

Periodontitis and rheumatoid arthritis: three messages from published literature to clinical practice

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Dear Editor,

We read with great interest the article entitled “The relationship between periodontal status and rheumatoid arthritis – systematic review”, recently published in *Reumatologia* [1].

This relationship, suggested for more than 20 years [2], has increasingly been reported in the last years, and two recent meta-analyses confirmed a statistically significant association between rheumatoid arthritis (RA) and periodontitis (PD) compared to healthy controls [3, 4].

Among the bacteria present in the periodontal biofilm, *Porphyromonas gingivalis* can induce the modification of peptidyl-arginine to peptidyl-citrulline, a prerequisite for production of anti-citrullinated peptides antibodies (ACPA). Moreover, *P. gingivalis* can activate different mechanisms leading to systemic inflammation and bone damage [5]. More recently, an independent statistical correlation between anti-*P. gingivalis* antibodies and anti-ACPA second generation (anti-ACPA2) concentrations was found [6]. To date, ACPA have the best specificity as a biomarker for RA diagnosis and prognosis; and it is common knowledge that serum ACPA can be found several years before clinical development of RA.

In clinical practice, three messages can be useful to keep in mind: 1) the consequences of PD for the RA activity indices, 2) the interference in PD from disease-modifying anti-rheumatic drugs (DMARDs), both conventional (cDMARDs) and biologic (bDMARDs), 3) the prognostic value of PD in naïve arthralgia patients.

In relation to the first point, a significant association between severe PD and RA disease activity by means of three indices (DAS28, DAS28–CRP, SDAI) was recently confirmed [7].

First message: PD – especially if severe – should always be treated before evaluating RA disease activity, so as to avoid unnecessary therapeutic modifications.

In relation to the second point, c-DMARDs can have a beneficial clinical effect on PD following its non-surgical treatment [8]. Instead, the impact of the b-DMARDs on PD is not uniform. Indeed, published literature highlighted that gingival inflammation improved with B-cell or interleukin 6 receptors and worsened with TNF blockers, whereas beneficial clinical effects on gingival bone destruction followed therapy with every type of bDMARD [6].

Second message: in a patient with RA, severe PD is not a contraindication for DMARDs. On the contrary, DMARDs can be useful in preventing dental loss. This message should be more and more shared with the dentist.

Finally, with respect to the third point, in a prospective study of 72 consecutive naïve arthralgia patients, patients with PD had higher risk for future methotrexate (MTX) treatment during 2-year follow-ups than patients without PD [9]. This risk has to be confirmed in other, multicenter studies.

Third message: in the preclinical stage of arthritis patients, the presence of PD could be evaluated as a warning for early use of MTX.

In conclusion, when a patient with RA enters our clinic... let’s not forget the mouth and the dentist.

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