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Original Article

Association Between Periodontitis and Ankylosing Spondylitis Considering the Effect of Drugs

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Abstract

Background: Periodontitis impairs teeth structure through inflammatory-related mechanisms and is found to be related to general diseases, including atherosclerosis, diabetes, and rheumatoid arthritis. With a similar pathogenesis, ankylosing spondylitis (AS) may also accompany periodontitis. But studies have reported contradictory and controversial results on the association of this chronic rheumatoid disorder, affecting the spine and sacroiliac joints, with periodontitis. In addition, in most studies, the efficacy of anti-AS drugs on periodontal parameters has not been discussed. Therefore, we designed this research to examine the relationship between periodontitis and AS considering the effect of consuming drugs.

Methods: Fifty-five patients from the rheumatology department were compared and matched with 20 patients (controls) referred to departments other than rheumatology; cases were divided into separate groups based on drug consumption (NSAIDS and/or anti-TNF). AS severity and periodontal indices, including plaque index (PI), gingival index (GI), clinical attachment loss (CAL), periodontal pocket depth (PPD), and gingival bleeding index (GBI) were recorded, and their association was analyzed using SPSS (16) by the Chi-square test, Mann–Whitney U test, and Kruskal–Wallis test. A p-value of less than 0.05 was regarded as statistically significant.

Results: Seventy-five individuals (average age of 39.00 ± 9.74 years) completed the study; PPD, CAL, PI, and GBI were similar between the study groups ($P > 0.05$), but new patients with AS had a larger value of GI than controls ($P = 0.008$). The mean Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) score did not differ significantly across the different levels of periodontitis severity ($P = 0.476$).

Conclusion: The similar frequency of periodontitis and periodontal indices among the study groups rejected a strong association between AS and periodontitis. The one parameter (GI) that was higher in newly diagnosed patients with AS shows the possible effect of drug consumption on the reduction of this index.

Keywords: Ankylosing, Periodontitis, Rheumatology, Spondylitis

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Introduction

Periodontitis is a common oral disease in which the accumulation of dental bacterial plaques results in chronic inflammation that develops into irreversible tissue damage to the supporting oral structures, including bone and connective tissue, ending in tooth loss.[1] Several risk factors have been counted for the development and advancement of periodontitis, particularly among adult populations.[2] Also, several systemic diseases have been found to be associated with periodontitis,[3] the mechanism of which is suggested to involve an acute systemic inflammatory response that impairs endothelial function and increases the risk of atherosclerosis, cardiovascular events, and diabetes[4,5] or genetic factors that make individuals susceptible to both conditions.[6] Evidence has also suggested that periodontal treatment can reduce atherosclerotic biomarkers in patients with cardiovascular disease and improve glycemic status in patients with diabetes.[7,8]

Notably, this association is not limited to endothelial-related diseases; other systemic diseases, especially those with inflammatory mechanisms, are found to be related to periodontitis. Rheumatoid arthritis is more prevalent in patients with moderate to severe periodontitis, attributed to the similar pathogenesis of the two diseases.[9] Ankylosing spondylitis (AS) is a chronic inflammatory rheumatic disorder that primarily involves the spine and sacroiliac joints, and some studies have reported periodontitis more frequently in AS patients, compared with control groups [10]; possibly related to genes causing the cytokine imbalance involved in both diseases.[11] A meta-analysis of five studies reported an odds ratio (OR) of 2.07 for

periodontitis in AS-affected patients.[12] The more frequent periodontitis in patients with AS was also confirmed in the extended meta-analysis of 12 studies.[13] However, the difference in the periodontal indices between the cases and controls has only been suggested by a few studies [14-18] and not confirmed by the meta-analysis studies.[12,13] Also, it is not clear whether anti-AS drugs influence periodontal condition. Considering the contradictory and controversial results on the relationship between AS and periodontitis and the limitations in subgroup analyses, therefore, the present study was designed to explore the relationship between these two conditions by considering drug effects.

