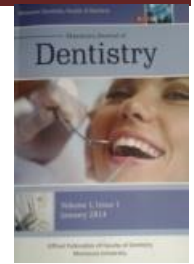




Maxillary molars distalization: a review



Kholod El-Bady Mahmoud El-Ashry, Maher Abd El-Salam Mohammad Fouda, Ahmad Mohammad Hafez

^aTeaching Assistant, Department of Orthodontics.

^bProfessor of Orthodontics Department of Orthodontics Faculty of Dentistry, Mansoura University.

^cAssistant professor of Orthodontics Department of Orthodontics Faculty of Dentistry, Mansoura University.

Abstract:

Objective: The aim of this study was to compare the disease activity of rheumatoid arthritis with chronic periodontitis before and after non-surgical periodontal therapy

Patients and methods: A total of forty patients will be selected and grouped as follow:

Group 1 consists of **20** patients with rheumatoid arthritis and chronic periodontitis (selected from outpatients coming to the Rheumatology and Rehabilitation Department, Faculty of Medicine, Mansoura University). Those patients will be sub grouped as follow:

Subgroup A: 10 patients with rheumatoid arthritis without any other systemic disease

Subgroup B: 10 patients with rheumatoid arthritis in association with other systemic disease

Group 2 consists of **10** patients with rheumatoid arthritis without chronic periodontitis (selected from outpatients coming to the Rheumatology and Rehabilitation Department, Faculty of Medicine, Mansoura University)

Group 3 consists of **10** patients with chronic periodontitis (selected from Periodontology and Oral medicine department, Faculty of Dentistry).

Study groups (group 1 and 3) had undergone full mouth supra gingival and sub gingival scaling and root planning **once** a week for **8** weeks.

Results: In this study, there was a positive correlation between periodontal parameters, total sub gingival bacterial count and DAS 28 ESR in all studied groups at base line. DAS28 ESR used for measuring RA disease activity showed significantly higher levels in RA patients with chronic periodontitis regarding visual analogue scale (VAS) and ESR.

Conclusion: Chronic Periodontitis and Rheumatoid Arthritis share many similarities regarding the clinical, laboratory and microbiological findings. Non- surgical periodontal therapy aids in improvement of the clinical signs and symptoms and a decrease in ESR level in RA patients.

Introduction

Chronic Periodontitis is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, leading to progressive destruction of the attachment apparatus of the teeth including periodontal ligament, cementum and alveolar bone with periodontal pocket formation, and recession of the gingival tissue⁽¹⁾. Severe generalized periodontitis affects 5–15% of any population worldwide and is a major cause of teeth loss after dental caries⁽²⁾.

The understanding of the etiology and pathogenesis of periodontal diseases and their chronic, inflammatory and infectious nature necessitates the possibility that these infections may influence events elsewhere in the body. So, Knowledge about the association between periodontal disease and systemic condition is growing rapidly⁽³⁾.

Meanwhile, a number of other chronic conditions that alters connective tissue metabolism, hormone imbalance and altered immune function have likewise been associated with increased risk of periodontal disease. Most of these associations can be explained by excessive production of inflammatory cytokines and other inflammatory mediators caused by periodontitis⁽⁴⁾.

Although the etiology of both diseases are separate, the underlying pathological processes have some similarity.

Individuals at risk of developing rheumatoid arthritis may also be at risk of developing periodontitis, or vice versa⁽⁵⁾. Both conditions are associated with destruction of bone, mediated by inflammatory cytokines such as prostaglandin E2, interleukin-1, and tumor necrosis factor α ⁽⁶⁾. New researches which focus on inflammation and its initiation by Porphyromonas gingivalis infection has described an immune reaction as a causative helper explaining the risk of patients with periodontitis to develop RA⁽⁷⁾.

Material and method:

This study was carried out on 40 subjects, 30 were selected from the Department of Rheumatology and Rehabilitation, Faculty of Medicine, Mansoura University. They were diagnosed as RA patients after fulfilling the 2010 ACR/ EULAR criteria for RA diagnosis⁽⁸⁾. The age of those subjects ranged from (25-60) years. They were grouped into group 1 A (involving rheumatoid arthritis patients with chronic periodontitis without any other systemic diseases), group 1B (rheumatoid arthritis patients with chronic periodontitis with any other systemic diseases),

group 2 (involving rheumatoid arthritis patients without chronic periodontitis) and 10 subjects were selected from the Department of Oral Medicine and Periodontology, Faculty of Dentistry, Mansoura University. They were diagnosed to have chronic periodontitis after thorough clinical examination without rheumatoid arthritis.

