

Reply to:

Assessing the role of pentraxin-3 in Takayasu arteritis

De Luca G. *et al.*

Sirs,

We read with great interest the letter with the comments of De Luca *et al.* on our manuscript investigating pentraxin-3 (PTX-3) levels in Takayasu's arteritis (TAK) (1). We had reported that plasma PTX-3 levels were significantly higher in TAK patients compared to healthy controls, but levels were similar in active vs. inactive patients when they were assessed with various activity assessment tools such as physician's global assessment (PGA), activity defined according to Kerr *et al.* and ITAS2010 (Indian Takayasu Clinical Activity Score-2010).

De Luca *et al.* mentioned in their letter that PTX-3 was observed to be a useful biomarker to determine disease activity in several previous studies. Among those studies, Dagna *et al.* (2) and Ishihara *et al.* (3) found that PTX-3 was discriminative for active disease, but Tombetti *et al.* reported that only CRP was higher in active disease and PTX-3 levels were similar between active and inactive patients (assessed by Kerr *et al.*), similar to our results. As a new observation, Tombetti *et al.* observed significantly higher PTX-3 levels only in a subset of patients showing 'detectable signs of vascular inflammation' by vascular imaging.

De Luca *et al.* suggest that the discrepancy between our results and their report could be explained by different definitions of active disease. In our study, we used PGA and

Kerr *et al.* definitions, used and widely accepted tools in TAK activity assessment. Kerr *et al.*, although its limitations are widely recognised, is used to define active disease in 49% of the studies in the literature until 2011 (5). We also used ITAS2010, a newly developed assessment tool, validated in two different ethnicities as discriminatory for activity during the routine follow-up of TAK patients (6). De Luca *et al.* report that they used a very stringent criteria to define 'activity' in their study, limited to major vascular signs/symptoms or new angiographic features. However, this approach (which is, in fact, quite similar to Kerr *et al.* definition of activity by two criteria) excludes minor clinical/laboratory features which might frequently lead to the clinician's decision to increase the immunosuppressive therapies. In this context, we believe our approach of defining disease activity by three different definitions (quite in correlation with each other. The total agreement between ITAS2010 and Kerr *et al.* was 86.5%), was appropriate to assess a candidate biomarker in routine follow-up of an inflammatory large-vessel vasculitis, which might mimic a wide variety of other inflammatory diseases such as infection.

In conclusion, we agree with De Luca *et al.* that there is a need for further, especially prospective follow-up studies with PTX-3 as a biomarker in TAK, including more patients with vascular progression demonstrated by angiographic methods such as MRI-angiography or 18-FDG-PET. Until then, routine use of PTX-3 (instead of another widely used pentraxin, CRP) for disease assessment can be too immature.

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Competing interests: none declared.

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