Assessing the role of pentraxin-3 in Takayasu’s arteritis

Comment on:
Plasma pentraxin-3 levels in patients with Takayasu’s arteritis during routine follow-up
Alibaz-Oner F et al.

Sirs,
We read with great interest the article ‘Plasma pentraxin-3 levels in patients with Takayasu’s arteritis during routine follow-up’, by Alibaz-Oner et al. (1). In this cross-sectional study, plasma Pentraxin-3 (PTX3) levels were significantly higher in 94 Takayasu’s arteritis (TAK) patients compared to healthy controls, but no differences were found between active and inactive patients. The authors then suggested that PTX3 is of limited aid in the assessment of TAK disease activity. However, this conclusion deserves some comments.

To date, several studies point at PTX3 as a useful biomarker for distinguishing active from inactive inflammation in patients with TAK and other vasculitides (2-4). Previous studies from our group showed that PTX3 determinations are more accurate than CRP and ESR in differentiating between active or inactive TAK (2). Also, PTX3 levels (but not CRP levels) may allow discriminating between patients with active vascular inflammation and those with quiescent disease at imaging (3).

The assessment of disease activity in TAK remains a challenge, as neither clinical symptoms nor currently available biomarkers can accurately differentiate active from inactive disease. Marked limitations of physical examination for the assessment of disease activity is debatable. Also, the scoring system of ITAS2010 places particular emphasis on cardiovascular and larger vessel involvement, thus having limited applicability to cases characterized by subclinical vascular inflammation or mild clinical symptoms. Finally, while acknowledging the relevance of this attempt for a quantitative clinical scoring system in TAK, ITAS2010 should be validated in more sizeable and differentiated cohorts as well as in larger clinical studies before it is incorporated into the clinical practice.

In our previous studies, we relied on particularly stringent criteria to define active and inactive disease at different time points. These included the repeated detection (or absence) of new arterial lesions on angiography, new onset of carotidopathy or pain over large vessels, new ischemic episodes, bruit or asymmetry in pulses, and fever without evidence of infection. When objective readouts are used to determine disease activity over time, PTX3 levels can accurately discriminate between active and inactive TAK (2). In conclusion, sound experimental evidence indicates that PTX3 levels correlate with disease activity in TAK patients (2-4). We advocate for additional investigations to unequivocally confirm and validate the usefulness of this biomarker in the clinical and research settings.

References