Correspondence

Subclinical coronary artery disease in recent-onset of rheumatoid arthritis

To the Editor

Patients with chronic inflammatory diseases such as rheumatoid arthritis (RA) have an increased prevalence of atherosclerotic coronary artery disease and the chronic inflammation accelerates the progression of atherosclerosis. Hannawi et al[1] has shown that even patients with early active RA have increased coronary artery atherosclerotic burden compared to healthy controls matched for age and gender. This is a significant finding in view of the fact that duration of disease in their cohort is less than a year and the patients have active disease with an average DAS-28 score of 5 and CRP of 28 (normal <5). This finding adds evidence to the importance of early control of disease activity and inflammation with synthetic and biological disease-modifying antirheumatic drugs once the diagnosis of RA is made and with the aim of reaching a target of sustained remission or low disease activity in every patient.[2] Such an approach may maximise a better outcome in the majority of patients with RA and may slow down the atherosclerotic process.

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Reply from the Author

Thank you very much for your interest in the results of our study entitled "Recent-onset of rheumatoid arthritis leads to increase in wall thickness of left anterior descending coronary artery. An evidence of subclinical coronary artery disease".[3]

Inflammation is the hallmark of rheumatoid arthritis (RA), and presently, several lines of evidence suggest that atherosclerosis has an important inflammatory component. The concept of inflammatory mediators is actively involved in the development of vascular damage in patients with RA was supported by previous data.[4,5] Hannawi et al showed that anti-inflammatory therapy with Disease Modifying Anti-Rheumatic Drugs (DMARDs) started early after RA diagnosis is able to improve vascular endothelial function which is regarded as an early stage of atherosclerosis[3] and retard the structural vascular changes.[4]

Different mechanisms have been postulated to explain the increased atherosclerosis in patients with RA. One hypothesis is related to the co-integration observed between RA and traditional cardiovascular disease (CVD) risk factors, such as lipid abnormalities, hyperinsulinemia, blood pressure, and so forth.[5] It is unlikely that these mechanisms are the major contributor to our study results, because none of these impending determinants of atherosclerosis was predominant in our patients. Another cause of increased atherosclerosis in RA is the adverse effect of systemic inflammation on the vascular wall. In our patients C-reactive protein (CRP), erythrocyte sedimentation rate, and RA disease activity markers were elevated. At the same time, there was no significant difference in traditional CVD risk factors between patients and controls.

Since elevations in CRP and some autoantibodies like rheumatoid factor (RF) and anti-citrullinated peptide (anti-CCP) predate onset of RA,[6] this suggests that the accelerated atherogenic process which characterizes RA is related to inflammation which precedes the onset of RA symptoms. RF is of high importance in the diagnosis of RA and the prediction of outcome. Our results showed a correlation between left anterior descending coronary artery wall thickness (LAD wall thickness) and the presence of RF. This may be important in atherogenesis, as RF stimulates the vessels locally, causing inflammation and direct injury to the endothelial cells.[7]

The results of the study have implications on the current clinical medical practice. One of the most important clinical implications is that every RA patient should be assessed for CV risk at the time of presentation with joint symptoms or soon after RA diagnosis. And, a strict control of inflammatory activity should be initiated as soon as possible after RA diagnosis is confirmed.

We suggest that our results be confirmed in a larger-scale multi-centre study, and that specific studies be designed to assess the prognostic implications of LAD wall thickness. Furthermore, it would be prudent as well to assess the effect of various medical management on reversal of such cardiovascular insult upon our RA population. In addition, it would be of paramount importance to have whole of life cardiovascular surveillance of this specific group of our patients.

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