

Psoriatic arthritis

Frequency and characteristics of axial involvement in psoriatic arthritis: results from the International Multicentre AXIS Study

Murat Torgutalp^{1,2,3,*}, Raquel Almodovar⁴, Valderilio F Azevedo⁵, Xenofon Baraliakos⁶, Filip Van den Bosch^{7,8}, Jürgen Braun^{6,9}, Vinod Chandran^{10,11,12,13}, Laura C Coates¹⁴, Atul Deodhar¹⁵, Torsten Diekhoff¹⁶, Filippo Fagni¹⁷, Alberto Floris¹⁸, Floris A van Gaalen¹⁹, Rodrigo Garcia-Salinas²⁰, Lianne S Gensler²¹, Niti Goel²², Alice B Gottlieb²³, Désirée van der Heijde¹⁹, Philip S Helliwell²⁴, Kay Geert A Hermann²⁵, Umut Kalyoncu²⁶, Uta Kiltz⁶, Francis Kynaston-Pearson²⁷, Robert GW Lambert²⁸, Ramasharan Laxminarayan²⁹, Ying Ying Leung^{30,31}, Maria Llop³², Clementina López-Medina³³, Alejandra López-Rodríguez³⁴, Michele M Luchetti Gentiloni³⁵, Miranda van Lunteren¹⁹, Marina Magrey³⁶, Ajesh B Maharaj³⁷, Hernán Maldonado-Ficco³⁸, Walter P Maksymowych³⁹, Helena Marzo-Ortega⁴⁰, Marco Massarotti⁴¹, Ashish J Mathew⁴², Philip Mease⁴³, Peter Nash⁴⁴, Victoria Navarro-Compán⁴⁵, Mikkel Østergaard^{46,47}, Fabian Proft¹, Mikhail Protopopov¹, Roberto Ranza⁴⁸, Sherry Rohekar⁴⁹, Carlo Salvarani⁵⁰, Ruxandra E Schiotis⁵¹, Nicholas Shenker^{52,53}, Joachim Sieper¹, Dilek Solmaz⁵⁴, Enrique R Soriano⁵⁵, Lai-Shan Tam⁵⁶, Ricardo Acayaba de Toledo⁵⁷, James CC Wei^{58,59}, Nelly Ziade⁶⁰, Dafna D Gladman^{10,11,13}, Denis Poddubnyy^{1,10,11,13}

¹ Department of Gastroenterology, Infectious Diseases and Rheumatology (including Nutrition Medicine), Charité – Universitätsmedizin Berlin, Berlin, Germany

² Department of Rheumatology and Clinical Immunology, Charité - Universitätsmedizin Berlin, Berlin, Germany

³ Fraunhofer Institute for Translational Medicine and Pharmacology ITMP, Immunology and Allergology IA, Berlin, Germany

⁴ Rheumatology Unit, Fundación Alcorcón University Hospital, Alcorcón, Madrid, Spain

⁵ Department of Internal Medicine, Federal University of Paraná, Curitiba, Paraná, Brazil

⁶ Ruhr-Universität Bochum, Rheumazentrum Ruhrgebiet, Herne, Bochum, Germany

⁷ VIB-UGent Center for Inflammation Research, Department of Internal Medicine and Pediatrics, Ghent University, Ghent, Belgium

⁸ Department of Rheumatology, University Hospital Ghent, Ghent, Belgium

⁹ Rheumatologisches Versorgungszentrum Steglitz, Berlin, Germany

¹⁰ Gladman Krembil Psoriatic Arthritis Program, Schroeder Arthritis Institute, Krembil Research Institute, University Health Network, Toronto, ON, Canada

¹¹ Division of Rheumatology, Department of Medicine, University of Toronto, Toronto, ON, Canada

*Correspondence to Dr Murat Torgutalp, Department of Gastroenterology, Infectious Diseases and Rheumatology (including Nutrition Medicine), Campus Benjamin Franklin, Charité – Universitätsmedizin Berlin, Berlin, Germany.

E-mail address: murat.torgutalp@charite.de (M. Torgutalp).

Dafna D Gladman and Denis Poddubnyy share senior authorship.

