paired 't' test was used to evaluate the significance of difference in various parameters at different time points, i.e. day 0, 45 and 90.

III. RESULT

The study was proposed to evaluate the association of periodontal health and DAS28 in RA patients, and to assess the effectiveness of phase I periodontal therapy on the severity of RA.

The subjects were randomly divided into 2 groups, Group A (Control) and B (Test), comprising of 50 in total initially. However, 2 no of subjects from group B left the study midway, making the total no of subject 48, consisting 25 in group A and 23 in group B. Control group received no treatment and test group received phase I periodontal therapy and were given oral hygiene instructions.

To determine the association of periodontitis and RA, correlation coefficient of DAS28 and the periodontal parameters were plotted. As shown in Table 2, the 'r' values between DAS28 and OHI-S, GI, PPD and CAL were found to be 0.54, 0.52, 0.44 and 0.55, respectively. This indicates a very strong positive correlation between the DAS28 and each of the periodontal parameters used, which is evidently prominent on scatter plot regression line, i.e. more the steepness towards right, stronger the correlation (Figure 2). Thus, it suggests that a subject with RA having higher DAS28 (Severe form) possesses higher values of OHI-S, GI, PPD and CAL in comparison to that of a subject with less severe RA.

Table 2: Correlation Coefficient of DAS28 with periodontal parameters

Parameters	Pearson's Correlation Coefficient	
	r-value	p value
DAS28& OHI-S	0.54	0.000***
DAS28& GI	0.52	0.000***
DAS28& PPD	0.44	0.002**
DAS28& CAL	0.55	0.000***

**highly significant; ***very highly significant

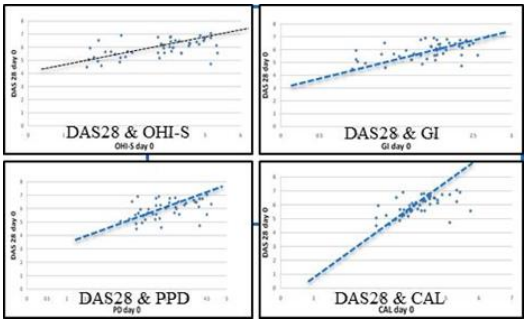

Fig. 2: DAS28 and periodontal parameters on day 0. Note the positive and highly significant correlation between DAS28 and all periodontal parameters, namely Oral hygiene index-S (OHI-S), Gingival Index (GI), Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL).

International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

Based on the positive correlation between RA and periodontal status, second part of the study was carried out to evaluate the effects of phase I periodontal therapy on DAS28 and periodontal parameters.

Disease Activity Score 28 (DAS28):

A comparison of the mean DAS28 between group A and B on day 0, 45 and 90 was carried out. On day 0, the mean DAS28 in group A was 5.84 ± 0.72 (range from 3.28 to 6.75), which decreased to 5.41 ± 0.69 (range from 3.92 to 7.58) and 4.52 ± 0.72 (range from 3.27 to 5.88) on day 45 and 90, respectively, as shown in Table 3. Thus, the differences in DAS28 score between day 0 and 45 (pair 1), day 45 and 90 (pair 2) and day 0 and 90 (pair 3) were 0.44, 0.88 and 1.32, respectively. In all the 3 pairs, the correlation between each variables was found to be positive and very highly significant statistically ($p<0.001$).

In group B, the mean DAS28 on day 0 was 6.04 ± 0.62 (range from 3.79 to 7.27). On day 45, it reduced to 5.26 ± 0.66 (range from 3.84 to 6.63), which is further reduced to 2.80 ± 0.64 (range from 1.82 to 3.68) on day 90. Thus, the differences in DAS28 score between day 0 and 45 (pair 1), day 45 and 90 (pair 2) and day 0 and 90 (pair 3) were 0.78, 2.46 and 3.24, respectively. In all the 3 pairs, the differences were statistically very highly significant ($p<0.001$) and the correlation between each variables was found to be positive and significant ($p<0.05$). In test group, DAS28 shifted from high to remission, i.e. the mean DAS28 value was 6.04 ± 0.62 on day 0, which became 2.80 ± 0.64 on day 90.

