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Evaluation of periodontal status of patients with rheumatoid arthritis: A retrospective study

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Abstract

Periodontal diseases may alter some systemic diseases and general health by changing the host response. In addition, some systemic diseases affect periodontal health. This study purposed to assess the periodontal status of individuals with rheumatoid arthritis (RA) receiving different drug treatments. A total of 160 individuals, 80 with RA and 80 systemically healthy individuals, were retrospectively included in the study. Individuals with RA were classified into two groups based on the medications they used, conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs)+glucocorticoids (n=40) and csDMARDs+TNF-α inhibitor (n=40). The participants' plaque index (PI), gingival index (GI), bleeding on probing (BOP), probing depth (PD), and clinical attachment loss (CAL) values were noted. In addition, duration of disease, type of medication, duration of medication use, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), Disease Activity Score (DAS) 28-ESR, DAS 28-CRP, tender joint count (TJC), swollen joint count (SJC), rheumatoid factor, and visual analog scale (VAS) values were recorded. Appropriate statistical tests were performed. Compared with individuals with RA, BOP, GI, PI, and PD values were higher in the systemically healthy individuals ($p<0.05$). When compared according to the drug treatments used, PI, GI, and BOP values of the patients using csDMARDs+TNF-α inhibitor were significantly lower and CAL was significantly higher than the systemically healthy individuals ($p<0.05$). Moreover, a positive correlation was found between disease duration and PD and CAL values in the patients using csDMARDs+TNF-α inhibitor. The worse periodontal parameters in patients with RA using csDMARDs+glucocorticoids compared with those using csDMARDs+TNF-α inhibitor may be due to differences in the mechanisms of action of the drugs. More comprehensive studies are needed to determine the relationship between periodontal status and RA.

Keywords: Disease modifying anti-rheumatic drug, periodontal disease, rheumatoid arthritis, tumor necrosis factor-alpha inhibitor

Introduction

Periodontal disease is a chronic inflammatory disease initiated by bacteria in the dental biofilm on the root surfaces of teeth. Long-term plaque accumulation causes chronic inflammation that can cause the degradation of the periodontal ligament and adjacent alveolar bone. Periodontal disease can be associated with a variety of systemic conditions, which include diabetes, coronary heart disease, myocardial infarction, premature birth, low birth weight, stroke, and rheumatoid arthritis (RA) [1].

RA is a chronic, autoimmune, and degenerative inflammatory disorder that mainly targets the joints. It is about three times more prevalent in females than in males, with a global occurrence rate of 0.5–1%. This long-term condition is marked by inflammation in the synovial lining of the joints, leading to progressive cartilage degradation, joint stiffness, pain, and marginal bone erosion. Additionally, RA is linked to increased mortality and systemic complications [2]. The majority of RA cases arise due to genetic susceptibility combined with environmental influences. While the precise cause remains unclear, studies suggest that bacterial and viral infections, cytokines, inflammatory mediators, and immune cells such as mast, T, and B cells contribute to synovial damage. Various biomarkers, including antibodies to citrullinated protein antigen (ACPA), C-reactive protein (CRP), rheumatoid factor (RF), and erythrocyte sedimentation rate (ESR), are utilized in RA diagnosis [3].

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The general aim of RA therapy is to provide remission of the disease and reduce disease activity. Disease-modifying antirheumatic drugs (DMARDs) are drugs that aim to control rheumatoid inflammation and disease progression effectively and are divided into two groups: synthetic DMARDs (sDMARDs) and biologic DMARDs (bDMARDs). Conventional sDMARDs (csDMARDs) are a subgroup of sDMARDs and are usually the first treatment option for newly diagnosed individuals [3]. However, if the disease continues to progress and poor prognostic factors are present, targeted sDMARDs (tsDMARDs) or bDMARDs may be used [4]. In addition, two or three sDMARDs or combinations of sDMARDs and bDMARDs may be used [3]. 'Tumor necrosis factor-alpha (TNF-α) inhibitors, a subset of bDMARDs, act by inhibiting the binding of TNF-α to its receptors on cells [3]. Glucocorticoids are given to rapidly control inflammation but have limited benefits and are used for the short term while starting or switching between csDMARDs [3,4].

RA and periodontitis share pathogenic similarities, including bone destruction, soft tissue inflammation, and smoking as a common risk factor [5,6]. Research indicates a strong relationship between these conditions, with odds ratios ranging from 1.82 to 20.57 [7,8]. Moreover, evidence suggests that treating one of these diseases can improve clinical parameters in the other [9,10]. However, the precise mechanisms underlying this relationship remain unclear [11]. Both diseases are characterized by local chronic inflammation and elevated levels of proinflammatory cytokines, several of which include interleukin (IL)-1β, IL-6, and TNF-α. Additionally, increased matrix metalloproteinase activity and activation of the receptor activator of nuclear factor kappa B ligand pathway promote osteoclast differentiation, immune cell infiltration—such as macrophages, neutrophils, T and B lymphocytes—and subsequent tissue damage [2].