<https://doi.org/10.1016/j.ard.2026.02.025>

0003-4967/© 2026 The Author(s). Published by Elsevier B.V. on behalf of European Alliance of Associations for Rheumatology (EULAR). This is an open access article under the CC BY license (<http://creativecommons.org/licenses/by/4.0/>)

- ¹² Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, ON, Canada
- ¹³ Institute of Medical Science, University of Toronto, Toronto, ON, Canada
- ¹⁴ Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences (NDORMS), University of Oxford, Oxford, UK
- ¹⁵ Division of Arthritis & Rheumatic Diseases (OP09), Oregon Health & Science University, Portland, OR, USA
- ¹⁶ Department of Radiology, Immanuel Clinic Ruedersdorf, University Hospital of Brandenburg Medical School, Ruedersdorf bei Berlin, Brandenburg, Germany
- ¹⁷ Department of Internal Medicine 3 – Rheumatology and Immunology, Friedrich-Alexander-University Erlangen-Nürnberg (FAU) and Universitätsklinikum Erlangen, Erlangen, Bavaria, Germany
- ¹⁸ Rheumatology Unit, AOU and University of Cagliari, Monserrato, Italy
- ¹⁹ Department of Rheumatology, Leiden University Medical Center, Leiden, the Netherlands
- ²⁰ Rheumatology Unit Hospital Italiano de La Plata– Universidad Nacional de La Plata, La Plata, Argentina
- ²¹ Department of Medicine/Rheumatology, University of California, San Francisco, CA, USA
- ²² Division of Rheumatology, Department of Medicine, Duke University School of Medicine, Durham, NC, USA
- ²³ Department of Dermatology, Icahn School of Medicine at Mt Sinai, New York, NY, USA
- ²⁴ Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK
- ²⁵ Department of Radiology, Charité – Universitätsmedizin Berlin, Berlin, Germany
- ²⁶ Division of Rheumatology, Department of Internal Medicine, Faculty of Medicine, Hacettepe University, Ankara, Türkiye
- ²⁷ University Hospitals of Derby and Burton NHS Trust, Derby, UK
- ²⁸ Department of Radiology and Diagnostic Imaging, University of Alberta, Edmonton, AB, Canada
- ²⁹ Department of Rheumatology, UHDB, Queen's Hospital, Burton on Trent, UK
- ³⁰ Department of Rheumatology & Immunology, Singapore General Hospital, Singapore
- ³¹ Duke-NUS Medical School, Singapore
- ³² Department of Rheumatology, Parc Taulí Hospital Universitari, Institut d'Investigació i Innovació Parc Taulí (I3PT-CERCA), Universitat Autònoma de Barcelona, Sabadell, Spain
- ³³ Department of Rheumatology, Reina Sofia University Hospital, Maimonides Biomedical Research Institute of Cordoba (IMIBIC), University of Cordoba, Cordoba, Spain
- ³⁴ Department of Rheumatology, Hospital San Javier, Centro de Investigación en enfermedades autoinmunes (CINEA), Guadalajara, Jalisco, México
- ³⁵ Medical Clinic, Department of Clinical and Molecular Sciences, Marche Polytechnic University & Department of Internal Medicine, Azienda Ospedaliero-Universitaria delle Marche, Italy
- ³⁶ Case Western Reserve University School of Medicine, University Hospitals, Cleveland, OH, USA
- ³⁷ Department of Internal Medicine and Therapeutics, Faculty of Health Sciences, Walter Sisulu University, Mthatha, Eastern Cape, South Africa
- ³⁸ Rheumatology Section, Hospital San Antonio de Padua and Clinica Regional Del Sud, Rio Cuarto, Cordoba, Argentina
- ³⁹ Department of Medicine, University of Alberta, Edmonton, AB, Canada
- ⁴⁰ NIHR Leeds Biomedical Research Centre, The Leeds Teaching Hospitals NHS Trust and Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK
- ⁴¹ Department of Rheumatology, University Hospitals Dorset NHS Foundation Trust, Christchurch Hospital, Christchurch, UK
- ⁴² Department of Clinical Immunology and Rheumatology, Christian Medical College, Vellore, Tamil Nadu, India
- ⁴³ Department of Rheumatology Research, Providence Swedish Medical Center and University of Washington, Seattle, WA, USA
- ⁴⁴ School of Medicine, Griffith University, Brisbane, QLD, Australia
- ⁴⁵ Department of Rheumatology, University Hospital La Paz, IdiPaz, Madrid, Spain
- ⁴⁶ Copenhagen Center for Arthritis Research, Center for Rheumatology and Spine Diseases, Centre for Head and Orthopaedics, Rigshospitalet, Glostrup, Denmark
- ⁴⁷ Department of Clinical Medicine, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark
- ⁴⁸ Rheumatology Unit, Hospital de Clinicas, Universidade Federal de Uberlandia, Uberlandia, Minas Gerais, Brazil
- ⁴⁹ Division of Rheumatology, Department of Medicine, Western University, London, ON, Canada
- ⁵⁰ Division of Rheumatology, Azienda USL-IRCCS di Reggio Emilia and Università di Modena e Reggio Emilia, Reggio Emilia, Italy
- ⁵¹ Clinical Hospital of Infectious Diseases, Rheumatology Outpatient Division, Cluj-Napoca, Romania
- ⁵² Cambridge University Hospitals NHS Trust, Cambridge, Cambridge, UK
- ⁵³ Department of Medicine, University of Cambridge School of Clinical Medicine, Cambridge, UK
- ⁵⁴ Division of Rheumatology, Department of Internal Medicine, Izmir Katip Celebi University School of Medicine, Izmir, Türkiye
- ⁵⁵ Rheumatology Service, Internal Medicine Service, Hospital Italiano de Buenos Aires, and University Hospital Italiano de Buenos Aires, Argentina
- ⁵⁶ Department of Medicine & Therapeutics, The Chinese University of Hong Kong, Hong Kong, People's Republic of China
- ⁵⁷ Rheumatology discipline, Medical School of São José do Rio Preto (FAMERP), São José do Rio Preto, São Paulo, Brazil
- ⁵⁸ Department of Allergy, Immunology & Rheumatology, Chung Shan Medical University Hospital, Taichung, Taiwan
- ⁵⁹ Graduate Institute of Integrated Medicine, China Medical University, Taichung, Taiwan
- ⁶⁰ Rheumatology Department, Saint Joseph University and Hotel-Dieu de France Hospital, Beirut, Lebanon