Table 3: Intergroup comparison of DAS28 at different time points

DAS28 (Day)	Groups	Mean ± SD (range)	Mean Difference	*p* value
0	A (n = 25)	5.84 ± 0.72 (3.28 - 6.75)	- 0.20	0.30^{ns}
	B (n= 23)	6.04 ± 0.62 (3.79 - 7.27)		
45	A (n = 25)	5.41 ± 0.69 (3.92 - 7.58)	0.14	0.46^{ns}
	B (n= 23)	5.26 ± 0.66 (3.84- 6.63)		
90	A (n = 25)	4.52 ± 0.72 (3.27 - 5.88)	1.72	0.000^{***}
	B (n= 23)	2.80 ± 0.64 (1.82 - 3.68)		

ns= not significant; ***= very highly significant

The mean differences in DAS28 score between group A and B were - 0.20, 0.14 and 1.72 on day 0, 45 and 90, respectively. The mean differences in DAS28 scores on day 0 and 45 were not statistically significant ($p = 0.30$ and $p = 0.46$, respectively). However, the intergroup difference in DAS28 score on day 90 was very highly significant statistically ($p > 0.001$).

Simplified Oral Hygiene Index (OHI-S)

Both intra- and intergroup comparison of the mean OHI-S was carried out on day 0, 45 and 90. On day 0, the mean OHI-S in group A was 3.71 ± 1.12 (range from 1.66 to 5.16), which further increased to 3.77 ± 1.11(range from 1.75 to 5.35) and 3.81 ± 1.18 (range from 1.32 to 5.18) on day 45 and 90, respectively. Thus, the mean difference in OHI-S between day 0 and 45 was -0.06, which was found to be statistically significant ($p<0.05$) and the correlation between the two variables was found to be positive and highly significant. In contrast, the mean difference in OHI-S between day 45 and 90 (-0.04) was not statistically significant ($p = 0.68$). Again, the mean difference in OHI-S between day 0 and 90 was -0.10, and found to be statistically significant ($p<0.05$).

As shown in Table 4, the mean OHI-S in group B was 3.67 ± 1.17 (range from 1.32 to 5.18) on day 0, which is reduced to 2.59 ± 1.14 (range from 1.21 to 4.15) and 1.55 ± 0.87 (range from 0.56 to 2.13) on day 45 and 90, respectively. Thus, the mean difference in OHI-S between day 0 and 45 was 1.08 and the correlation between the two variables was found to be positive and highly significant. Again, the mean differences of OHI-S between day 45 and 90, and day 0 and 90 were 1.04 and 2.12, respectively. At both time points, the correlation between the two variables was found to be positive and very highly significant ($p<0.001$).

As shown in Table 4, the mean differences in OHI-S between group A and B were 0.04, 1.18 and 2.26 on day 0, 45 and 90, respectively. The mean differences on day 45 and 90 were statistically very highly significant ($p<0.001$), though it was found to be not significant statistically on day 0 ($p=0.90$).

Gingival index (GI):

As shown in Table 4, the mean GI score in group A was 1.92 ± 0.44 (range 0.89 to 2.56), which is found to be raise to 2.05 ± 0.49 (range 1.18 to 2.84) and 2.24 ± 0.46 (range 1.94 to 2.62) on day 45 and 90, respectively. Thus, the mean differences in GI score between the day 0 and 45 (pair 1), was - 0.13, while between the day 45 and 90 (pair 2), and day 0 and 90 (pair 3), were - 0.18

and -0.32, respectively. In all the 3 pairs, the correlation between each variables was found to be positive and highly significant ($p<0.05$), except the pair 2 where $p>0.05$.

In group B, the mean GI of day 0 was 1.91 ± 0.46 (range from 0.89 to 2.87). On day 45, it reduced to 1.35 ± 0.46 (range from 1.05 to 2.86), which further reduced to 0.92 ± 0.15 (range from 1.03 to 2.86) on day 90 (Table 4). Thus, the mean differences in GI scores between the day 0 and 45, day 45 and 90, and day 0 and 90 were 0.56, 0.43 and 0.99, respectively. The differences at all three points were statistically very highly significant ($p<0.001$) and the correlation between each variables was found to be positive and statistically significant ($p<0.05$).

