Reply to New-onset aortitis manifesting as relapsing giant cell arteritis successfully managed with tocilizumab monotherapy by Guarda *et al.*

Sirs,

We thank Drs Guarda *et al*. for their thoughtful comments and for showing this interesting and illustrative case (1). In fact, relapses of giant cell arteritis (GCA) are well described in clinical practice and occur both in patients taking glucocorticoids alone (2) in those receiving tocilizumab (3) or when therapy has been discontinued.

Since the frequency of relapses following tocilizumab discontinuation after 12 months of therapy is high, clinicians often decide to continue with this biologic but optimise, currently changing the weekly dose of subcutaneous tocilizumab to every two weeks. However, the risk of relapse in patients undergoing optimisation with tocilizumab still persists (4).

We fully agree with the comments raised by Guarda *et al*., as they accurately stated that optimised long-term treatment with tocilizumab does not prevent potential relapses when this biologic is discontinued. Certainly, disease relapses or recurrences when treatment is discontinued most commonly occur within the first 5 years after disease diagnosis, but may be observed later (2). This fact makes periodic follow-up of patients with GCA advisable, particularly when treatment has been discontinued.

The occurrence of relapses in patients undergoing tocilizumab therapy or who received treatment with this biologic agent for at least two years raises our concern about the efficacy of this anti-IL-6 receptor blocker in achieving complete remission of GCA in all patients. In this regard, we have extensively used 18F-FDG PET/CT for the detection and follow-up of patients with large-vessel vasculitis (5). Based on this, we evaluated a series of 30 GCA patients under tocilizumab treatment in whom normalisation of vascular 18F-FDG uptake was assessed by 18F-FDG PET/CT scanning. Overall, a decrease in vascular uptake was observed consistent with clinical improvement. However, complete normalisation of vascular 18F-FDG uptake was only observed in about one third of patients despite clinical remission (6). This fact leads to an intriguing question as to whether the persistent increase in FDG uptake in the vessel wall in patients who are clinically in remission corresponds to the persistence of subclinical vasculitis, vascular remodelling, or a combination of both. For this reason, we strongly recommend close follow-up of GCA patients in whom FDG uptake still persists.

In conclusion, we fully support the comments of Guarda *et al*. (1). The current limitations both in the identification of relapses and in the ideal agent and adequate duration of GCA treatment are still present in our minds.

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