Methods

Study design

Patients who were referred to a Medical Education Center in Iran, in 2022 were enrolled. The sample size was calculated at 75 patients, based on Table 2 of the study by Saeed Ali Mohammed and colleagues,[19] a confidence interval of 95%, a study power of 80%, and an effect size of g = 4 using the standard sample size equation. The samples were selected by a convenience sampling method (non-randomized) among patients referred to the center if they were at least 18 years old and had at least 20 teeth (regardless of wisdom teeth and remaining roots). The patients who had any systemic diseases or used any drugs affecting periodontal conditions (such as diabetes), who smoked or abused drugs, who had a history of periodontal disease during the previous 6 months, used antibiotics or corticosteroids over the previous 3 months, pregnant or lactating women, and those who used fixed or removable appliances were not included in the study. Twenty non-AS individuals referred to departments other than rheumatology were selected as controls, and 55 individuals referred to the rheumatology department were selected as the case group; 15 new cases (case group 1, newly diagnosed with AS who were neither using NSAIDs nor anti-TNF medications, and only rarely used opioid drugs or acetaminophen), 20 AS patients under treatment with non-steroidal anti-inflammatory drugs (NSAIDs; case group 2), and 20 patients with AS who were taking anti-tumor necrosis factor (TNF) drugs (case group 3). The groups were matched in terms of age, sex, and body mass index (BMI).

The diagnosis of AS was established by an experienced rheumatologist, according to the Modified New York criteria,[20] supported by laboratory findings such as HLA typing (HLA-B27), erythrocyte sedimentation rate (ESR), and clinical evaluation based on the Assessment of Spondyloarthritis International Society (ASAS) guidelines. To estimate the severity of AS, the Bath Ankylosing Spondylitis Disease Activity (BASDAI) index was considered; this index measures the patient's discomfort, pain or swelling in the spine and joints, fatigue, and severity of morning stiffness by six questions. The total score (0–50) is divided by 5 resulting in a score of 0 (indicating no problem) to 10 (the worst condition); scores ≥ 4 indicate inadequate disease control (these patients are candidates for treatment modification).[21]

To evaluate the oral health and periodontal conditions of the patients, Silness and Leo plaque index (PI) was used, which measures oral health based on the recording of soft debris and mineral deposits on maxillary right first molar, right lateral, left first premolar, mandibular left first molar, left lateral, and right first premolar. This index is calculated as the sum of the scores for the six teeth divided by six. For each tooth, all surfaces, buccal, lingual, mesial, and distal, are scored from 0 to 3. The sum of the scores of the four surfaces is divided by 4 to show the plaque index: 0 = no plaque, 1 = a layer of plaque on free gingival margin and attached to the next teeth (revealer liquid or probe might be required for observing the plaque), 2 = moderate soft deposit collection within the gingival pocket or gingival margin, observable by the naked eye, and 3 = the accumulation of soft materials within the gingival pocket and/or on the teeth or gingival margin.[22]

Leo and Silness gingival index (GI) was used to evaluate the severity and extent of gingival inflammation by calculating the mean gingival bleeding score from four sites: mesial, distal, facial, and lingual. 0 none, 1 mild (without bleeding), 2 moderate with bleeding on probing, and 3 severe with spontaneous bleeding.[23] Ainamo & Bay gingival bleeding index (GBI) was also used to evaluate whether probing results in bleeding at the four surfaces; the percentage of positive sites was also reported, as well.[24] Furthermore, the periodontal pocket depth (PPD) and clinical attachment loss (CAL) were recorded using a Williams probe. The CAL was considered the space between the cementoenamel junction and the apical extent of the probe and between the free gingival margin and the base of the sulcus, both in millimeters at six points (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual, and distolingual) for each tooth. Periodontitis presence and severity were classified into stages as follows: Stage I, early periodontitis with clinical attachment and bone loss confined to the most coronal portion of the root; Stage II, moderate periodontal destruction involving the coronal third of the root with probing depths up to 5 mm; Stage III, severe periodontitis with increased risk of tooth loss, often associated with furcation involvement and infrabony defects; and Stage IV, advanced periodontitis with extensive tissue destruction and potential loss of more than five teeth.[25] One clinician, who was blinded to the patients' medical history (systemic diseases and medications), performed the oral examinations under the supervision of a periodontist.

The Ethics Committee of the University of Medical Sciences endorsed the study protocol. The study objectives and procedures were clearly explained to eligible participants, and written informed consent was obtained from all individuals prior to enrollment.