In a literature review, no studies were found evaluating the periodontal status of individuals with RA receiving different drug treatments. This research aims to assess the periodontal status of individuals with RA on different drug therapies and to compare them with systemically healthy individuals. This study hypothesizes that the periodontal status of individuals treated for RA is worse than that of systemically healthy individuals.

Material and Methods

This study enrolled individuals who visited the Periodontology Clinic at the Faculty of Dentistry, Recep Tayyip Erdoğan University, for routine periodontal examinations and treatment between January 2022 and October 2024. Participants were selected by retrospectively reviewing their anamnesis and digital panoramic radiographs based on the inclusion criteria. Ethical approval was obtained from the Non-Interventional Clinical Research Ethics Committee of Recep Tayyip Erdoğan University (Decision no: 2024/284), and informed consent was secured from all participants.

Power analysis determined that, with a test power of 0.80, a type-1 error (α) of 0.05, and an effect size of 0.5 (mean t-test effect size), a total of 128 samples would be required for a two-group study, with at least 64 participants per group. To enhance study power, 160 individuals were included—80 in each group. The corresponding post-hoc power value was calculated as 88.2%.

The inclusion criteria for the study were determined as being aged 18-65 years during the period when the examination was performed and the information was recorded, having complete systemic and dental examination information and periodontal index information (gingival index (GI) [12], plaque index (PI) [13], bleeding on probing (BOP) [14], clinical attachment loss (CAL), probing depth (PD)) having panoramic radiography with sufficient image quality, and having no conditions such as pregnancy and breastfeeding causing hormonal changes. Care was also taken to exclude patients with diabetes and any inflammatory disease (except RA). Patients with missing clinical and radiographic data necessary for disease diagnosis were excluded from the study.

Individuals who had a systemic disease that could affect periodontal health, smokers, were not aged 18-65 years, who had insufficient systemic and dental examination information, periodontal index information, and who reported receiving periodontal therapy in the last 6 months were excluded from the study.

The RA-related data of the participants (disease duration, type of medication used, duration of medication use, Disease Activity Score (DAS) 28-CRP [15], DAS 28-ESR [15], swollen joint count (SJC), tender joint count (TJC), RF, CRP, ESR, and visual analog scale (VAS)) were noted by querying the patient records. DAS 28 is used to measure RA activity. It is calculated using the TJC, SJC, ESR, and the patient's evaluation of disease activity assessed using a VAS [16].

The individuals were classified into two groups, patients with RA (n=80) and systemically healthy controls (n=80), and the periodontal parameters of the groups were compared. In addition, patients with RA were categorized into two subgroups to assess the influences of different therapy methods on periodontal parameters.

Study groups

Group 1: Patients with RA (n=80)

- Patients with RA using csDMARDs+glucocorticoids (n=40)
- Patients with RA using csDMARDs+TNF-α inhibitor (n=40): (Patients who previously received csDMARDs+glucocorticoids and started using csDMARDs+TNF-α inhibitor because of insufficient response to treatment)

Group 2: A systemically healthy control group (n=80) was formed

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Statistical analysis

Since the data were normally distributed in the study, parametric tests were used. Descriptive statistics for the continuous parameters in the study are stated as standard deviation (SD), mean, percentage (%), and number (n). The independent t-test and one-way analysis of variance (ANOVA) were utilized to comparing continuous measurements between groups. After variance analysis, the Duncan test was performed to determine different groups. The Chi-square test was utilized to identify the associations between categorical variables, and Pearson correlation coefficients were computed to identify the associations between continuous measurements. In the analysis, the SPSS (IBM SPSS for Windows, ver. 26) statistical package program was utilized. The level of statistical significance was taken as $p<0.05$.

Results

There was a statistically important difference in disease duration (5.85±3.99 vs. 9.93±5.53 years, respectively) and drug usage duration (5.48±3.88 vs. 3.47±2.04 years, respectively) between the csDMARDs+glucocorticoids and csDMARDs+TNF-α inhibitor groups ($p<0.05$). No statistically significant difference was found between these groups regarding TJC, SJC, DAS 28-ESR, DAS 28-CRP, CRP, ESR, and VAS parameters ($p>0.05$). RF was more positive in the csDMARDs+TNF-α inhibitor group (64.1%), and was more negative in the csDMARDs+glucocorticoids group (63.4%) (Table 1).

