

3 **PATIENTS WITH RHEUMATOID ARTHRITIS AND PERIODONTITIS HAVE HIGHER DISEASE ACTIVITY AND A MORE PRONOUNCED ANTIBODY RESPONSE AGAINST *PORPHYROMONAS GINGIVALIS***

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**Background** Co-existence of periodontitis and rheumatoid arthritis (RA) has been reported. These diseases have similarities in aetiology, pathogenesis and risk factors. *Porphyromonas gingivalis* (PG) is a periodontal pathogen that has the ability to citrullinate endogenous proteins, human fibrinogen and  $\alpha$ -enolase, which are auto-antigens in RA. This study aimed to assess prevalence of periodontitis, and occurrence of PG and of the antibody response against PG in RA patients.

**Methods** In 95 consecutive dentate RA patients, periodontal condition was examined using the Dutch Periodontal Screening Index for treatment needs (DPSI). DPSI category

A patients have no periodontitis, category B patients suffer from moderate periodontitis and category C patients have severe periodontitis. Prevalence of periodontitis was compared to a control population consisting of 500 age- and gender matched individuals attending a general dental practice in the same geographic area of the Netherlands. RA disease activity was scored with the disease activity score-28 (DAS28). Serum was investigated for C reactive protein (CRP), immunoglobulin M rheumatoid factor (IgM-RF) and anticitrullinated peptide antibodies (ACPA). IgG- and IgM antibody titres to PG were measured by an inhouse ELISA. Subgingival plaque samples were tested for presence of PG by culture technique. Serum and subgingival plaque measures were compared to a second control group, matched for age, gender, smoking- and periodontal status without RA or other systemic diseases (n=44).

**Results** 27% of the RA patients had severe periodontitis, which is significantly higher than the 11% found in the control population ( $p<0.0001$ ). Within the RA patients group C had higher DAS28-scores ( $p<0.001$ ), higher CRP-levels ( $p<0.05$ ) and were older ( $p<0.01$ ) than group A and B, although RA disease duration was equal. RA patients group C had higher IgG-anti PG titres ( $p<0.05$ ) than group A and B. No differences were seen in IgM-RF, ACPA and IgM-anti PG titres within the groups. Prevalence of PG in subgingival plaque samples was not different in RA patients compared to matched healthy controls (n=44). RA patients with severe periodontitis showed both higher IgM- and IgG- anti PG titres ( $p<0.01$  resp.  $p<0.05$ ) compared to severe periodontitis patients without RA. There was a significant difference between PG culture positive and negative RA patients, but not in controls, concerning IgM- and IgG- anti-PG titres ( $p<0.01$  resp.  $p<0.001$ ).

**Conclusions** Prevalence of severe periodontitis is high in RA patients. These patients have higher disease activity of RA and a more pronounced antibody response against PG. These findings support the view that both PG and periodontitis attribute to RA disease activity.