Statistical analysis

Initially, all recorded data were entered into SPSS software for Windows (version 16.0; SPSS Inc., Chicago, IL, USA)

for statistical analysis. Descriptive data were presented as frequencies, percentages, means, and standard deviations (SD). Based on the Shapiro–Wilk test for normality, we compared the groups using the independent samples t-test, when data were normally distributed, and and non-normally distributed data using the Mann–Whitney U test. For comparisons involving more than two groups, the Kruskal–Wallis test was performed. The Chi-square test was used for categorical variables, and associations between variables were assessed using Spearman's correlation coefficient. The significance level was set at $P<0.05$.

Results

A total of 75 individuals, aged 23-66 years (average of 39.00 ± 9.74 years), completed the study, of whom 39 were men (52%). For comparison between the groups, first, we report the results with three case groups combined (Table 1), which indicated no significant difference in terms of demographics and periodontal measures between the groups (Table 1; $P>0.05$).

In the next step, we evaluated the three case groups separately in order to determine differences among the case groups. The mean age, BMI, and sex distribution of the participants were not different among the four study groups (three cases and one control group). Also, the periodontal indices among the four study groups showed no difference in terms of GI, PPD, CAL, PI, and GBI (Table 2; $P>0.05$). The pairwise comparison of the periodontal values between all groups showed non-significant results; It should be noted that there was no difference between case group 1 (new cases) and the control group in terms of PPD, CAL, PI, and GBI ($P=0.841$, 0.491, 0.176, and 0.377, respectively) but GI differed significantly between the groups ($P=0.008$) with a higher mean value in cases, compared with the control group (0.66 vs. 0.35, respectively; Table 2).

Investigating the severity of periodontitis showed that 28 patients were normal (37.3%), 28 with stage I (37.3%), 19 with stage II (25.3%) of periodontitis. The comparison of the severities among the study groups showed no significant difference ($P=0.888$; Table 2).

The mean BASDAI score was 1.8 ± 0.6 in all patients, 1.3 ± 1.0 in patients without periodontitis, 1.3 ± 1.0 in patients with stage I and 1.56 ± 1.01 in patients with stage II of periodontitis. No difference was detected in the mean BASDAI scores based on the severity of periodontitis ($P=0.476$). The results of the correlation between AS severity and periodontal indices are shown in Table 3; as observed, the BASDAI was not correlated with any of the periodontal indices. However, many of the periodontal indices were correlated with each other ($P<0.05$; Table 3).

Table 1. Comparison of demographic and periodontal indices between the control group and all patients with ankylosing spondylitis (case group)

Parameters	Case (N = 55)	Control(N = 20)	*P* value
Age (mean ± SD)	39.03 ± 9.56	38.90 ± 10.49	0.95
Sex (male/female)	29/26	10/10	0.83*
Body Mass Index (mean ± SD)	23.88 ± 3.33	23.47 ± 3.28	0.86
periodontitis % (n)	74.5 (35)	25.5 (12)	0.77
Periodontal pocket depth (mean ± SD)	1.27 ± 0.03	1.30 ± 0.05	0.44
Clinical attachment loss (mean ± SD)	1.67 ± 0.18	1.45 ± 0.29	0.52
Plaque index (mean ± SD)	1.02 ± 0.07	0.89 ± 0.12	0.29
Gingival index (mean ± SD)	0.55 ± 0.06	0.35 ± 0.07	0.11
Gingival bleeding index (%)	26.87	22.85	0.46
BASDAI (mean ± SD)	1.8 ± 0.6	-	-
Erythrocyte sedimentation rate (mean ± SD)	26.43 ± 17.67	-	-
Duration of AS (mean ± SD)	5.36 ± 8.62	-	-
NSAIDs % (n)	36.35 (20)	-	-
Anti-TNF α % (n)	36.35 (20)	-	-
Newly diagnosed % (n)	27.3 (15)	-	-

*Chi–square test, Abbreviations: AS; Ankylosing spondylitis, BASDAI; Bath Ankylosing Spondylitis Disease Activity Score, NSAIDs; Non-steroidal anti-inflammatory drugs