➤ Periodontal assessment:

The following clinical indices were measured at baseline in all groups and eight weeks after conventional non-surgical periodontal treatment in the form of scaling and/or root planning in both of group 1 and group 3; Plaque Index (PI) ⁽⁹⁾, Gingival index (GI) ⁽⁹⁾, Papillary Bleeding Index (PBI) ⁽¹⁰⁾, Periodontal Probing depth Index (PPD) ⁽¹¹⁾ and the Clinical attachment loss index (CAL) ⁽¹²⁾.

➤ Methods:

I. Initial Blood samples collection:

20cm blood will be collected from the antecubital vein of all candidates for evaluation of RF IgM, Anti-Cyclic Citrullinated protein (Anti-CCP), ESR level at baseline.

II. Sub- gingival plaque sample collection:

The subgingival plaque was collected by sterile paper points from the most affected sites by periodontitis at baseline.

III. Rheumatological Assessment of patients in group 1&2

All the thirty patients with RA were diagnosed using the 2010 ACR / EULAR criteria for RA diagnosis ⁽¹³⁾, showed in figure 4. Then the disease activity score for each patient was determined using DAS 28 ESR scoring ^(14,15). The DAS 28 ESR is depending on an external standard of RA disease activity, and involves information from swollen joints, tender joints, the acute phase protein in the form of ESR measure and general health on visual analogue scale into one continuous measure of rheumatoid inflammation ⁽¹⁶⁾.

IV. Treatment phase:

Periodontitis patients had undergone scaling, and root planning by using hand and ultrasonic instruments once a week for eight weeks and the patients were instructed to establish good oral hygiene in the form of regular tooth brushing & dental flossing. No antibiotics were prescribed during the course of treatment. Two months following the treatment, periodontal assessment, DAS 28 ESR for RA patients and total sub-gingival bacterial count were re-assessed.

Results:

Table (1). Comparison of periodontal parameters before and after periodontal treatment in group 1 and 3.

	Treat ment	Group1 (n=20)						Group 3 (n=10)	
		1A+1B (n=20)		1A (n=10)		1B (n=10)		Mean	SD
		Mean	SD	Mean	SD	Mean	SD		
Plaque index(PI)	Before	2.10	0.29	2.14	0.31	2.05	0.29	2.14	0.32
	After	0.86	0.29	0.93	0.35	0.79	0.22	0.84	0.28
	P	*<0.001		*<0.001		*<0.001		*<0.001	
Gingival index(GI)	Before	1.78	0.28	1.78	0.24	1.78	0.33	1.88	0.17
	After	0.68	0.18	0.74	0.22	0.63	0.12	0.84	0.17
	P	*<0.001		*<0.001		*<0.001		*<0.001	
Bleeding on Probing (%)(BOP)	Before	88.01	5.10	89.58	3.70	86.43	5.98	88.89	4.34
	After	17.19	4.01	18.28	3.90	16.09	4.01	18.36	3.71
	P	*<0.001		*<0.001		*<0.001		*<0.001	
Periodontal Pocket Depth (mm)(PPD)	Before	3.70	0.68	3.88	0.41	3.53	0.86	3.88	0.67
	After	2.47	0.51	2.48	0.43	2.46	0.60	3.17	0.59
	P	*<0.001		*<0.001		*<0.001		*<0.001	
Clinical Attachment Level (mm)(CAL)	Before	4.36	0.81	4.63	0.81	4.09	0.75	4.19	0.64
	After	3.05	0.46	3.06	0.43	3.04	0.51	3.37	0.46
	P	*<0.001		*0.001		*<0.001		*<0.001	

Discussion:

Rheumatoid arthritis is an inflammatory disease causing the destruction of extracellular space, mainly that of the joints ⁽¹⁶⁾. Periodontitis is a chronic inflammatory process occurring in the periodontal crevice that is considered a mini joint, in a fluid filled compartment adjacent to the bone ⁽¹⁷⁾. Thus, both disorders are chronic inflammatory processes occurring in a fluid filled tissue adjacent to the bone, which is then gradually eroded. Relation between rheumatoid arthritis (RA) and periodontitis has been considered since a lot of years. Tooth extraction was early treatment was to eradicate any source of oral infection ⁽¹⁸⁾. A lot of studies reported that RA and periodontitis may have similar occurrence and nearly 5% of the affected patients are about 50 years or older ⁽¹⁹⁾. Similarities in the clinical and pathological features have been suggested between periodontitis and RA. It has been reported that patients with RA are more likely to have periodontitis ⁽²⁰⁾, while patients with moderate or severe periodontitis have a higher occurrence of RA than those without periodontitis ⁽²¹⁾. These bidirectional relationships between the two diseases might be related to the common host immune response as well as similar pathology.