As shown in Table 4, the mean differences in GI score between group A and B were 0.01, 0.70 and 1.32 on day 0, 45 and 90, respectively. The mean intergroup differences in GI on day 45 and 90 were found to be statistically very highly significant ($p<0.001$). However, the difference in GI on day 0 was not significant statistically ($p = 0.95$).

Probing Pocket Depth (PPD):

Both intra- and inter- group comparison of the mean PPD was carried out on day 0, 45 and 90. On day 0, the mean PPD in group A was found to be 3.37 ± 0.53(range 2.59 to 4.21), which further increased to 3.44 ± 0.51(range 2.45 to 4.50) and 3.69 ± 0.65 (range 1.56 to 4.91) on day 45 and 90, respectively, as shown in Table 4. Thus, the mean differences in PPD between the day 0 and 45 (pair 1), was -0.07, while between day 45 and 90 (pair 2), and day 0 and 90 (pair 3), were -0.25 and -0.32, respectively. The correlation between each variables was found to be positive and highly significant ($p<0.001$) in all the 3 pairs.

In group B, the mean PPD on day 0 was 3.51 ± 0.70 (range 2.68 to 4.35). The mean PPD was reduced to 3.22 ± 0.63 (range 2.31 to 4.21) on day 45, which further reduced to 2.85 ± 0.70 (range 1.21 to 4.15) on day 90. Thus, the mean differences in PPD between day 0 and 45 (pair 1) was 0.29, while PPD between day 45 and 90 (pair 2), and day 0 and 90 (pair 3) were 0.37 and 0.66, respectively. In all the 3 pairs, the differences were statistically very highly significant ($p<0.001$) and the correlation between each variables was found to be positive and very highly significant ($p<0.001$).

As shown in Table 4, the mean differences in PPD between group A and B were -0.14, 0.22 and 0.82 on day 0, 45 and 90, respectively. The mean difference in PPD on day 90 was found to be 90 (pair 2), and day 0 and 90 (pair 3), were - 0.18 and -0.32, respectively. In all the 3 pairs, the statistically very highly significant ($p<0.001$). However, the differences in PPD on day 0 and 45 were not significant statistically ($p=0.42$ and 0.19, respectively.

Clinical attachment level (CAL):

Both intra- and inter- group comparison of the mean CAL was carried out on day 0, 45 and 90. On day 0, the mean CAL in group A was found to be 4.06 ± 0.65 (range 2.99 to 5.75), which further increased to 4.07 ± 0.60 (range 2.73 to 5.35) and 4.16 ± 0.73 (range 2.89 to 5.41) on day 45 and 90, respectively (Table 4). Thus, the mean differences in CAL between the day 0 and 45 (pair 1) was -0.01, between on day 45 and 90 was -0.09 (pair 2) and between the day 0 and 90 was -0.10 (pair 3). In first two pairs, the correlation between each variables was found to be not significant statistically ($p>0.05$), while in pair 3, the correlation between each variables was found to be very highly significant statistically ($p<0.001$).

The mean CAL in group B was 4.30 ± 0.66 (range 2.59 to 5.25) on day 0, which reduced to 3.39 ± 0.85 (range 1.56 to 4.91) and 2.88 ± 0.90 (range 1.56 to 3.91) on day 45 and 90, respectively. Gain in attachment was seen after phase I periodontal therapy in group B. Thus, the mean differences in CAL between the day 0 and 45 (pair 1) was 0.91, between on day 45 and 90 was 0.51 (pair 2) and between the day 0 and 90 was 1.42 (pair 3). In all the 3 pairs, the differences were statistically very highly significant ($p<0.001$) and the correlation between each variables was found to be positive and very highly significant ($p<0.001$).

The mean intergroup differences in CAL on day 45 and 90 (0.69 and 1.28, respectively) were found to be statistically highly significant ($p<0.001$). However, the difference in CAL on day 0 (- 0.23) was found to be not significant statistically ($p=0.22$).

