Correspondence on 'No efficacy of anti-IL-23 therapy for axial spondyloarthritis in randomised controlled trials but in post-hoc analyses of psoriatic arthritis-related 'physician-reported spondylitis'?' by Braun and Landewé

We have read with interest the recently published *Viewpoint* regarding a potential difference, or lack thereof, in the pathophysiology and response to treatment between patients with axial spondyloarthritis (axSpA) and those with psoriatic arthritis (PsA) and axial involvement (axPsA), a domain of PsA characterised by inflammation of the axial skeleton.[1] While post hoc analyses of patients with axPsA from the phase III PSUMMIT-1 and PSUMMIT-2[2] and DISCOVER-1 and DISCOVER-2[3] studies were constrained by various aspects of trial design and available assessment tools, the results highlighted the need for clinical trials focusing on axPsA. We believe this important topic merits additional discussion.

We have previously acknowledged the limitations of the PSUMMIT-1 and PSUMMIT-2 axPsA cohort and agree that clinical judgement alone is insufficient to categorise patients as having axPsA in clinical trials.[2] We also have noted that relying only on radiographic sacroiliitis to confirm the presence of axPsA is a potential limitation to the DISCOVER-1 and DISCOVER-2 post hoc analyses.[3] Results of the guselkumab post hoc analyses, however, indicated potential efficacy of interleukin-23 inhibition in patients with axPsA, in contrast to a prior study in patients with ankylosing spondylitis, which also relied on locally read radiographs.[4] Taken together, the aforementioned findings provided a framework on which to build imaging-related inclusion criteria in the STAR study (see further below). As such, the ustekinumab and guselkumab post hoc analyses should be considered part of our evolving understanding of how to accurately define and evaluate patients with axPsA. Accruing genetic, clinical and radiographic evidence of differential pathophysiology, presentation and clinical course between patients with axPsA and patients with axSpA[5–11] has also informed current schools of thought in this regard.

As we continue to refine axPsA diagnostic criteria and treatment paradigms, additional evidence is already being gathered. Specifically, the unmet need for evidence-based criteria to classify patients as having axPsA is being addressed in the AXIS trial (NCT04434885). Undertaken by the Group for Assessment of Psoriasis and Psoriatic Arthritis and the Assessment of Spondyloarthritis International Society, the multinational, multicentre, cross-sectional AXIS study of 400 patients with PsA presenting with peripheral involvement will assimilate epidemiological, clinical, imaging and laboratory findings, with the aim of developing universally accepted classification criteria for axPsA. Similarly, the ongoing STAR study (NCT04929210) of guselkumab versus placebo, in which study participants are required to meet Classification Criteria for Psoriatic Arthritis (CASPAR) and have inflammation of the spine and/or sacroiliac joints detected by centrally-read MRI, will aid in both characterising patients with axPsA and identifying clinical and imaging outcomes well suited for evaluating changes in their axial symptoms and inflammation over time. Although the Bath Ankylosing Spondylitis Disease Activity Index and the Ankylosing Spondylitis Disease Activity Score currently provide the only means of assessing changes in axial-related symptoms in patients with PsA, in the absence of specific assessment tools validated for axPsA, we also know that improvements in extra-axial symptoms, such as peripheral arthritis, enthesitis, general pain and fatigue, can contribute to improvements in both of these scores.

We are now poised to generate data required to develop unified criteria and universally accepted assessments tools for axPsA. It is our hope that these continued advancements will lead to improved care of patients suffering from PsA.

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