Subclinical atherosclerosis: A hidden threat for patients with ankylosing spondylitis

In the current study entitled "Assessment of subclinical atherosclerotic cardiovascular disease in patients with ankylosing spondylitis" Anatol J Cardiol 2019; 22: 185-91, Hatipsoylu et al. (1), authors have failed to demonstrate the relationship between AS and atherosclerosis progression. Nevertheless, it was a study in which some main traditional risk factors for CVD were controlled. Two noninvasive techniques were used, carotid intima media thickness (cIMT) and pulse wave velocity, which were considered as important indicators for evaluating the severity of atherosclerotic intimal lesions and arterial stiffness; however, these two instruments cannot assess endothelial function. It is possible to compare the vascular function outputs of different instruments (2). However, further research is needed to confirm the relation of endothelial dysfunction and/or arterial stiffness assessed non-invasively with the atherosclerotic process in patients with AS (2-5). In fact, the relationship between atheroscolerosis and AS is not as easy to highlight, as in rheumatoid arthritis (6).

Inconsistent results in this study suggest vascular beneficial effects of tumor necrosis factor (TNF) inhibitors slowing the progression of subclinical atherosclerosis because approximately 70% of patients with AS were using TNF inhibitors (1). Indeed, a reduction of cIMT in patients with rheumatoid arthritis and spondyloarthritis treated by TNF inhibitors compared with the control group has been reported (5, 7-9). The mechanism suggested was an improvement of the endothelial function by lowering the retinol-binding protein 4 level, an agent of oxidative vascular damage (10). A recently published study reported a rapid and sustained reduction of complement activation in patients with spondyloarthritis patients using TNF inhibitors and suggested that the observed decrease in cardiovascular morbidity is partly owing to its beneficial effect on complement (9). Nevertheless, the duration of the use of the TNF inhibitors was not reported in this study, and we can suggest that a multivariate analysis of the study data will confirm this protective effect of TNF therapy (1).

The characteristics of the student population may also explain the results of this study. In fact, the biologic inflammation and disease activity were moderate (Table 1 in reference 1). Indeed, chronic systemic inflammation plays a key role in the development of atherosclerotic progression (11, 12). The high level of inflammatory markers during SA may be considered as a major cause of accelerated progression of atherosclerosis because pro-inflammatory cytokines are known to independently predict CVD events (4, 13). In addition, controls have significantly higher total cholesterol level than patients; consequently, they have higher atherogenic indices than patients, which blurs the difference between the two groups. Nevertheless, these traditional risk factors are only partially implicated in CVD, and their presence does not fully explain the accelerated progression of atherosclerosis in AS (3, 14).

In conclusion, further research is needed to improve the models of prediction of the cardiovascular risk in patients with AS, and longitudinal prospective studies are needed to confirm the effect of anti-TNF therapy on carotid IMT.

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