Discussion

We compared the periodontal indices of AS patients vs. matched controls; to consider the effect of AS treatment, we categorized the cases into newly diagnosed, those receiving NSAIDs, and those receiving TNF inhibitors. The results showed no significant differences in the periodontal indices among the study groups and no association between periodontal indices and BASDAI. The main goal of categorizing patients with AS into different groups was to distinguish between new cases and patients receiving different medications for a longer period, which has not been addressed previously. With the aim of comparing the effect of AS treatment on periodontitis, Enginar and colleagues compared 200 patients with AS and showed that the frequency of periodontitis was similar in patients receiving anti-TNF medications and NSAIDs; considering periodontal indices, PI, PD, GBI, and CAL were similar. They also showed no significant difference in BASDAI scores between patients with and without periodontitis,[26] which is similar to the current study, although they neither considered new cases nor a control group in their study. Sezer and colleagues also showed no difference in the periodontal indices in AS patients vs. the controls, confirmed by the logistic regression analysis, indicating a similar frequency of AS among patients with CAL > 3 and ≤ 3.[18] Other studies have also shown similar periodontal values in AS patients vs. controls,[19,27] which are also consistent with the findings of the present study. Health-related and protective behaviors have also been suggested as important factors influencing periodontal status.[28]

In the present study, periodontitis was present in 60% of the control group and 63.6% of the case group without significant differences. Sezer and colleagues also reported a similar frequency of periodontitis (CAL ≥ 3) in patients and controls,[18] which aligns with our study. Similarly, Kang and colleagues found that the frequency of moderate-to-severe periodontitis did not differ according to AS status

Table 2. Comparison of periodontal indices among the study groups

		Case group 1 (new cases; N=15)	Case group 2 (using NSAIDs, N=20)	Case group 3 (using TNFs, N=20)	Control group (N=20)	*P* value
Gingival index		0.66±0.43	0.43±0.39	0.58±0.54	0.35±0.34	0.108*
Periodontal pocket depth		1.33±0.23	1.28±0.24	1.23±0.21	1.30±0.22	0.374*
Clinical attachment loss		1.73±1.38	1.58±1.16	1.71±1.55	1.45±1.31	0.905*
Plaque index		1.13±0.55	0.88±0.57	1.08±0.50	0.89±0.56	0.342*
Gingival bleeding index		26.46±15.43	27.47±19.69	26.63±18.37	22.85±16.54	0.900*
Severity of periodontitis, N (%)	Normal	5(33.3)	6(31.6)	9(42.9)	8(40.0)	0.888†
	Stage I	5(33.3)	9(47.4)	6(28.6)	8(40.0)	
	Stage II	5(33.3)	4(21.1)	6(28.6)	4(20.0)	

*The results of the Kruskal-Wallis test, †The result of the Chi-square test

Table 3. The correlation between the severity of ankylosing spondylitis and periodontal indices

	BASDAI		GI		PPD		CAL		PI		GBI	
	R	*P*	*R*	*P*	*R*	*P*	*r*	*P*	*r*	*P*	*R*	*P*
BASDAI	1.000	-	-0.016	0.905	0.193	0.158	0.123	0.373	0.199	0.145	0.150	0.274
GI	-0.016	0.905	1.00	-	0.417	0.002	0.346	0.010	0.457	<0.001	0.534	<0.001
PPD	0.193	0.158	0.417	0.002	1.00	-	0.341	0.011	0.292	0.031	0.345	0.010
CAL	0.123	0.373	0.346	0.010	0.341	0.011	1.00	-	0.368	0.006	0.512	<0.001
PI	0.199	0.145	0.457	<0.001	0.292	0.031	0.368	0.006	1.00	-	0.575	<0.001
GBI	0.150	0.274	0.534	<0.001	0.345	0.010	0.512	<0.001	0.575	<0.001	1.00	-

r indicates the Spearman's correlation coefficient

(70.2% vs. 66.6% in controls),[17] which is similar to our results, but the results of studies are controversial in this regard. In one study, periodontitis was even less common in AS patients than in controls (56% vs. 69%, $P \leq 0.01$).[16] Others have suggested that patients with AS had a greater rate of prior history of periodontitis, compared with the control group.[10] In contrast to the findings of the present study, two previously conducted meta-analyses reported a higher prevalence of periodontitis among patients with AS [13] with an odds ratio of 2.07.[12] Also, different studies have reported different rates for periodontitis, varying from 38-88% in AS patients and 26-71% in healthy individuals.[13] This wide range in the frequencies may be related to differences in the factors that influence periodontitis among the participants of different studies, such as oral health, routine dental visits, and dental care. Additionally, periodontitis was found more prevalent in studies involving participants above 40 years, compared with studies in which the mean participant age was below 35 years.[12] An increased prevalence and severity of periodontal diseases have also been reported in other general diseases, compared with healthy individuals.[29] Other factors, like smoking, have also been neglected in some studies, while we have excluded all of them. Furthermore, the definition used for periodontitis differs among studies.

