

Are the cognitive functions of patients with rheumatoid arthritis associated with disease activity, with carotid atherosclerotic changes or with mood disorders such as depression and anxiety?

Sirs,

I read with interest the article entitled "Cognitive function of patients with rheumatoid arthritis is associated with disease activity but not carotid atherosclerotic changes", recently published by Lee *et al.* (1).

In multivariable regression analysis, the authors found that the total score of the neuropsychological batteries they used (CERAD-K) had a significant negative correlation with disease activity score with 28 joint counts-erythrocyte sedimentation rate (DAS28-ESR). In particular, DAS28-ESR was 4.14 ± 0.99 in RA patients with memory impairment, and 2.60 ± 0.88 in RA patients without it. Instead, they found no association between these scores for cognitive function and carotid atherosclerotic changes, assessed through intima-media thickness (IMT).

As we know, in the DAS-28 a patient global assessment of disease activity (PtGA) or general health (GH) on a visual analogue scale (VAS) is required (2). The correlation between this assessment and the DAS-28 is low (3, 4).

Some mood disorders such as depression and anxiety can influence tender joints and patient global assessment, leading to DAS-28 (5, 6). On the other hand, depression and anxiety can influence cognitive functions, especially in older patients (7). Lastly, RA itself can favour the onset of mood disorders, especially depression and anxiety.

If that is the case, the authors's declaration that in their study "all subjects with any neurological or psychiatric diseases that

could affect cognition . . . were excluded" appeared questionable.

Furthermore, the lack of neuroimaging findings in this article did not allow evaluating if cognitive impairment (not only memory . . .) was primary or secondary. For example, if cognitive impairment is due exclusively to depression or anxiety, magnetic resonance imaging (MRI), positron emission tomography (PET) and single photon emission computed tomography (SPECT) findings are different from those we can find in primary cognitive impairment, not due to another mental disorder (8, 9). As indirectly confirmed in a large population-study, recently published, the role of carotid atherosclerotic changes does not help a differential diagnosis (10). This would explain why in Lee's study, cognitive impairment was not associated with IMT.

To determine whether or not disease activity of RA contributes to cognitive impairment has relevant operational consequences: if it does, the rheumatologist has to try to have a better control of RA activity; if it does not, instead, he has to propose completely different strategies.

In clinical practice, these evaluations must be taken into account to avoid diagnostic and therapeutic mistakes. Therefore, despite what the authors declared, their conclusions seem to be misleading, and should be read with caution.

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Competing interests: none declared.

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