IV. DISCUSSION

Systemic health has often been closely linked to the state of the oral cavity. Therefore, in the recent past, there has been greater concern in understanding the association of periodontal disease with many systemic conditions. Indeed, animal- and population-based studies now suggest that periodontal diseases may be linked with systemic diseases including cardiovascular diseases, diabetes mellitus, adverse pregnancy outcomes, osteoporosis, respiratory diseases and rheumatoid arthritis (RA) [23-26].

International Journal Dental and Medical Sciences Research
Volume 3, Issue 1, Jan-Feb 2021 pp 289-298 www.ijdmsrjournal.com ISSN: 2582-6018

Table 4: Periodontal parameters in control and Test at various time

Days	OHI-S Mean ± SD (Range)		Gingival Index Mean ± SD (Range)		Probing Pocket Depth (in mm) Mean ± SD (Range)		Clinical Attachment Level (in mm) Mean ± SD (Range)	
	Group A (n= 25)	Group B (n= 23)	Group A (n= 25)	Group B (n= 23)	Group A (n= 25)	Group B (n= 23)	Group A (n= 25)	Group B (n= 23)
Day 0	3.71 ± 1.12 (1.66 - 5.16)	3.67 ± 1.17 (1.32 - 5.18)	1.92 ± 0.44 (0.89 - 2.56)	1.91 ± 0.46 (0.89 - 2.87)	3.37 ± 0.53 (2.59 - 4.21)	3.51 ± 0.70 (2.68 - 4.35)	4.06 ± 0.65 (2.99 - 5.75)	4.30 ± 0.66 (2.59 - 5.25)
Day 45	3.77 ± 1.11 (1.75 - 5.35)	2.59 ± 1.14 (1.21 - 4.15)	2.05 ± 0.49 (1.18 - 2.84)	1.35 ± 0.46 (1.05 - 2.86)	3.44 ± 0.51 (2.45 - 4.50	3.22 ± 0.63 (2.31 - 4.21)	4.07 ± 0.60 (2.73 - 5.35)	3.39 ± 0.85 (1.56 - 4.91)
Day 90	3.81 ± 1.18 (1.32 - 5.18)	1.55 ± 0.87 (0.56 - 2.13)	2.24 ± 0.46 (1.94 - 2.62)	0.92 ± 0.15 (1.03 - 2.86)	3.69 ± 0.65 (1.56 - 4.91)	2.85 ± 0.70 (1.21 - 4.15)	4.16 ± 0.73 (2.89 - 5.41)	2.88 ± 0.90 (1.56 - 3.91)
Day 0 *vs* 45	- 0.06*	1.08***	- 0.13*	0.56***	-0.07***	0.29***	-0.01 ^{ns}	0.91***
Day 0 *vs* 90	- 0.10*	2.12***	- 0.32*	0.99***	-0.32***	0.66***	0.10***	1.42***
Day 45 *vs* 90	- 0.04^{ns}	1.04***	- 0.18^{ns}	0.43***	-0.25***	0.37***	-0.09 ^{ns}	0.51***

SD = Standard deviation ns= not significant (p >0.05) *= Statistically significant (p < 0.05)
= highly significant (p < 0.01) *= very highly significant (p < 0.001)

RA and periodontitis are chronic inflammatory diseases with very similar cytokine profile seen globally [23,13]. The association of periodontitis and RA is drawing attention of researchers recently. A number of studies suggested a higher incidence and severity of periodontal diseases in the subjects with RA[12,27,10,28,29] and have also shown improvement in the severity of RA after periodontal treatment[16,18,31,32]. In contrast, few other studies have failed to prove the association between RA and periodontitis[15,30]. Thus, the association between periodontitis and RA remains questionable. Considering this fact, the present study was carried out involving 48 nos of RA patients to evaluate the association of periodontitis and RA and also to evaluate the effectiveness of phase I periodontal therapy on the severity of RA.

The evaluation of rheumatological condition was carried out based on ACR/EULAR 2010 Classification criteria. DAS28 was used to evaluate the disease severity, as it is more objective in application. Besides, it describes the severity of RA using both clinical and laboratory data, as Erythrocyte sedimentation rate (ESR) is considered to calculate the severity of RA in terms of disease activity.