Table 1. Comparison of data from rheumatoid arthritis patients using different medications

	csDMARDs+glucocorticoids	csDMARDs+TNF-α inhibitor	p
Disease duration (year) (mean±SD)	5.85±3.99	9.93±5.53	0.001*
Duration of medication use (year) (mean±SD)	5.48±3.88	3.47±2.04	0.005*
TJC (mean±SD)	1.58±1.38	1.20±1.22	0.201
SJC (mean±SD)	0.98±1.07	0.72±1.20	0.329
DAS 28-CRP (mean±SD)	2.49±0.81	2.21±0.83	0.137
DAS-28 ESR (mean±SD)	2.85±1.03	2.57±1.11	0.255
CRP (mean±SD)	10.44±10.95	14.22±21.43	0.323
ESR (mean±SD)	16.92±12.49	16.75±14.18	0.953
RF (N/%)	14/35.9	25/64.1	0.014**
VAS (mean±SD)	4.73±2.52	3.58±2.91	0.063

*: Significance levels according to Independent sample T-test results, **: Significance level according to chi-square test results, TJC: tender joint count, SJC: swollen joint count, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, DAS: Disease Activity Score, RF: rheumatoid factor, VAS: visual analog scale, csDMARDs, conventional synthetic disease-modifying anti-rheumatic drugs, SD: standard deviation, N: number

A significant difference in age was observed between individuals with rheumatoid arthritis and periodontally healthy individuals (53.80±8.55 vs. 50.06±6.56, respectively) (p=0.002) (Table 2). The csDMARDs+TNF-α inhibitor group (55.00±8.12) had the highest mean age and was significantly different from the healthy control group (50.06±6.56) (p=0.004). In the csDMARDs+glucocorticoids group (52.60±8.91), the mean age was similar to the other two groups. No meaningful difference was detected between the groups regarding sex (p=0.603) (Table 3).

Table 2. Comparison of age and periodontal parameters according to groups

	Rheumatoid arthritis	Control	p
	Mean±SD	Mean±SD	
Age	53.80±8.55	50.06±6.56	0.002*
PI	1.16± 0.43	1.54±0.72	0.001*
GI	1.23±0.40	1.54±0.79	0.003*
BOP	33.25±21.08	50.68±36.97	0.001*
PD	2.41±0.53	2.68±0.97	0.029*
CAL	2.61±0.62	1.40±2.00	0.001*

*: Significance levels according to Independent sample T-test results, SD: standard deviation, PI: plaque index, GI: gingival index, BOP: bleeding on probing, PD: probing depth, CAL: clinical attachment loss

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Table 3. Comparison of clinical findings according to the type of medication used

	csDMARDs+glucocorticoids	csDMARDs+TNF-α inhibitor	Control	p
Age (mean±SD)	52.60±8.91[ab]	55.00±8.12[a]	50.06±6.56[b]	0.004*
PI (mean±SD)	1.25±0.40[b]	1.06±0.45[b]	1.54±0.72[a]	0.001*
GI (mean±SD)	1.32±0.35[ab]	1.14±0.44[b]	1.54±0.79[a]	0.005*
BOP (mean±SD)	36.77±21.50[b]	29.73±20.31[b]	50.68±36.97[a]	0.001*
PD (mean±SD)	2.47±0.46	2.35±0.59	2.68±0.97	0.076
CAL (mean±SD)	2.68±0.55[a]	2.54±0.69[a]	1.40±2.00[b]	0.001*
Sex (m/f) (N)	14/26	13/27	33/47	0.603**

*: Significance levels according to one-way ANOVA; a, b, c: Shows the difference between groups (Tukey post-hoc test); **: Chi-square test, SD: standard deviation, PI: plaque index, GI: gingival index, BOP: bleeding on probing, PD: probing depth, CAL: clinical attachment loss, csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs

A comparison of periodontal parameters across study groups is given in Table 2 and Table 3. When the RA group in comparison with the healthy control group, it was determined that BOP, GI, PI, and PD values were significantly higher in the control group, whereas the CAL value was significantly lower in the control group ($p<0.05$) (Table 2).

Further analysis categorized RA participants based on their medication use and compared them to the control group. PI and BOP values were significantly higher in the control group than in the csDMARDs+glucocorticoids and csDMARDs+TNF-α inhibitor groups ($p=0.001$), with no significant difference between the csDMARDs+glucocorticoids and csDMARDs+TNF-α inhibitor groups. The highest GI score was observed in the control group ($p=0.005$), while the csDMARDs+glucocorticoids group exhibited similar results to the other two groups. The CAL value was significantly lower in the control group than in the other groups ($p=0.001$), with no notable difference between the remaining groups. Regarding PD values, no statistically significant difference was detected among the groups ($p=0.076$) (Table 3).