In the present study, GI was higher in new cases, vs. the healthy group, while treatment appeared to attenuate this effect and making GI and other periodontal indices similar to those of the healthy group. This finding suggests the possible role of AS treatment in periodontitis, although other indices were not different. In the study conducted by Pischon and colleagues, the use of AS treatments, including NSAIDs and anti-TNF agents, was similar between patients with AS and periodontitis and those without periodontitis.[14] However, some studies have suggested a positive role for AS treatment on periodontitis, like the study by Fabri and colleagues, which showed that 6-month treatment with anti-TNF medications improved periodontal attachment level assessments in patients with AS (PPD change from 2.18 to 1.94 mm, $P=0.02$).[30] It has also been suggested that patients with AS who underwent treatment for periodontitis have decreased TNF-alpha and erythrocyte sedimentation rate (ESR) levels,[31] which suggests the benefit of common treatments for improvement of both conditions whereas another study evaluating patients with AS at baseline and 1-1.5 months after periodontal treatment showed no improvement in clinical and biochemical parameters.[32] Therefore, additional studies are required to establish the definitive efficacy of treating periodontitis on AS and, vice versa, the effect of treating AS on periodontal status.

The initial idea for the possible link between AS and periodontitis was prompted by animal studies, which showed a significant relationship between HLA-B27 (which participates in AS pathogenesis) and alveolar bone loss [33]; a further study also proposed the possible role of HLA-27 in the association between AS and aggressive periodontitis.[34] Also, the increased inflammatory markers, like C-reactive protein and ESR, highlighted the role of non-MHC genes involved in regulating the cytokine network, including TNF and interleukins, in both diseases.[12] However, some have suggested that the association between periodontitis and AS may be related

to non-inflammatory factors.[10,18] Further studies are needed to investigate to reveal the mechanisms involved in this association.

The present study successfully evaluated AS patients in different subgroups in order to differentiate between newly diagnosed cases, who were neither using NSAIDs nor anti-TNF medications, and previously diagnosed patients, who were also classified based on the type of treatment they received. Furthermore, we compared the findings with a control group to determine the differences between patients with AS and healthy individuals. In addition, we excluded any controllable factors that could influence either condition (periodontitis or AS), to evaluate the isolated effect of the diseases. However, we also faced some limitations, as well. The first limitation was the small number of patients in each subgroup, enrolled using a non-randomized method from a single study center, which limits the generalizability of the results to the whole population. Secondly, we performed a cross-sectional study and could, therefore, not comment on the prognosis or improvement of the conditions over time. Last but not least, there might be some factors influencing the results that were out of the researchers' control, like the oral hygiene habits or other medications used by the patients. Therefore, future studies should be performed, considering the aforementioned limitations.

Conclusion

No significant differences in the frequency of periodontitis or periodontal indices across the study groups do not suggest a correlation between AS and periodontitis. However, the results of studies are controversial, and further studies are required to reach a definite conclusion. A similar controversy exists regarding the effect of AS medications on periodontal conditions. The one parameter (GI) that showed a higher mean in newly diagnosed AS patients suggests the possible impact of drug consumption on reducing this index, which should be confirmed by future cohort studies.

Authors' Contribiution

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Competing Interests

The authors of this manuscript declare that they have no conflicts of interest.

Ethical Approval

This stydy was approved by Ethical committee of Golestan University of Medical Sciences Ethical code (IR.GOUMS.REC.1401.229).

