Editorial

Rheumatoid Arthritis - At the Cross Roads of Inflammation and Atherosclerosis!

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It is a well known fact that rheumatoid arthritis (RA) is the commonest inflammatory polyarthritis seen in clinical practice. Much less appreciated is the fact that what hurts in RA is the ‘joint’ but what kills is the ‘heart’. Over the past decade it has become clear that cardiovascular disease is the major killer in RA. Commensurate with this is the growing evidence that inflammation is the prime driver for atherosclerosis. The mechanistic model for atherosclerosis has witnessed a paradigm shift from an inert metabolic disease to an active immune-inflammatory disease. In this context inflammatory rheumatic diseases like RA and lupus offer a fertile ground, where, novel and traditional risk factors converge leading to increased risk of premature atherosclerosis. We and others have demonstrated that this could be particularly important in India where metabolic syndrome is widespread.1-7

Cardiovascular disease in RA has some distinctive features that need reiteration: silent coronary artery disease is common, the disease burden is more, mortality rates are higher and the risk begins early, it may even precede the ACR (American College of Rheumatology) criteria based diagnosis of RA.8-10

The paper by Chatterjee et al11 in this issue of the journal has 2 key messages: structured treatment with traditional disease modifying anti rheumatic drugs (DMARDs) is effective in controlling disease activity and this translates into reduction of cardiovascular risk in early RA. Rheumatic diseases were, for long, plagued by lack of quantitative measures of disease activity. For physicians used to numbers, for example, blood sugar in diabetes, valve areas in stenotic cardiac lesions, assessment of disease activity in rheumatic disorders like RA was difficult. Subjective impressions like ‘doing well’ were the norm. With Metrology entering the clinical domain of Rheumatology, this has dramatically changed for the better and subjective impressions have given way to objective assessments using standardised measures. Chatterjee et al have employed a validated instrument ‘disease activity score’-DAS 28 and used a response driven strategy to espouse the principle of ‘measure and treat’ using a combination of hydroxychloroquine, methotrexate, leflunomide and sulfasalazine. They demonstrate that judicious use of traditional DMARDs can help achieve disease control obviating the need for expensive biologic DMARDs in many cases. The message for the practicing clinician is clear: escalate or de-escalate treatment in RA not arbitrarily but according to disease activity.

The second message pertains to reduction of CV risk RA by controlling disease activity. Here, the surrogate markers employed by the authors have been rather basic: serum lipids and carotid intimo-medial thickness (CIMT). The short duration of the study (1 year), the small numbers and the large 34% drop out make it difficult to draw firm conclusions. It would be ideal to follow up inception cohorts of RA patients for long periods to study incident cardiovascular events like angina, myocardial infarction or sudden cardiac deaths. Surrogate markers like CIMT, lipids, endothelial dysfunction or coronary calcium scores are employed for ease in cross-sectional studies but do not substitute for hard clinical end points. Longitudinal studies with prolonged follow up of asymptomatic RA patients with dyslipidemia, increased IMT or impaired endothelial dysfunction are required. The lack of longitudinal studies or disease registries in India is a major shortcoming in the field of bio-medical research in our country.

Notwithstanding their study limitations, Chatterjee et al.11 have drawn attention to the what can perhaps be termed as the most important extra-articular complication in RA– accelerated atherosclerosis. Physicians caring for patients with RA need to look beyond the joints and incorporate CV risk assessment in their routine practice. The ‘heart’ is no longer a theoretical concern but a clinical reality that no physician or rheumatologist can ignore in RA.

References


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