No correlation was found between periodontal parameters and age and RA disease-related parameters in the csDMARDs+glucocorticoids group ($p>0.05$) (Table 4). In the csDMARDs+TNF-α inhibitor group, a positive correlation was shown between disease duration and PD and CAL values ($p<0.05$) (Table 5).

Table 4. Correlation analysis results in csDMARDs+glucocorticoids group

		Age	PI	GI	BOP	PD	CAL
Disease duration (year)	**r**	0.016	-0.045	0.075	0.023	-0.211	-0.228
	p.	0.924	0.781	0.647	0.888	0.192	0.156
Duration of medication use (year)	**r**	-0.023	-0.069	0.094	0.068	-0.214	-0.221
	p.	0.886	0.674	0.562	0.675	0.184	0.171
TJC	**r**	-0.027	-0.232	-0.161	-0.200	-0.045	-0.103
	p.	0.870	0.149	0.322	0.216	0.784	0.526
SJC	**r**	-0.009	-0.202	-0.162	-0.136	0.082	0.025
	p.	0.955	0.212	0.319	0.402	0.614	0.879
VAS	**r**	0.065	-0.167	-0.145	-0.076	-0.026	-0.076
	p.	0.692	0.303	0.370	0.642	0.873	0.642
DAS 28-CRP	**r**	-0.004	-0.193	-0.129	-0.034	0.065	0.004
	p.	0.982	0.232	0.427	0.835	0.691	0.979
DAS 28-ESR	**r**	0.172	-0.114	-0.177	-0.216	0.050	-0.039
	p.	0.290	0.485	0.274	0.182	0.759	0.814
CRP	**r**	-0.034	-0.152	-0.076	-0.020	-0.057	-0.063
	p.	0.835	0.350	0.642	0.904	0.726	0.699
ESR	**r**	0.204	0.133	-0.047	-0.107	0.038	-0.028
	p.	0.207	0.415	0.774	0.511	0.817	0.865

r: Pearson correlation coefficients, TJC: tender joint count, SJC: swollen joint count, VAS: visual analog scale, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, DAS: Disease Activity Score, csDMARDs: conventional synthetic disease-modifying anti-rheumatic drugs, PI: plaque index, GI: gingival index, BOP: bleeding on probing, PD: probing depth, CAL: clinical attachment loss

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Table 5. Correlation analysis results in csDMARDs+TNF-α inhibitor group

		Age	PI	GI	BOP	PD	CAL
Disease duration (year)	**r**	-0.074	-0.132	-0.178	-0.164	0.420**	0.393*
	p.	0.649	0.417	0.273	0.312	0.007	0.012
Duration of medication use (year)	**r**	-0.074	-0.150	-0.146	-0.204	0.107	0.110
	p.	0.648	0.355	0.369	0.206	0.512	0.498
TJC	**r**	0.132	0.072	-0.016	-0.079	-0.161	-0.028
	p.	0.418	0.657	0.920	0.628	0.320	0.864
SJC	**r**	0.156	0.072	0.007	-0.004	-0.238	-0.118
	p.	0.338	0.658	0.967	0.983	0.139	0.470
VAS	**r**	0.122	0.054	0.055	-0.017	-0.119	-0.046
	p.	0.455	0.743	0.738	0.915	0.463	0.779
DAS 28-CRP	**r**	0.115	0.096	0.110	0.066	-0.177	-0.094
	p.	0.481	0.555	0.498	0.685	0.276	0.565
DAS 28-ESR	**r**	0.149	0.004	0.105	0.113	-0.116	-0.038
	p.	0.359	0.982	0.519	0.488	0.477	0.817
CRP	**r**	0.182	0.078	-0.048	-0.104	-0.194	-0.107
	p.	0.262	0.634	0.769	0.524	0.231	0.513
ESR	**r**	0.140	-0.014	0.080	0.215	-0.082	-0.025
	p.	0.389	0.933	0.625	0.183	0.617	0.881

r: Pearson correlation coefficients, TJC: tender joint count, SJC: swollen joint count, VAS: visual analog scale, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, DAS: Disease Activity Score, csDMARDs, conventional synthetic disease-modifying anti-rheumatic drugs, PI: plaque index, GI: gingival index, BOP: bleeding on probing, PD: probing depth, CAL: clinical attachment loss, *: $p<0.05$

Discussion

To our knowledge, that is the first research to assess the influences of different treatment methods on periodontal status in individuals with RA. According to our findings, CAL was determined to be higher in patients with RA and other periodontal parameters were lower in these patients. When evaluated according to the RA drugs used, PI, GI, and BOP were observed to be significantly lower in patients using csDMARDs+TNF-α inhibitor in comparison to healthy individuals.