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References

1. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. Nat Rev Dis Primers 2017;3:17038. doi:10.1038/nrdp.2017.38
2. Shahdadi E, Shahravan A, Hatami N, Manchery N, Abdollahi F, Pooladi A, et al. Prevalence of apical periodontitis and quality of root canal among the Iranian adult population: a cross-sectional radiographic study. J Oral Health Oral Epidemiol 2025;14(1):1-8. doi:10.34172/johoe.2403.1628
3. Könönen E, Gursoy M, Gursoy UK. Periodontitis: a multifaceted disease of tooth-supporting tissues. J Clin Med 2019;8(8):1135. doi:10.3390/jcm8081135
4. Damgaard C, Reinholdt J, Enevold C, Fiehn NE, Nielsen CH, Holmstrup P. Immunoglobulin G antibodies against *Porphyromonas gingivalis* or *Aggregatibacter actinomycetemcomitans* in cardiovascular disease and periodontitis. J Oral Microbiol 2017;9(1):1374154. doi:10.1080/20002297.2017.1374154
5. Palm F, Lahdentausta L, Sorsa T, Tervahartiala T, Gokel P, Buggle F, et al. Biomarkers of periodontitis and inflammation in ischemic stroke: a case-control study. Innate Immun 2014;20(5):511-8. doi:10.1177/1753425913501214
6. Arigbede AO, Babatope BO, Bamidele MK. Periodontitis and systemic diseases: a literature review. J Indian Soc Periodontol 2012;16(4):487-91. doi:10.4103/0972-124x.106878
7. Wang X, Han X, Guo X, Luo X, Wang D. The effect of periodontal treatment on hemoglobin a1c levels of diabetic patients: a systematic review and meta-analysis. PLoS One 2014;9(9):e108412. doi:10.1371/journal.pone.0108412
8. D'Aiuto F, Gkranias N, Bhowruth D, Khan T, Orlandi M, Suvan J, et al. Systemic effects of periodontitis treatment in patients with type 2 diabetes: a 12 month, single-centre, investigator-masked, randomised trial. Lancet Diabetes Endocrinol 2018;6(12):954-65. doi:10.1016/s2213-8587(18)30038-x
9. Araújo VM, Melo IM, Lima V. Relationship between periodontitis and rheumatoid arthritis: review of the literature. Mediators Inflamm 2015;2015:259074. doi:10.1155/2015/259074
10. Keller JJ, Kang JH, Lin HC. Association between ankylosing spondylitis and chronic periodontitis: a population-based study. Arthritis Rheum 2013;65(1):167-73. doi:10.1002/art.37746
11. Hu KF, Huang KC, Ho YP, Lin YC, Ho KY, Wu YM, et al. Interleukin-10 (-592 C/A) and interleukin-12B (+16974 A/C) gene polymorphisms and the interleukin-10 ATA haplotype are associated with periodontitis in a Taiwanese population. J Periodontal Res 2009;44(3):378-85. doi:10.1111/j.1600-0765.2008.01116.x
12. Ratz T, Dean LE, Atzeni F, Reeks C, Macfarlane GJ, Macfarlane TV. A possible link between ankylosing spondylitis and periodontitis: a systematic review and meta-analysis. Rheumatology (Oxford) 2015;54(3):500-10. doi:10.1093/rheumatology/keu356
13. Pandey A, Rajak R, Pandey M. Periodontal diseases and its association with disease activity in ankylosing spondylitis/SpA: a systematic review. Eur J Rheumatol 2021;8(3):168-79. doi:10.5152/eurjrheum.2020.20177
14. Pischon N, Pischon T, Gülmez E, Kröger J, Purucker P, Kleber BM, et al. Periodontal disease in patients with ankylosing spondylitis. Ann Rheum Dis 2010;69(1):34-8. doi:10.1136/ard.2008.097212
15. Ziebolz D, Douglas D, Douglas D, Schmickler J, Patschan D, Müller GA, et al. Periodontal condition is associated with

