Response to: 'Irritable bowel syndrome symptoms in axial spondyloarthritis more common than among healthy controls: is it an overlooked comorbidity?' by Wallmann *et al*

We read with interest the letter by Wallman et al, that reported the prevalence of irritable bowel syndrome (IBS) in patients with axial spondyloarthritis (axial SpA). The authors present data from the population based SPondylARtrit TvÄrsnitts-Kohort Universitetssjukhuset i Skåne (SPARTAKUS) study,² showing that symptoms meeting IBS criteria were significantly more frequent among patients with axial SpA without known inflammatory bowel diseases (IBD) (30%), than in controls (16%; OR: 2.5 (95% CI 1.1 to 5.7)). Authors conclude that IBS may be an overlooked frequent comorbidity of axial SpA warranting further research and increased awareness. In our view, this suggested high prevalence of IBS in patients with axial SpA might be overestimated for multiple reasons. Even though Wallman et al discussed the limitations of their approach, we would weight them differently resulting in a different appraisal.

First, the clinically evident IBD is present in 6%-14% of axial SpA patients, undiagnosed in a significant part of them.³ Microscopic gut inflammation—which does not have to be associated with a pronounced clinical picture of IBD-is even more common, reaching around 60% in axial SpA patients.³ As IBD was solely retrospectively excluded based on the clinical history in the study population of patients with axial SpA, with no endoscopy performed, symptoms meeting IBS criteria could also be explained by the presence of an undiagnosed IBD in this group of patients. Authors speculate that it is not likely based on no differences in IBS symptoms between patients with axial SpA with elevated versus normal C-reactive protein (CRP) and faecal calprotectin levels. However, both CRP and faecal calprotectin levels may be elevated in patients with axial SpA even without IBD. At the same time, more than 40% of the axial SpA patients included in the SPARTAKUS study were treated with tumour necrosis factor inhibitors, that could influence both CRP and faecal calprotectin levels.

Second, the authors report that abdominal symptoms were observed almost twice more frequently in patients receiving non-steroid anti-inflammatory drugs (NSAID). Furthermore, NSAID related microscopic colitis or known side effects similar to IBD and IBS symptoms were not excluded as well.

Third and most importantly, authors defined IBS as gut symptoms meeting ROME III criteria for IBS.⁵ According to these criteria, IBS should be a diagnosis of exclusion and may be considered when other potential causes for bowel symptoms were actively excluded. Given that, using these criteria in such a retrospective analysis—where exclusion of other causes of bowel

symptoms that may be attributable to IBD is not fully possible—can lead to false conclusions.

Therefore, we think that the prevalence of IBS presented by the authors may be overestimated—given the fact, that in the described setting other explanations of bowel symptoms are more likely—and should therefore be handled with caution. Making the diagnosis of IBS in a clinical context of axial SpA without performing the full diagnostic work-up and having full information (eg, endoscopy) one could miss IBD as a manifestation of axial SpA or side effects of NSAIDs treatment that in both cases would have relevance for the proper management strategy.

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