Several studies have reported varying findings regarding the association between RA and periodontal parameters. Erciyas et al. [17] examined this relationship and found that among RA patients, 63% had gingivitis, 22% had chronic periodontitis, 4.6% had aggressive periodontitis, and 10.8% were periodontally healthy. Similarly, Yetkin Ay et al. [18] observed significantly higher periodontal clinical parameters in RA patients in comparison with healthy controls.

De Pablo et al. [19] showed that individuals with RA were approximately twice as likely to develop periodontitis and 2.27 times more probably to be edentulous than healthy individuals. Rodríguez-Lozano et al. [8] found that all periodontal parameters were significantly elevated in RA patients compared to the control group, with a notable correlation between RA disease activity and periodontitis severity. Their study revealed that 97.33% of RA patients had periodontitis, compared to 66.24% in the healthy group. Furthermore, severe periodontitis was significantly more prevalent in RA patients (44.92%) than in controls (12.1%) [8]. This situation may be due to the lack of manipulation due to joint disorders in patients with RA. Mayer et al. [16] observed no significant difference in PI between groups using bDMARDs, those not using bDMARDs, and a control group, and reported that BOP, GI, PD, and CAL parameters were lower in those using bDMARDs. In our study, it was found that PI, GI, BOP, and PD values were lower in patients with RA than in healthy controls, but CAL values were higher. In addition, periodontal parameters were higher in individuals using csDMARDs+glucocorticoids than in individuals using csDMARDs+TNF-α inhibitor, although not statistically significant. Similar to our findings, some studies found that PI [20,21], GI [21], BOP [21], and PD [21,22] were significantly lower in patients with RA in comparison to the control group, and CAL [21-24] values were significantly higher in patients with RA. This can be clarified by the fact that individuals with RA pay more attention to oral care and that the use of TNF-α inhibitor drugs may mask the signs of inflammation and affect the inflammatory process due to the mechanism of action.

In our study, the disease duration was longer in patients using csDMARDs+TNF-α inhibitor and the duration of drug use was longer in those using csDMARDs+glucocorticoids. Mayer et al. [16] also found the disease duration to be longer in patients using bDMARDs. This may be because patients with RA are first given non-steroidal anti-inflammatory drugs (NSAIDs), DMARDs, and glucocorticoids, and if these drugs are not sufficient, they

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are switched to TNF-α inhibitor drugs. In the current study, TJC, SJC, DAS 28-CRP, DAS 28-ESR, ESR, and VAS values were higher in patients using csDMARDs+glucocorticoids than in patients using csDMARDs+TNF-α inhibitor, although not statistically significant. Mayer et al. [16] also showed that DAS 28 values were lower in patients using bDMARDs than in those not using bDMARDs, although not significant, similar to our findings. Also, there was no difference in RA positivity between patients using bDMARDs and those not using bDMARDs. In the current study, RA positivity was higher in those using csDMARDs+TNF-α inhibitors.

Karakus [25] reported that PD was positively correlated with CRP and ESR, BOP with RF and CRP, and CAL with CRP and DAS 28 in individuals with RA. In our study, only PD and CAL were positively correlated with disease duration in the csDMARDs+TNF-α inhibitor group. This may be attributed to the fact that RA and periodontitis have similar pathogenesis, periodontal clinical parameters are higher in RA patients and periodontal destruction may increase with increasing disease duration.

Our study has some limitations. First, the small sample size and the fact that mostly patients with dental and periodontal problems were evaluated, because only patients who applied to our faculty for examination were evaluated. Secondly, there are no values such as RF, CRP, ESR in systemically healthy individuals. Another limitation is that the groups were not matched for age and sex. Studies with larger sample sizes and gender and age-matched groups can be planned.

Conclusion

According to our findings, TNF-α inhibitor treatment significantly reduced PI, GI, and BOP values. We can attribute this to the mechanism of action of TNF-α inhibitor treatment, suppressing periodontal inflammation findings, and the fact that these patients pay more attention to oral care. More comprehensive studies with larger sample sizes, similar average ages and sex, and microbiologic and biochemical parameter inclusion are needed.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
Ethical approval was received from the Non-Interventional Clinical Research Ethics Committee of Recep Tayyip Erdoğan University (Decision no: 2024/284). Informed consent was obtained from all participants who were included in the research.

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