

2. Pre-disease—What happens before diagnosis?

1 NO EVIDENCE OF ACCELERATED ATHEROGENESIS IN THE PRECLINICAL AND VERY EARLY PHASE OF RHEUMATOID ARTHRITIS

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Background and objectives The increased risk of cardiovascular disease (CVD) in patients with rheumatoid arthritis (RA) has been largely ascribed to the chronic inflammatory state. Whether the CVD risk is restricted to RA patients with long-standing disease and whether RA-associated autoantibodies also associate with accelerated atherogenesis remains to be elucidated.

Methods The authors measured intima media thickness (IMT) in early arthritis patients diagnosed with RA according to the 2010 American College of Rheumatology/European League Against Rheumatism criteria (disease duration <6 months) (n=20), a group of individuals at risk of developing RA (identified by the presence of arthralgia and elevated serum levels of IgM rheumatoid factor and/or anticitrullinated protein antibodies (ACPA) in the absence of clinical evidence of arthritis) (n=50), and healthy controls (n=70) to study if the CVD risk is increased in the earliest phases of RA. To evaluate the relationship between IMT and RA-associated autoantibodies as well as other parameters, uni and multivariate regression analyses were performed.

Results Both in subjects at risk of developing RA and in very early RA patients, mean IMT was comparable to the mean IMT of healthy controls (0.65 (0.18) and 0.62 (0.21) versus 0.66 mm (0.17) p=0.44 and p=0.14, respectively). In the early RA group,

IMT was associated with age, whereas in the individuals at risk of RA IMT was associated with apoB, total cholesterol, low density lipoprotein-cholesterol, body mass index, systolic blood pressure (SBP), ACPA and erythrocyte sedimentation rate levels in univariate analysis. Upon multivariate linear regression analysis only age and SBP remained significantly associated with IMT.

Conclusion The absence of (a trend towards) increased IMT does not support accelerated atherogenesis during the earliest stages of RA. These findings lend further support to active treatment of inflammation and treatment of CVD risk factors according to national guidelines in RA patients from the onset of clinically manifest arthritis in order to decrease morbidity and mortality from CVD in more advanced RA.