Letter

Carotid intima-media thickness and endothelial function: useful surrogate markers for establishing cardiovascular risk in patients with inflammatory rheumatic disease

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See related editorial by Veldhuijzen van Zanten and Kitas, http://arthritis-research.com/content/10/1/102
and related response by Veldhuijzen van Zanten and Kitas, http://arthritis-research.com/content/10/3/404

We read with interest the editorial by Veldhuijzen van Zanten and Kitas [1], in which they consider whether carotid artery intima-media thickness (IMT) - a surrogate marker of atherosclerosis - might be a good predictor of future cardiovascular events in patients with rheumatoid arthritis (RA). They state that it remains an open question, because no long-term studies have documented such an association in patients with RA. We are pleased to provide the readers of this journal with an answer to this question.

We recently reported [2] that carotid artery IMT had good ability to predict development of cardiovascular events over a 5-year period of follow up in 47 patients with RA without clinically evident cardiovascular disease at the time of evaluation by carotid ultrasonography. In our study carotid IMT, categorized in quartiles, was strongly associated with cardiovascular events; specifically, none of the RA patients with carotid IMT less than 0.77 mm suffered cardiovascular events. However, six of the 10 patients with carotid IMT greater than 0.91 mm experienced cardiovascular events. When logistic regression models were performed, carotid IMT at the time of ultrasonographic study had high power to predict development of cardiovascular events over the 5-year period of follow up. Although the area under the receiver operating characteristic curve was 0.86 when using age at the onset of the study, it was greater in models that included carotid IMT. In this regard, the area under the receiver operating characteristic curve was 0.93 for a model that included only carotid IMT. Based on these findings, we propose that ultrasonographic assessment of the carotid artery should be performed in all patients with RA in order to identify the subgroup of patients at high risk for cardiovascular complications.

In the same editorial, Veldhuijzen van Zanten and Kitas [1] emphasize that endothelial function is highly dependent on current levels of inflammation. We agree entirely with the authors on this point; we observed endothelial dysfunction in patients with biopsy-proven giant cell arteritis (GCA) - an inflammatory disease that involves large and middle-sized blood vessels. However, steroid therapy was able to improve endothelial function. This effect was observed when laboratory markers of inflammation returned to normal levels [3]. Whether normalization of endothelial function might lead to 'protection' against development of accelerated atherosclerosis in chronic inflammatory diseases remains to be determined, but this is in intriguing possibility. It could explain why GCA mortality in very distant regions (such as Rochester, Minnesota, USA and Lugo, north west Spain) is comparable to that observed in the general population of the same age [4]. In this regard, we recently reported that the carotid IMT was not increased in biopsy-proven GCA patients who had ended steroid therapy compared with matched control individuals from the same population [5].

GCA = giant cell arteritis; IMT = intima-media thickness; RA = rheumatoid arthritis.

Taking all of these considerations into account, we support the use of surrogate markers to determine the cardiovascular risk of patients with inflammatory rheumatic diseases.

Competing interests

The authors declare that they have no competing interests.

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