Evaluation of Periodontal Health in Rheumatoid Arthritis Patients

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Abstract

Background and aims. There is a hypothesis that people with rheumatoid arthritis (RA) can also be at risk of periodontal disease. This study aimed to assess the periodontal health in patients with and without rheumatoid arthritis.

Materials and methods. Thirty RA patients with RA and 30 healthy individuals without RA were included. Information regarding demographic characteristics and periodontal parameters (probing pocket depth, attachment loss, plaque index, simplified oral hygiene index and modified gingival index) were recorded.

Results. There was no significant difference in periodontal parameters between participants with and without RA.

Conclusion. According to the cross-sectional pattern of the present study, further evaluation is needed to determine the possible role of RA in the periodontal status of patients.

Key words: Rheumatoid arthritis, periodontal health.

Introduction

Periodontitis is an inflammatory disease of tooth-supporting tissues, which is caused by specific microorganisms, mostly anaerobic and gram-negative bacteria and leads to progressive destruction of periodontal ligament and alveolar bone.1

Rheumatoid arthritis (RA) is a chronic autoimmune disease with an unknown cause. Its characteristic feature is long-standing inflammatory synovitis which involves peripheral joints in a symmetric pattern.2 Systemic involvement of other organs such as skin, muscles, eyes, lungs, vessels and neurons is also common among RA patients.2 This disease affects about 1% of the world population with a female-to-male ratio of 3:1. In Iran it is estimated that 60000 individuals suffer from RA.3

Forty percent of RA patients present some extra-articular manifestations.4,5 For example, in these patients, periodontal disease may be observed as destruction of alveolar bone. Gingival hyperplasia is another manifestation of RA.6 Usually the most im-
important sign of RA in orofacial region is temporomandibular joint (TMJ) involvement which is observed in almost 60% of RA patients.4

Many microorganisms involved in the induction of RA in a genetically susceptible patient have some common features with the organisms observed in periodontitis. It is also found that the serum levels of IgG antibodies associated with some periodontitis organisms such as *P. gingivalis* and *P. intermedia* are high in RA patients.7,9

Kaber et al10 reported that in RA patients, gum hemorrhage, probing depth, attachment loss and missed teeth was increasingly higher than control groups. Mercado et al1 in a study found that the prevalence of RA in patients referring for periodontal treatments was 3.95% higher than those referring for other dental treatments and concluded that patients suffering from moderate or severe periodontal disease are in an increased risk of RA.

Some other studies have also found a relationship between periodontal disease and RA.11-15 Rosenstein et al7 proposed that humoral immunity response to oral bacteria is a stimulus for development of RA. Mikael et al16 in their study concluded that periodontal diseases are related to the higher level of TNF-α in RA patients, but Mercado et al11 did not find a difference between RA and healthy individuals regarding periodontal indices.

Considering the existing controversies, in this study we aimed to assess the periodontal health in RA patients in comparison to healthy individuals.

**Materials and Methods**

Thirty RA patients referring to Khatam-al-Anbia Clinic, Yazd, Iran, participated in this study as a case group. Thirty healthy people which were matched regarding age, gender and smoking status were selected as a control group. In each group there were 26 females and 4 males.

Inclusion criteria for the case group consisted of definite diagnosis of RA by a rheumatologist. Exclusion criteria for both groups consisted of a history of other systemic diseases affecting the periodontium, including diabetes mellitus, leukemia, neutrophilic defects, hormonal disorders, Down syndrome, hepatitis B, diseases which needed prophylactic treatment before examination such as infectious endocarditis, pregnancy, patients who consumed drugs such as anti-epileptics, calcium canal blockers, or hormones that increase gingival thickness.

Data was recorded using a questionnaire consisting of two parts. The first part included demographic data, medical history, and drug consumption and the second part included the results of periodontal examinations. Periodontal examinations were performed under the dental unit light, using Williams probe. Probing pocket depth (PPD), attachment loss (AL), plaque index (PI), simplified oral hygiene index (OHI-S) and modified gingival index (MGI)17 were assessed. The OHI-S was calculated by the scores given to the debris and calculus on the tooth surfaces according to the following range: 0: no debris/calculus present; 1: debris/calculus covering not more than 1/3 of the tooth surface; 2: debris/calculus covering not more than 2/3 but more than 1/3 of the tooth surface; 3: debris/calculus covering more than 2/3 of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth.