ARTICLE INFO

Article history:

Received 1 September 2025

Received in revised form 27 February 2026

Accepted 28 February 2026

Handling editor: Josef Smolen

ABSTRACT

Objectives: The Axial Involvement in Psoriatic Arthritis (AXIS) cohort aimed at evaluating the frequency of and clinical and imaging features of axial involvement in psoriatic arthritis (PsA).

Methods: AXIS (NCT04434885) is a prospective, multicentre, cross-sectional study conducted in 19 countries, by the Assessment of SpondyloArthritis International Society and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis. Participants with a diagnosis of PsA meeting CLASSification criteria for Psoriatic ARthritis with musculoskeletal symptom duration ≤ 10 years and no prior exposure to biological or targeted synthetic disease-modifying anti-rheumatic drugs were consecutively included. Standardised clinical, laboratory, and imaging assessments (radiography and magnetic resonance imaging of the axial skeleton, including sacroiliac joints [SIJs] and spine), were performed. Imaging was reviewed locally and centrally to detect axial involvement. The presence of axial involvement was determined by local investigator judgement before and after central-imaging review.

Results: Among 409 participants, axial involvement was identified in 153 (37.4%) based on the investigator's initial assessment and was decreased to 112 (27.4%) in the final evaluation after incorporating central-imaging review. Participants with axial involvement were younger (45.2 ± 13.8 vs 47.6 ± 12.6 years), more often male (56.3% vs 51.5%), and had a higher frequency of human leukocyte antigen (HLA)-B*27 positivity (22.4% vs 10.8%), inflammatory back pain (IBP) (74.7% vs 43.4%), and elevated C-reactive protein (CRP) (52.7% vs 37.4%). Active inflammatory and structural imaging changes were highly discriminative between participants with and without axial involvement. The central review identified imaging signs of axial involvement (active inflammation or structural lesions) in 95 participants (23.2%).

