Correspondence

Comment on: Variables associated subclinical atherosclerosis among rheumatoid arthritis patients of Gulf Cooperative Council countries

To the Editor

I read with interest the study by Hannawi et al[1] on variables associated with subclinical atherosclerosis among patients with rheumatoid arthritis in the Gulf Cooperative Council countries. The findings are interesting but the main limitation of the study is the absence of a control group.

Regarding the correlation of carotid intima-media thickness and eosinophils, there are other possible explanations. Upon activation, eosinophils release inflammatory cytokines such as Interleukin-6 and TNFα contributing to the inflammatory process within the carotid vessels. Eosinophils also secrete leukotrienes and prostaglandins as well as toxic proteins (MBP, eosinophil cationic protein [ECP], eosinophil-derived neurotoxin, and erythropoietin) aggravating the intimal vascular inflammation.[2]

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Reply from the Author

We thank Prof. Jawad for his interest in the results of our study "Variables associated with subclinical atherosclerosis among patients with rheumatoid arthritis in the Gulf Cooperative Council Countries".[1]

The study has no control arm as the main objective of the study was to look at the determinants of the subclinical atherosclerosis in rheumatoid arthritis (RA) patients in the Gulf Cooperation Council Countries (GCC).

There is abundant evidences that RA patients have an increased cardiovascular disease (CVD) compared to their age, gender, and CVD risk factors matched controls. The increase of CVD in RA has been reported using different surrogate markers for atherosclerosis such as carotid intima media thickness (cIMT), endothelial dysfunction and left anterior descending coronary artery intima thickness.[3-5] Therefore, the focus of our study was to explore the possible determinants of the cIMT among RA patients of the GCC region. The study is ongoing and further clarification, including the comparison with a control subjects shall be available soon.

In regards of the eosinophils, many evidences connected eosinophils to CVD, but it is not known which specific aspect of the atherosclerosis pathophysiology they contribute to. But clearly that eosinophils are multifunctional leukocytes, with roles beyond immunity to involve homeostasis, as supported by scientific evidence. The main focus of the article was to discuss all the cIMT determinants in short rather than detailed discussion of the role of the eosinophil in the CVD. Thus, some of the role of the eosinophils in the atherosclerosis, in addition to what had been mentioned by Dr. Jawad and addition to what had been mentioned in our article, are i) eosinophils enhance von Willebrand factor exposure on endothelial cells and augment platelet adhesion, ii) ECP can stimulate fibroblast migration and fibrosis which hypothetically could be of importance for atherosclerosis.[6] Eosinophil cationic protein also interact with several other proteins, such as complement factors and coagulation proteins, and had been shown to shorten coagulation time. And, 3-activated eosinophils have prothrombotic functions.[7] Eosinophils is many-sided leukocytes that contributes to many pathological and physiological processes related to CVD risk. The contribution of eosinophils in CVD risk depend on the activation status and on the location of the eosinophils. Role of the eosinophils in the CVD need to be further explored.

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