This study was performed in accordance with the Declaration of Helsinki and subsequent revisions18 and was approved by the Ethics Committee of Shahid Sadoughi University of Medical Sciences. An informed consent was obtained from all the participants.

SPSS (Version 16, SPSS INC) software was used for analysis of data. T-test and Mann-Whitney test were used to compare the two groups. P<0.05 was considered to be statistically significant.

**Limitations**

Difficulties in finding RA patients without any other systemic diseases or overlapping syndromes.

**Results**

In this study 60 participants (30 cases and 30 controls) were assessed. Mean age of all the subjects was 35.00±5.85 years. Mean age of the case group was 35.50±5.93 years and mean age of the control group was 34.50±5.82 years, with no significant differences (P=0.513). Table 1 shows the periodontal status in both groups. There were no significant dif-

<table>
<thead>
<tr>
<th>Periodontal indices</th>
<th>Healthy M±SD</th>
<th>Patient M±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MGI</td>
<td>1.16±0.79</td>
<td>1.3±0.87</td>
<td>0.536</td>
</tr>
<tr>
<td>OHIS</td>
<td>1.18±0.65</td>
<td>1.21±0.67</td>
<td>0.883</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>1.81±0.42</td>
<td>1.92±0.54</td>
<td>0.509</td>
</tr>
<tr>
<td>PI</td>
<td>0.49±0.19</td>
<td>0.51±0.19</td>
<td>0.584</td>
</tr>
</tbody>
</table>

T-test and Mann-Whitney test
ferences in PPD, MGI, OHI-S and PI between the case and control groups (Table 1). It should be mentioned that mean of attachment loss (AL) was zero in most of the patients without normal distribution. There were no significant differences between the mean of AL between the two groups (P>0.05).

In this study, according to the median age of the patients and healthy individuals, both groups were divided into two subgroups of <35 or ≥ 35 years of age. Mean of the periodontal indices in the two groups, in terms of age, are shown in Table 2.

According to the median of PI, the case and control groups were divided into subjects with PI≤0.5 and PI>0.5. Mean of the indices other than plaque-related ones, in terms of PI values, are shown in Table 3.

**Discussion**

Hard and soft tissue damages in rheumatoid arthritis and periodontal disease have many similarities with each other, in terms of pathological processes and mechanisms of inflammation. Therefore, this hypothesis seems to indicate that patients with rheumatoid arthritis are more likely to develop periodontal disease than healthy individuals. Therefore, this study was designed to evaluate the periodontal health of patients with rheumatoid arthritis. Since the oral health status, as a confounding factor, could affect the results of the study, participants in both groups were matched in terms of oral health status. For this purpose, simplified oral hygiene index (OHIS) and plaque index (PI) were used. In addition, smoking and age are also effective in periodontal parameters. Therefore, considering the fact that in this study PI and OHIS indices were not significantly different between the two groups (P>0.05) and all the participants were non-smokers and were matched for age and sex, the evaluation of possible association between periodontal disease and rheumatoid arthritis was more reliable.

There were no significant differences in PPD, MGI and AL between the case and control groups. In a study in 2001, Mercado et al compared the periodontal status of patients with rheumatoid arthritis and the control group and found no differences in plaque index and gingival index, which is consistent with our study.

In Taheri et al study in 2002, patients with rheumatoid arthritis showed significant increases in plaque index, papillary bleeding index and number of missing teeth, compared to the healthy control group. Two latter results might be attributed to a significant difference between the mean plaque indices in both groups, which was not observed in the present study. In the study above, the mean age of patients with RA (45 years) was higher than the mean age of the participants in our study (35 years). The age difference might be another reason for the difference between these two studies because with aging, due to the cumulative nature of periodontal disease, periodontal destruction and AL will also increase, which is independent from the presence of rheumatoid arthritis. On the other hand, it seems that older people, due to the prolonged exposure to RA disease and the effect of inflammatory factors, may show worse periodontal status. Three patients also had secondary Sjögren’s syndrome in Taheri study, which might have affect their periodontal indices.