Conclusions: Axial involvement was identified in 27.4% of participants with PsA after final diagnostic assessment, with associated features including HLA-B*27 positivity, IBP, elevated CRP, and imaging changes in SIJ or spine.

INTRODUCTION

Psoriatic disease is a chronic immune-mediated condition that commonly affects the skin, nails, and musculoskeletal system. Psoriatic arthritis (PsA) involves peripheral musculoskeletal structures and, in some cases, axial structures such as the sacroiliac joints (SIJs) and spine and can occur in up to 30% of patients with psoriasis [1,2]. The prevalence of axial involvement in PsA has been reported to range from 25% to 70% of patients, depending on the definition used [3–9], and this involvement is associated with more severe disease and reduced quality of life [10]. However, there is currently no universally accepted definition of axial involvement in PsA, which poses challenges for diagnosis, classification, and research.

Although axial involvement in PsA shares similarities with axial spondyloarthritis (axSpA), there are features of PsA that might be relevant for the diagnosis and classification of axial involvement. These include a reported higher frequency of isolated spinal involvement, later onset of back pain, and lower frequency of inflammatory back pain (IBP) and of human leukocyte antigen (HLA)-B*27 positivity [11,12]. Furthermore, the efficacy of biologic or targeted synthetic disease-modifying antirheumatic drugs (DMARDs) in the axial domain of PsA remains an area of uncertainty, as few clinical trials have specifically focused on this subgroup [13], with most analyses being post hoc from clinical trials for peripheral PsA [14,15].

In response to the need for a more precise definition, the Assessment of SpondyloArthritis International Society (ASAS) and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) began efforts to determine the prevalence of axial involvement, and ultimately to develop a uniform consensus definition for axial involvement in PsA. To address the current knowledge gap, the Axial Involvement in Psoriatic Arthritis Cohort (AXIS) study was initiated [16]. The aims of the AXIS study were to determine the prevalence of axial

involvement in PsA, identify associated factors, and support the consensus definition development process.

The objective of the present work was to perform a comprehensive evaluation of the clinical and imaging characteristics of the participants with PsA included in the cohort and to identify similarities and differences between participants with and without axial involvement.

METHODS

Study design, population, and eligibility

The AXIS study (NCT04434885) is a multicentre, multinational, cross-sectional study. It represents an observational cohort study conducted within the ASAS-GRAPPA collaborative initiative. The study is designed to generate data to inform the development of a uniform definition for axial involvement in PsA. The present manuscript reports the main descriptive findings from this cohort, focusing on the frequency and characteristics of axial involvement in PsA. Further details regarding the study protocol, eligibility criteria, and data collection methods have already been published [16]. Briefly, eligible participants were prospectively recruited from selected study centres and underwent standardised examinations. A scientific committee appointed a national coordinator for each participating country. In total, 41 investigators from 19 countries—rheumatologists who were members of ASAS or GRAPPA—enrolled participants consecutively as they presented at their clinics.

Inclusion criteria required participants to be:

- Adults (≥ 18 years) with a confirmed diagnosis of PsA by their treating rheumatologist.
- Meeting the CLASSification criteria for Psoriatic ARthritis [17].

WHAT IS ALREADY KNOWN ON THIS TOPIC

- The prevalence of axial involvement in psoriatic arthritis (PsA) has been reported to range from 25% to 70% of participants, depending on the definition used, and this involvement is associated with more severe disease and reduced quality of life.
- Axial involvement in PsA is poorly characterised compared with axial spondyloarthritis, with limited understanding of its clinical and imaging features.