In the present study, subjects with a history of smoking in the last one year were not included, as smoking is strongly related to RA and periodontal disease [33, 34]. Thus, they may be strong confounders of the association between RA and periodontal disease and may influence the results. Again, subjects affected with any other systemic diseases were not considered in the present study, as those diseases could be a risk factor for periodontal disease[35]. Again, to eliminate the probability of underestimating the true extent of periodontal disease, subjects with less than 20 teeth were not included in the present study.

The periodontal parameters considered were Simplified Oral hygiene index, Gingival index, Probing Pocket Depth and Clinical Attachment Level. Both the periodontal and rheumatologic parameters were recorded on day 0, 45 and 90.

OHI-S was used as it is a sensitive, simple and rapid method for assessing the oral hygiene quantitatively for an individual or a group. The GI was used for estimation of gingival health based on the bleeding on probing. This is considered as an objective sign of gingival inflammation as the inflamed gingiva bleeds on gentle probing because of the ulcerations in the pocket epithelium and the fragility of the underlying vasculature[1]. PPD was considered, as it is an important parameter for evaluating the success of periodontal therapy by comparing the post treatment PPD value to that of prior to the periodontal therapy. In the present study, the subjects were assessed for PPD on day 45 and 90 following therapy. Opinions differ in the literature regarding the proper timing for assessment of the healing response to non-surgical periodontal therapy. Morrison *et al.*, (1980)[36]

suggested a period of 1 month time after therapy is ideal for reassessment. Similar was reported by Lowenguth and Greenstein (1995)[37]. CAL was used as it gives real value of periodontal tissue damage [38].

Pearson's correlation analysis was performed to determine the relationship between RA and periodontitis. A statistically significant positive correlation between all the periodontal parameters and DAS28 was observed. Our observation supports the findings of Ishi *et al*., 2008, Ortiz *et al*., 2009 and Konopka *et al*.[27, 16, 8].

Here, we have applied DAS28 index as Disease Activity Score as it is widely used as an indicator of RA disease. We have observed very highly significant reduction in DAS28 index in the test group after periodontal therapy and reduced by 25.16% & 53.64% on day 45 and 90, respectively. The reduction in the DAS28 index indicates that periodontitis may significantly influence the severity of RA. This supports the findings of Ortiz *et al*., (2009)[16] and Roman-Torres *et al*., (2015)[32], who have suggested that promotion of dental care and oral health knowledge may play a significant role in improvement of the severity of RA patients. The present findings demonstrate a strong association between the periodontal health condition and the disease severity in RA patient.

The periodontal health status in terms of reduction in OHI-S, GI, PPD and gain in CAL was noted in test group after phase I periodontal therapy. This supports the findings of Ishi *et al*., (2008) [27], Erkiyas *et al*., (2012) [18] and Pischon *et al*., (2008) [39].

To ensure the validity as well as to limit the potential biases in the present study, several measures were taken. Periodontal examinations were performed by only one examiner (SZ) to eliminate inter-examiners variability. The subjects of both the control and test groups were selected from the same source, namely the Rheumatology Clinic, Department of Medicine, Gauhati Medical College.

Our observation clearly demonstrates a strong association between the periodontal health condition and severity of RA. Reduction in DAS28 index was seen with the improvement in periodontal health. However, further studies are needed to confirm our findings involving larger sample size with long follow-up.

V. CONCLUSION

There is a positive correlation between the periodontal health and severity of RA, suggesting poorer the periodontal health, greater is the severity of RA. Again, DAS28 index is reduced significantly after periodontal therapy, indicating an improvement in severity of RA. The findings of the present study thus suggest that phase I periodontal therapy may play an important role in the management of RA.

If periodontitis is confirmed as a risk factor for RA, this will open one of the doors to prevent the dreaded debilitating condition. Thus, it suggests a greater integration of medicine and dentistry will likely require that dental surgeons take more responsibility for the management of their patients' systemic health and conversely that physicians assume a more active role in their patients' oral health.