In a study in 2012, Torkzaban et al reported no significant differences in the PI means between RA and non-RA subjects, consistent with the present study. According to Torkzaban et al study, there was no statistically significant correlation between RA and any of the periodontal indices and much more evidence is needed to support the cause-and-effect relationship between RA and periodontal indices.

### Table 2. Comparison of periodontal indices on the basis of age

<table>
<thead>
<tr>
<th>Indices</th>
<th>Healthy M±SD</th>
<th>Patient M±SD</th>
<th>Healthy M±SD</th>
<th>Patient M±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MGI</td>
<td>1.06±0.57</td>
<td>1.2±0.86</td>
<td>1.28±0.29</td>
<td>1.04±0.91</td>
<td>0.711</td>
</tr>
<tr>
<td>OHIS</td>
<td>0.95±0.62</td>
<td>1.24±0.68</td>
<td>1.46±0.59</td>
<td>1.18±0.61</td>
<td>0.218</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>1.89±0.45</td>
<td>1.78±0.47</td>
<td>1.92±0.40</td>
<td>1.86±0.68</td>
<td>0.520</td>
</tr>
<tr>
<td>PI</td>
<td>0.41±0.19</td>
<td>0.51±0.19</td>
<td>0.56±0.17</td>
<td>0.51±0.19</td>
<td>0.196</td>
</tr>
</tbody>
</table>

T-test and Mann–Whitney test

### Table 3. Comparison of periodontal indices on the basis of plaque index values

<table>
<thead>
<tr>
<th>Indices</th>
<th>Healthy M±SD</th>
<th>Patient M±SD</th>
<th>Healthy M±SD</th>
<th>Patient M±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MGI</td>
<td>0.78±0.42</td>
<td>0.73±0.45</td>
<td>1.5±0.89</td>
<td>1.86±0.83</td>
<td>0.711</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>1.80±0.35</td>
<td>1.89±0.30</td>
<td>2±0.46</td>
<td>1.75±0.70</td>
<td>0.218</td>
</tr>
</tbody>
</table>

T-test and Mann–Whitney test
Bakhtiari et al\textsuperscript{21} in 2009 reported that developing periodontal disease in patients with rheumatoid arthritis is more common than in healthy people and 60\% of RA patients in this study had moderate to severe periodontitis. Mean clinical attachment loss (AL) in patients with RA and healthy controls were 5.4 mm and 0, respectively, which was not consistent with our study.

According to a study by Javed et al\textsuperscript{22} in 2014, there was no significant difference in socioeconomic status, educational status, self-perceived oral symptoms, and periodontal parameters (plaque index, bleeding on probing, probing depth, clinical attachment loss, number of missing teeth, and marginal bone loss) among chronic periodontitis patients with and without RA. Javed et al reported that self-perceived oral health and periodontal parameters are mainly governed by the intensity of chronic periodontitis and the role of RA in this context seems to be rather secondary. The results of this study are consistent, to some extent, with our study.

Rajkarnikar et al\textsuperscript{23} in 2013 compared RA patients’ specific measures for periodontitis, including plaque index, gingival index, number of missing teeth and radiographic alveolar bone loss scores with a control group matched in relation to age and gender with those of rheumatoid arthritis group. The average alveolar bone loss and gingival index was statistically higher in the RA group, although there was a similar median value of missing teeth and plaque index in both groups. Thus, the general concept that the functional upper limb disabilities in patients with RA contributed to poor manual dexterity resulting in poor plaque control was not confirmed in our study and was not validated in this study, either. Significantly higher value of gingival index or bone loss found in RA patients as compared to the controls could be attributed to the increased secretion of pro-inflammatory mediators in the RA group. This difference might be attributed to differences in rheumatoid arthritis severity or the higher reported occurrence of periodontitis among RA patients in Rajkarnikar et al study in comparison to ours.

**Conclusion**

Because of the cross-sectional pattern of this study, it is not possible to determine whether the cause of periodontal disease is rheumatoid arthritis or whether periodontal disease is a risk factor for rheumatoid arthritis, or both diseases have a common pathology or not. However, it is recommended that the findings of other similar studies be taken into account and more attention be paid to oral hygiene, regular examinations and dental treatments in patients with rheumatoid arthritis.

**References**