WHAT THIS STUDY ADDS

- This study systematically evaluated axial involvement in PsA using a standardised, multicentre approach with comprehensive imaging. This included systematic assessment of 4 axial imaging modalities (sacroiliac joint [SIJ] and spine radiographs and magnetic resonance imaging) with both local and central evaluation.
- Axial involvement was identified in 27.4% of participants with PsA after final investigator's diagnostic evaluation. Participants with axial involvement had distinct clinical and imaging features compared with participants without axial involvement, including a higher frequency of human leukocyte antigen-B*27 positivity, inflammatory back pain, elevated acute phase reactants, and imaging evidence of SIJ and spinal involvement.
- Estimates of axial involvement differed across staged evaluations (local initial, local-final [postcentral imaging], and central clinical review), illustrating how case definitions vary depending on the setting. This provides an empirical benchmark for future study design by clarifying how different evaluation strategies can influence cohort composition and the observed phenotype.
- A subset of participants showed spinal imaging abnormalities in the absence of SIJ changes, supporting evaluation of both anatomical regions when imaging is used to define axial involvement.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- This study underscores the importance of imaging in the objective confirmation of axial involvement in PsA.
- The findings provide a foundation for developing a uniform definition for axial involvement in PsA for clinical trials. The observed shifts across evaluation layers highlight which combinations of clinical imaging a future definition will need to address explicitly.

- Having a musculoskeletal symptom duration of PsA ≤ 10 years.
- Treatment-naïve to biologic or targeted synthetic DMARDs.
- Able to understand and complete study questionnaires.

Data collection

We collected data using a standardised electronic case report form, which captured information in the following 3 main categories detailed in the study protocol [16]:

1. Clinical: demographic and disease (both psoriasis and PsA)-related characteristics were collected as described in the protocol [16] (Supplementary material Methods).
2. Laboratory: C-reactive protein (CRP), erythrocyte sedimentation rate, and HLA-B*27 status were assessed at the local laboratories of each centre. In addition, blood

samples of participants were sent to the central laboratory at the University of Leeds for detailed genotyping.

3. Imaging: complete imaging of the axial skeleton that included both conventional radiographs and magnetic resonance imaging (MRI) of the SIJ and the spine according to a standard imaging protocol [16].

Study procedures and diagnostic assessments

The study diagnostic procedures included 2 main parts: local investigator's assessments, and in parallel, central-imaging and central clinical reviewers' assessments (Supplementary Fig S1).

Local investigators' assessments of imaging and axial involvement

Local investigators were responsible for making 3 key assessments:

1. Imaging assessments: local investigators provided their assessment of the presence of imaging changes indicative of axial involvement, including radiographs and MRIs. The sacroiliac (SI) radiographs were assessed based on the modified New York (mNY) criteria [18], whereas spinal radiographs were assessed to determine whether the changes were indicative of axial involvement of PsA. The findings on the SI and spinal MRIs included a global evaluation of whether each MRI was indicative of axial involvement, along with evaluations of the presence of typical active inflammatory and structural changes [16].
2. Initial diagnostic assessment: at this initial step, site principal investigators integrated clinical, laboratory, and imaging data (interpreted locally at this stage) to determine the presence of axial involvement. The level of confidence (LoC) in this determination was recorded on a scale ranging from -5 (definitely not) to $+5$ (definitely yes).
3. Final diagnostic assessment: in conclusion, after reviewing all available information, including the central image review reports (see below), the site principal investigator, who was the only clinician who actually assessed the patient, provided a final diagnostic assessment. They indicated whether the participant's clinical, laboratory, and imaging findings were compatible with axial involvement, along with their LoC in this determination.

Central assessments of imaging and axial involvement

Two central assessments were carried out independently by 2 specialised groups: the central-imaging experts and the central clinical reviewers:

- Central-imaging assessment: the central-imaging reviewers—3 rheumatologists (XB, WPM, and MØ) and 3 musculoskeletal radiologists (TD, KGH, and RGWL) with expertise in axSpA and PsA—conducted an independent review of the imaging data. Two primary central reviewers, 1 radiologist and 1 rheumatologist, independently evaluated the images of each case. If there was a disagreement between the 2 reviewers, an adjudicator—an independent radiologist who was not involved in the initial review—resolved the discrepancy. Further details on the specific imaging evaluation parameters and detailed evaluation criteria are provided in the Supplementary material (Methods—Central imaging assessment).

