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Axial disease in psoriatic arthritis

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Axial disease in Psoriatic arthritis

Pure axial involvement is seen in about 5% of cases of psoriatic arthritis (PsA) but axial involvement (defined by symptoms and/or radiography), along with peripheral involvement, can be seen in over 50% of cases (1). Looking at the disease from the starting point of axial spondyloarthritis (axSpA), there is an ongoing debate about the influence of psoriasis on inflammatory axial disease, and, as such, the taxonomy of axSpA. Many believe that axial involvement in psoriatic arthritis can have two phenotypes: a classical ankylosing spondylitis (AS) phenotype and a different expression, with less symptoms, less symmetry, less involvement of the sacroiliac joints, relatively more involvement of the cervical spine, morphologically different syndesmophytes and less HLA-B27 positivity (2). This is important for several reasons but most importantly for classification purposes as existing criteria for axSpA may not encompass this diversity of expression. There is an urgent need for an evidence-based and widely accepted definition axial involvement in PsA that would allow us to define a homogeneous group of patients in which appropriate epidemiologic, clinical and interventional studies can be performed.

In this issue of the journal Feld and colleagues compare patients with axial involvement who already have a diagnosis of psoriatic arthritis with patients who have a diagnosis of ankylosing spondylitis, and who may or may not also have psoriasis *(include reference to article here)*. They found clear differences between the cohorts both clinically, on imaging, and genetically. However, it is important to be clear about the methodology. This was data collected prospectively but not with this study aim in mind – the data analysis was retrospective. The criteria for including patients in each cohort differed: In Toronto patients with any psoriasis, irrespective of their musculoskeletal symptoms, are likely to be referred to the PsA clinic, rather than the AS clinic, which may have biased the cohorts. Further, inclusion in the AS cohort required the patients to fulfil the modified New York criteria (that is to have back pain and radiographic sacroiliitis) which was not a requirement for determining axial involvement in the PsA clinic, the latter being based purely on radiographic sacroiliitis. These ‘limitations’ are discussed appropriately by the authors. It may, however, have influenced the comparison of the two cohorts. The main conclusion

from this study was that AS patients, irrespective of the presence of psoriasis, were more severe clinically and radiographically and had a higher proportion of HLA-B27 positivity. The authors conclude, and agree with other studies, that axial involvement in PsA is different to that seen in AS. This study adds weight to a recent review by the same group where axial involvement in AS and PsA was compared clinically, genetically, with imaging, and by response to treatment (3).

There is some tautology here and it stems from the definitions used and the inclusion criteria for each cohort. In addition, a third PsA cohort has not been discussed, or included – those patients with syndesmophytes without sacroiliitis, potentially up to a third of all patients with axial involvement in PsA (4). Such patients have contributed to comparative cohorts in the past, may be relatively less symptomatic, less severe and contribute to the recorded morphological differences in radiographic expression between AS and PsA.

Where does this leave us? There is increasing evidence of the dichotomy in phenotype of inflammatory axial disease in PsA (figure). There is now emerging genetic evidence to underpin this with classical symmetric sacroiliitis being associated with HLA-B27 and the asymmetric, less severe sacroiliitis being associated with HLA-B08 (5). The practical importance of recognising this is threefold. Firstly, axial disease cannot be ruled out in PsA without sacroiliac and spinal radiographs, irrespective of symptoms. As yet, there are no data regarding the difference in expression of axial involvement using MRI but it will be fascinating to see just such a comparative study using this alternative imaging. Secondly, more information is needed on the alternative, psoriatic, phenotype – genetics, natural history, assessment, impact and response to treatment. Could IL-23 inhibition work for this alternative phenotype, as hinted by the recent sub-analysis of the data with ustekinumab (6)? Thirdly, and perhaps most importantly for epidemiologic and interventional studies, are the current classification criteria for axSpA appropriate for this alternative phenotype? The lower HLA-B27 positivity in psoriatic spondyloarthritis means that patients are less likely to achieve the ‘clinical’ arm of the ASAS criteria and, with less sacroiliitis, less likely to achieve the imaging arm of these criteria (7).

Recognising the clinical, radiographic and genetic differences of the 'psoriatic' phenotype has prompted a joint effort by ASAS and GRAPPA to develop a new definition of axial spondyloarthritis in the presence of PsA (8). This collaborative effort has already had an expert consensus, an online discreet choice experiment (in which physicians overwhelmingly opted for mandatory positive axial imaging) and is now proposing a prospective study to develop further appropriate classification criteria for psoriatic spondyloarthritis. The results are eagerly awaited.

Figure caption

Differences between the classical ankylosing spondylitis phenotype, and the alternative (psoriatic) phenotype. The area in red indicates the main findings from the study by Feld et al, in the current issue of Rheumatology.

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