REFERENCES

[1]. Newman MG. Takei HH. Klokkevold PR. Carranza FA. Carranza's Clinical Periodontology. 11th ed. Philadelphia: PA Saunders, 2011.

[2]. Hunter W. Oral sepsis as a cause of disease. Br Med J 1900;2(2065):215-216.

[3]. Thoden van Velzen SK. Abraham-Inpijn L. Moorer WR. Plaque and Systemic disease: a reappraisal of the focal infection concept. J Clin Periodontol 1984;11(4):209-220.

[4]. Jameson JL, Fauci AS. Kasper DL, Longo DL, Braunwald E, Hauser SL, Loscalzo J. Harrison's Principles of Internal Medicine. 20th ed. McGraw-Hill Education, 2008.

[5]. Ellamurugan N. Karthikeyan I. Periodontitis and Rheumatoid arthritis - A Review. IOSR-JDMS 2013; 10(4):102-105.

[6]. Malaviya AM. Cytokine network and its manipulation in rheumatoid arthritis. J Assoc Physicians India 2006;54(suppl):15-18.

[7]. Farquharson D. Butcher JP. Culshaw S. Periodontitis, Porphyromonas, and the

pathogenesis of rheumatoid arthritis. Mucosal Immunol 2012;5(2):112-120.

[8]. Konopka L. Pietrzak A. Brzezinska-Blaszczyk E. Effect of scaling and root planing on interleukin-1β, interleukin-8 and MMP-8 levels in gingival crevicular fluid from chronic periodontitis patients. J Periodont Res 2012;47(6):681-688.

[9]. Ogrendik M. Rheumatoid arthritis is linked to oral bacteria: etiological association. Mod Rheumatol 2009;19(5):453-456.

[10]. dePablo P. Dietrich T. McAlindon TE. Association of periodontal disease and tooth loss with rheumatoid arthritis in the US population. J Rheumatol 2008;35(1):70-76.

[11]. Disale PR.Zope SA.Suragimath G.Varma AS. Pisal A. Prevalence and severity of periodontitis in patients with established rheumatoid arthritis and osteoarthritis. J Family Med Prim Care 2020;9(6):2919-2925.

[12]. Mercado F. Marshall RI. Klestov AC. Bartold PM. Is there a relationship between rheumatoid arthritis and periodontal disease? J Clin Periodontol 2000;27(4):267-272.

[13]. Kaur S.White S.Bartold M. Periodontal Disease as a Risk Factor for Rheumatoid Arthritis: A Systematic Review. JBI LibrSyst Rev 2012;10(42 Suppl):1-12.

[14]. Al-Katma MK. Bissada NF. Bordeaux JM. Sue J. Askari AD. Control of periodontal infection reduces the severity of active rheumatoid arthritis. J Clin Rheumatol 2007;13(3):134-137.

[15]. Pinho MN. Oliveira RDR. Novaes AB Jr. Voltarelli JC. Relationship Between Periodontitis and Rheumatoid Arthritis and the Effect of Non-Surgical Periodontal Treatment. Braz Dent J 2009; 20(5):355-364.

[16]. Ortiz P. Bissada NF. Palomo L. Han YW. Al-Zahrani MS. Panneerselvam A. Askari A.Periodontal Therapy Reduces the Severity of Active Rheumatoid Arthritis in Patients Treated With or Without Tumor Necrosis Factor Inhibitors. J Periodontol 2009;80(4):535-540.

[17]. Lü Z. Li C. Lü J. He W. Gao L. Wu Y. Periodontal therapy for rheumatoid arthritis: a systematic review. 2011;29(4):375-378.

[18]. Erciyas K. Sezer U. Üstün K. Pehlivan Y. Kısacık B. Şenyurt SZ. Tarakçıoğlu M. Onat AM. Effects of periodontal therapy on disease activity and systemic inflammation in rheumatoid arthritis patients. Oral Dis 2013;19(4):394-400.

[19]. Kay J, Upchurch KS. ACR/EULAR 2010 rheumatoid arthritis classification criteria. Rheumatology, 2012;51(6): vi5–vi9,

[20]. Greene JC. Vermillion JR. The Simplified Oral Hygiene Index. J Am Dent Assoc 1964;68:7-13.