- **Central clinical assessment:** the central clinical reviewers, comprising 4 expert rheumatologists (FVdB, DDG, PM, and DP), undertook a comprehensive review of all available clinical, laboratory, and imaging data, including the assessments and reports provided by the central-imaging reviewers. The local investigator's diagnosis was not provided to ensure that the central clinical reviewers remained blinded to this information during their data review. Each case was randomly assigned to 2 of the central clinical reviewers, who then conducted an independent assessment of whether the overall presentation of the case was indicative of axial involvement in PsA and indicated their LoC. In the event of a discrepancy between the 2 reviewers' assessments regarding the presence of axial involvement, an independent adjudicator, who was not involved in the initial review, resolved the conflicting assessment.

Statistical analysis

Diagnostic assessments were made and presented according to 3 different diagnostic definitions: the local investigator's initial diagnostic assessment, the local investigator's final diagnostic assessment, and the central clinical reviewers' diagnostic assessment. For each of these assessments, participants were classified into 2 categories: 'Axial involvement' and 'No axial involvement'. Descriptive statistics summarised participant

characteristics within these categories (with full details of statistical comparisons provided in the [Supplementary material](#)) [19,20].

RESULTS

A total of 428 participants were screened, and 409 were enrolled in the AXIS study between July 2021 and November 2023, across 41 centres in 19 countries ([Supplementary Figs S1 and S2](#)).

Diagnostic assessments after each step

Figure 1 illustrates the flow of diagnostic assessments across 3 stages: the local investigator's initial assessment, the central-imaging review, and the local investigator's final assessment after consideration of the central-imaging results. In the local investigator's initial assessment, 153 (37.4%) participants were determined as having the presence of axial involvement. According to the central-imaging review, 96 (23.5%) participants exhibited evidence of at least 1 imaging modality indicative of axial involvement. In the final assessment, the local investigators determined that 112 (27.4%) participants had axial involvement. Overall, reidentification occurred in both directions, and the full branching structure is shown in Figure 1.