disease duration and motoric disabilities in patients with ankylosing spondylitis: results of a cross-sectional study. Rheumatol Int 2018;38(5):855-63. doi:10.1007/s00296-018-4012-6
16. Bautista-Molano W, van der Heijde D, Landewé R, Lafaurie GI, de Ávila J, Valle-Oñate R, et al. Is there a relationship between spondyloarthritis and periodontitis? A case-control study. RMD Open 2017;3(2):e000547. doi:10.1136/rmdopen-2017-000547
17. Kang EH, Lee JT, Lee HJ, Lee JY, Chang SH, Cho HJ, et al. Chronic periodontitis is associated with spinal dysmobility in patients with ankylosing spondylitis. J Periodontol 2015;86(12):1303-13. doi:10.1902/jop.2015.150202
18. Sezer U, Erciyas K, Pehlivan Y, Ustün K, Tarakçioğlu M, Senyurt SZ, et al. Serum cytokine levels and periodontal parameters in ankylosing spondylitis. J Periodontal Res 2012;47(3):396-401. doi:10.1111/j.1600-0765.2011.01448.x
19. Mohammed SA. Association between ankylosing spondylitis and chronic periodontitis in Duhok: a case-control study. Egypt Dent J 2017;63(2):1581-9. doi:10.21608/edj.2017.74554
20. van der Linden S, Valkenburg HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the New York criteria. Arthritis Rheum 1984;27(4):361-8. doi:10.1002/art.1780270401
21. Garrett S, Jenkinson T, Kennedy LG, Whitelock H, Gaisford P, Calin A. A new approach to defining disease status in ankylosing spondylitis: the Bath Ankylosing Spondylitis Disease Activity Index. J Rheumatol 1994;21(12):2286-91.
22. World Health Organization (WHO). Oral Health Surveys: Basic Methods. WHO; 2013.
23. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967;38(6):Suppl:610-6. doi:10.1902/jop.1967.38.6.610
24. Rebelo MA, de Queiroz AC. Gingival indices: state of art. In: Panagakos FS, Davies RM, eds. Gingival Diseases-Their Aetiology, Prevention and Treatment. London: IntechOpen; 2011. p. 41-54. doi:10.5772/26236
25. Wiebe CB, Putnins EE. The periodontal disease classification system of the American Academy of Periodontology--an update. J Can Dent Assoc 2000;66(11):594-7.
26. Enginar A, Daltaban Ö, Kaçar C, Tuncer T. Relationship between periodontal status and disease activity in patients with ankylosing spondylitis. Reumatologia 2021;59(1):35-40. doi:10.5114/reum.2021.103643
27. Daltaban Ö, Enginar A, Üstün K, Hatipoğlu M, Kaçar C, Tuncer T. Evaluating the relationship between ankylosing spondylitis and periodontal disease: a case-control study. Clin Oral Investig 2023;27(1):411-20. doi:10.1007/s00784-022-04776-4
28. Saraç Atagün Ö, Ceylan Şen S, Ustaoğlu G. Evaluation of periodontitis patients according to the health protective behaviours scale. J Oral Health Oral Epidemiol 2024;13(4):156-61. doi:10.34172/johoe.2402.1618
29. Kaheh A, Norouzifard A, Ahmadzadeh S. Different type of periodontitis and gingivitis in patients with major thalassemia comparing to healthy people. J Oral Health Oral Epidemiol 2015;4(1):24-9.
30. Fabri GM, Pereira RM, Savioli C, Saad CG, de Moraes JC, Siqueira JT, et al. Periodontitis response to anti-TNF therapy in ankylosing spondylitis. J Clin Rheumatol 2015;21(7):341-5. doi:10.1097/rhu.0000000000000300
31. Ortiz P, Bissada NF, Palomo L, Han YW, Al-Zahrani MS, Panneerselvam A, et al. Periodontal therapy reduces the severity of active rheumatoid arthritis in patients treated with or without tumor necrosis factor inhibitors. J Periodontol 2009;80(4):535-40. doi:10.1902/jop.2009.080447
32. Białowąs K, Radwan-Oczko M, Duś-Ilnicka I, Korman L, Świerkot J. Periodontal disease and influence of periodontal treatment on disease activity in patients with rheumatoid arthritis and spondyloarthritis. Rheumatol Int 2020;40(3):455-63. doi:10.1007/s00296-019-04460-z
33. Tatakis DN, Guglielmoni P. HLA-B27 transgenic rats are susceptible to accelerated alveolar bone loss. J Periodontol 2000;71(9):1395-400. doi:10.1902/jop.2000.71.9.1395
34. Agrawal N, Agarwal K, Varshney A, Agrawal N, Dubey A. Is there a common pathogenesis in aggressive periodontitis & ankylosing spondylitis in HLA-B27 patient? Med Hypotheses 2016;90:63-5. doi:10.1016/j.mehy.2016.03.005