[21]. Loe H. Silness, J. Periodontal disease in pregnancy I. Prevalence and severity, Acta Odont Scand 1963;21(6):533-551.

[22]. Clark DC.Quee TC. Bergeron MJ. Chan ECS. Lautar-Lemay C. deGruchy K. Reliability of Attachment Level Measurements Using the Cementoenamel Junction and a Plastic Stent. J Periodontol 1987;58(2):115-118.

[23]. Malaviya AN. Kapoor SK. Singh RR. Kumar A. Pande I. Prevalence of rheumatoid arthritis in the adult Indian population. Rhematol Int 1993;13(4):131-134.

[24]. DeBowes LJ. The effects of dental disease on systemic disease. Vet Clin North Am Small Anim Pract 1998; 28(5):1057- 1062.

[25]. Fowler EB. Breault LG.Cuenin MF. Periodontal disease and its association with systemic disease. Mil Med 2001;166(1): 85 - 89.

[26]. Das K, Das SJ, Sarma J. Effects of Phase I Periodontal Therapy on the Quality of Life in COPD Patients. JMSCR 2017: 5(10): 28724-28732.

[27]. Ishi EP. Bertolo MB. RossaJr C. Kirkwood KL. Onofre MA. Periodontal condition in patients with rheumatoid arthritis. Braz Oral Res 2008;22(1):72-77.

[28]. Ghalaiani P. Esfahanian V. Sarrafan N. Pishva SS. Hojjati H. The Relationship between Rheumatoid Arthritis and Periodontitis. J Isfahan Dent School 2010;6(1):44-48.

[29]. Abdelsalam SK. Hashim NT. Elsalamabi EM. Gismalla BG. Periodontal status of rheumatoid arthritis patients in Khartoum state. BMC Res Notes 2011;4:460.

[30]. Arkema EV. Karlson EW. Costenbader KH. A Prospective Study of Periodontal Disease and Risk of Rheumatoid Arthritis. J Rheumatol 2010;37(9):1800-1804.

[31]. Bıyıkoğlu B. Buduneli N. Aksu K. Nalbantsoy A. Lappin DF. Evrenosoğlu E. Kinane DF. Periodontal therapy in chronic periodontitis lowers gingival crevicular fluid interleukin-1beta and DAS28 in rheumatoid arthritis patients. Rheumatol Int 2013;33(10):2607-2616.

[32]. Roman-Torres CVG. Neto JS. Souza MA. Schwartz-Filho HO. Brandt WC. Diniz REAS. An Evaluation of Non-Surgical Periodontal Therapy in Patients with Rheumatoid Arthritis. Open Dent J 2015;9:150-153.

[33]. Haffajee AD. Socransky SS. Relationship of cigarette smoking to the sub gingival microbiota. J Clin Periodontol 2001; 28(5):377-388.

[34]. Ragghianti MS. Greghi SLA. Lauris JRP. Sant'ana ACP. Passanezi E. Influence of Age, Sex, Plaque and Smoking On Periodontal Conditions In A Population From Bauru, Brazil. J Appl Oral Sci 2004;12(4):273-279.

[35]. Page RC. The pathobiology of periodontal diseases may affect systemic diseases: inversion of a paradigm. Ann Periodontol 1998;3(1):108-120.

[36]. Morrison EC. Ramfjord SP. Hill RW. Short term effects of initial, nonsurgical periodontal treatment (hygienic phase). J Clin Periodontol 1980;7(3):199-211.

[37]. Lowenguth RA. Greenstein G. Clinical and microbiological response to nonsurgical mechanical periodontal therapy. Periodontol 2000 1995;9:14-22.

[38]. Armitage GC. The complete periodontal examination. Periodontol 2000 2004; 34(1):22-33.

[39]. Pischon N. Pischon T. Kröger J. Gülmez E. Kleber BM. Bernimoulin JP. Landau H. Brinkmann PG. Schlattmann P. Zernicke J. Buttgereit F. Detert J. Association among rheumatoid arthritis, oral hygiene, and periodontitis. J Periodontol 2008;79(6):979-986.