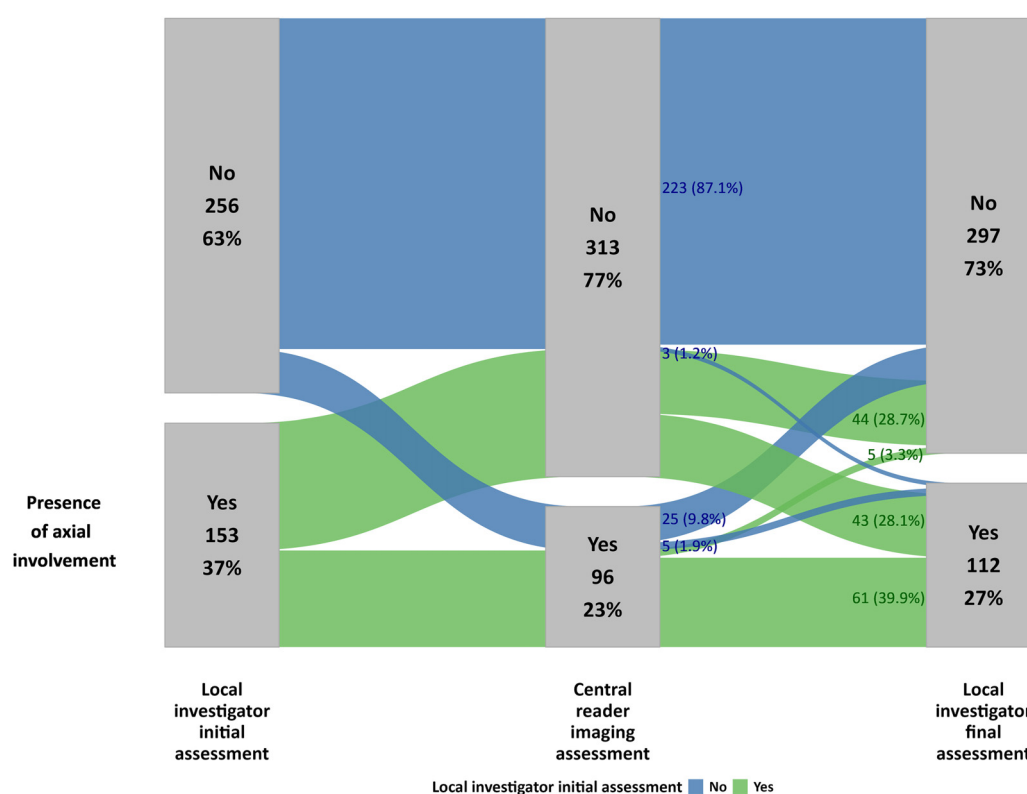


Figure 1. Parallel set plot showing the flow of changes from the local investigator's initial to the final assessment. The flow of axial-involvement identification through 3 successive assessments: local investigator initial assessment → central reader imaging assessment → local investigator final assessment. Grey bars show at each stage the absolute n and the percentage of the full study sample (N = 409). Colour coding: blue ribbons denote patients identified as having no axial involvement by the local investigator's initial assessment; green ribbons denote patients identified as having the presence of axial involvement at that stage. Ribbon labels display 'n (x.x%)', where n is the number of patients following that exact pathway and x.x% is that n expressed as a percentage of the total in the ribbon's source bar (ie, the group at local investigator initial assessment for that colour). For example: The top blue ribbon (Local No → ... → Final No) is labelled 223 (87.1%), meaning that 223 of the 256 patients (87.1%) locally defined as 'No' at initial assessment, had no compatible imaging changes according to central-imaging readers, and remained 'No' on central clinical review. The bottom green ribbon (Local Yes → ... → Final Yes) labelled 61 (39.9%), meaning that 61 of the 153 patients (39.9%) locally defined as 'Yes' at initial assessment were identified as having axial involvement by both central imaging and on final assessment.