This study was carried to discover the relation between chronic periodontitis and rheumatoid arthritis. It was conducted on forty patients with no significant differences between all the studied groups regarding age and gender to avoid bias. In our selected patients, we excluded those with a history of local and /or systemic antibiotic therapy within the last three months before the start of the study due to the likelihood of bacterial resistance expected in the micro flora

of such individual⁽²²⁾. In addition, subjects who have had oral prophylaxis three months prior to the baseline examination were also avoided due to the increased proportion of coccid cells after scaling and root planning.⁽²³⁾ Furthering, smoking is a strong predictor of unsuccessful periodontal therapy and smokers have less favorable clinical response than nonsmokers⁽²⁴⁾. Also, patients with acute oral infection, faulty fixed or removable prosthesis causing mucosal inflammation, pregnancy or lactation also were excluded as may interfere with the study design or the accuracy of the results.

On the other hand, patients with group 2 have significant high plaque index, gingival index and bleeding on probing. This may be related to poor oral hygiene of rheumatoid arthritis patients due to the concept that the functional upper limb disabilities contributed to poor manual dexterity. That finding were in accordance with **Sjostrom L, Laurell L, Hugoson A, Hakansson**⁽²⁵⁾ that showed higher plaque index in RA patients than non-rheumatoid arthritis patients. Contradictory results were found by **Sjostrom et al**⁽²⁶⁾ where periodontal status were similar between RA and the control group. The higher gingival index in RA patients could be attributed to the secretion of pro-inflammatory mediators.

Regarding the sub gingival bacterial count, there was significant difference between group 1 (RA+PO), group 2 (RA only) and group 3 (chronic PO only) ($p=0.033$). This result was in contrary with **Smit et al**⁽²⁷⁾, that found no difference in the presence and proportions of any periodontal pathogens between RA group and NRA control group.

Erythrocyte sedimentation rate (ESR), showed elevated level in all groups. RA and periodontitis are chronic inflammatory diseases, and so are considered to increase the systemic inflammation markers as serum ESR and CRP levels. This finding came in accordance with **Mercado et al**⁽²⁸⁾ reported the increase of ESR in both diseases and there was an association between ESR level and PD severity.

Anti-cyclic citrullinated protein antibodies (ACCPA) titres and rheumatoid factor (RF) showed no significant difference between group 1 (RA with periodontitis) and group 2 (RA only) ($p=0.758, 0.860$ respectively). This results were in accordance with **Btytkoglu et al**⁽²⁹⁾ study. This study showed that RA and periodontitis coexistence does not appear to affect systemic markers of RA or clinical periodontal findings. That may be due to anti-inflammatory treatment of RA+ subjects. On the other hand, **Hitchon CA, et al**⁽³⁰⁾ showed positive correlation between periodontitis severity and systemic markers of RA, thus suggesting that periodontitis may play role in the risk and progression of RA.

On the contrary, **de Smit et al**⁽³¹⁾, reported that patients with RA and severe periodontitis had a significant elevated DAS28 scores. But, visual analogue scale (VAS) here showed significant difference between group 1 and 2 ($p=0.031$). This finding was in accordance with **Karadottir H et al**⁽³²⁾, founding that values of VAS were 1.5 to 2 times higher in patients without periodontal diseases and RA patients under periodontal treatment than RA patients with severe periodontitis.

The basal rheumatoid factor (RF) was positive in 30% of patients in group III that included patients with chronic periodontitis only and had no RA or any other systemic diseases. In addition, there was a significant positive correlation between basal RF and basal plaque (PI) and basal gingival index (GI), ($r=0.713, p=0.021$; $r=0.692, p=0.026$ respectively). This may suggest that periodontitis a risk factor for developing RA in the future. This came in accordance with recent study made by **Mikulic et al**⁽³³⁾ had shown that the presence of antibodies to P gingivalis in patients with chronic periodontitis is associated with the presence of RA-related autoantibodies among individuals at increased risk for disease but who have not yet developed RA.

The basal anti-citrullinated protein antibody (ACPA), showed a significant correlation with basal periodontal pocket depth (PPD) in group 1A (RA+ periodontitis), ($r=0.773, p=0.009$). That may suggest that periodontitis may have relation with several RA disease activity. This came in accordance with **Miriovsky BJ et al**⁽³³⁾, that found an associations of PD with higher concentrations of RF and anti-CCP-2 autoantibodies that perhaps most, given the associations of these measures with poor long-term outcomes in R

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