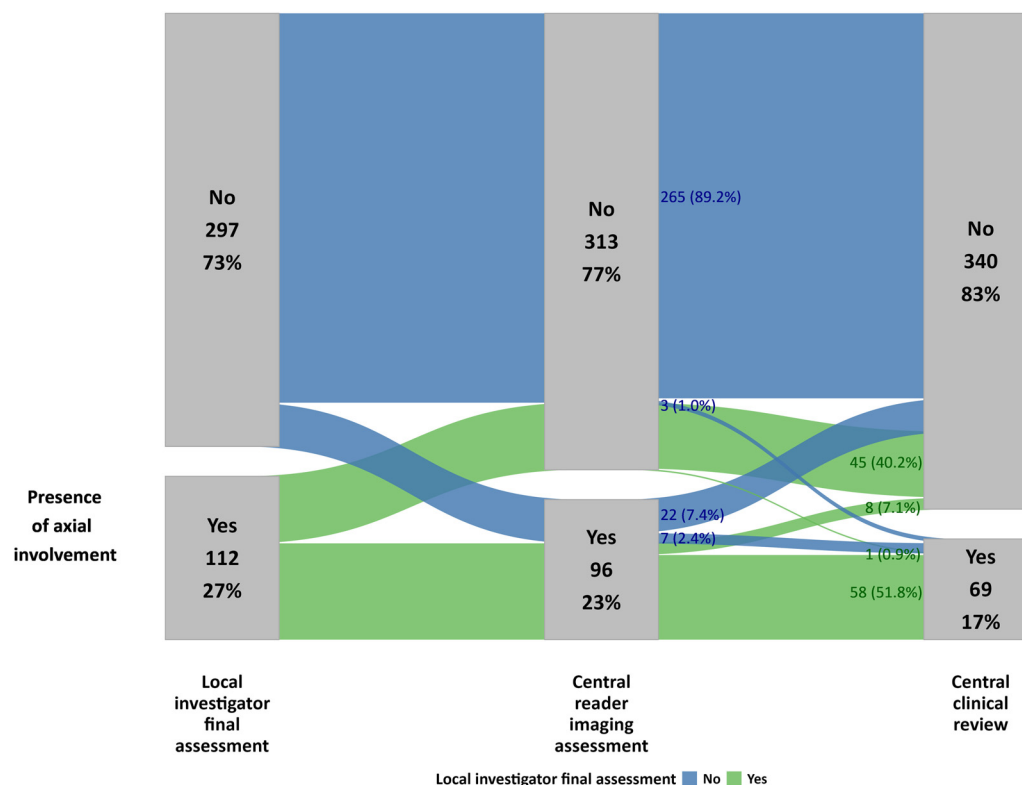


Figure 2. Parallel set plot showing the flow of changes from local-final assessment to central clinical review consensus assessment. The flow of axial-involvement identification through 3 successive assessments: local investigator final assessment → central reader imaging assessment → central clinical review. Grey bars show at each stage the absolute n and the percentage of the full study sample (N = 409). Colour coding: blue ribbons denote patients identified as having no axial involvement by the local investigator's final assessment; green ribbons denote patients identified the presence of axial involvement. Ribbon labels display “n (x.x%)”, where n is the number of patients following that exact pathway and x.x% is that n expressed as a percentage of the total in the ribbon's source bar (ie, the group at local investigator final assessment for that colour). For example: The top blue ribbon (Local No → ... → Clinical No) is labelled 265 (89.2%), meaning that 265 of the 297 patients (89.2%) locally defined as ‘No’ at final assessment, had no compatible imaging changes according to central-imaging readers, and remained ‘No’ on central clinical review. The bottom green ribbon (Local Yes → ... → Clinical Yes) labelled 58 (51.8%), meaning that 58 of the 112 patients (51.8%) locally defined as ‘Yes’ were identified as having axial involvement by both central imaging and central clinical review.

Central-imaging breakdown among 112 patients who identified as having axial involvement at the final investigator assessment stage: The central clinical review identified axial involvement in 59 patients (52.7%) and no axial involvement in the remaining 53 patients (47.3%). Among these 59 positive cases, 58/59 = 98.3% were identified as ‘Yes’ by central image readers, whereas 1/59 = 1.7% were identified as ‘No’. Among the 53 cases identified as ‘No’ by central clinical review, 45/53 = 84.9% had also been identified as ‘No’ by central-imaging readers, while 8/53 (15.1%) had nonetheless been identified as ‘Yes’ by central-imaging readers.

Of the 256 participants initially identified by the local investigators as having no axial involvement, 223 (87.1%) remained identified as such in the final assessment. Eight participants (3.1%) were reclassified as having axial involvement: 5 based on positive central-imaging findings and 3 despite negative central imaging. Among participants initially identified as having axial involvement (n = 153), 44 (28.7%) had their diagnosis revised to no axial involvement following central-imaging findings considered not indicative of axial involvement, whereas 43 (28.1%) remained identified as having axial involvement despite central-imaging findings not indicative of axial involvement.

Figure 2 illustrates the flow of assessment of axial involvement in PsA through the following 3 stages: the local investigator's final assessment, the central-imaging review, and the central clinical review. The central clinical review identified 69 (17%) participants as having axial involvement. Of the 112 participants with axial involvement in the local investigator's final assessment, 59 (52.7%) were identified by the central clinical review, whereas of the 297 participants without axial involvement, 287 (96.6%) were identified as such by the central clinical review.

Demographic characteristics

Comparisons between participants with and without axial involvement—based on both the local investigators' initial and final diagnostic assessments and consensus diagnosis of the central clinical review—are provided in the tables. Here, we highlight key comparisons based on the local investigators' final diagnostic assessment (as it reflects an integrated view combining local clinical judgement and central-imaging information) and, where necessary, comparison with central clinical review. This approach provides a balanced overview without implying that any single assessment represents a definitive reference/gold standard.

The demographic characteristics were largely comparable between participants with (mean age 45.2 ± 13.8 years, 56.3% male) and without axial involvement (mean age 47.6 ± 12.6 years, 51.5% male) based on final investigator assessment. However, according to the central clinical review, participants with axial involvement were more often male (73.9% vs 48.5%), younger (43.7 ± 13.9 vs 47.6 ± 12.7), and with lower mean body mass index (27.7 ± 14.0 vs 28.4 ± 5.9 kg/m²) (Table 1 and Fig 3).

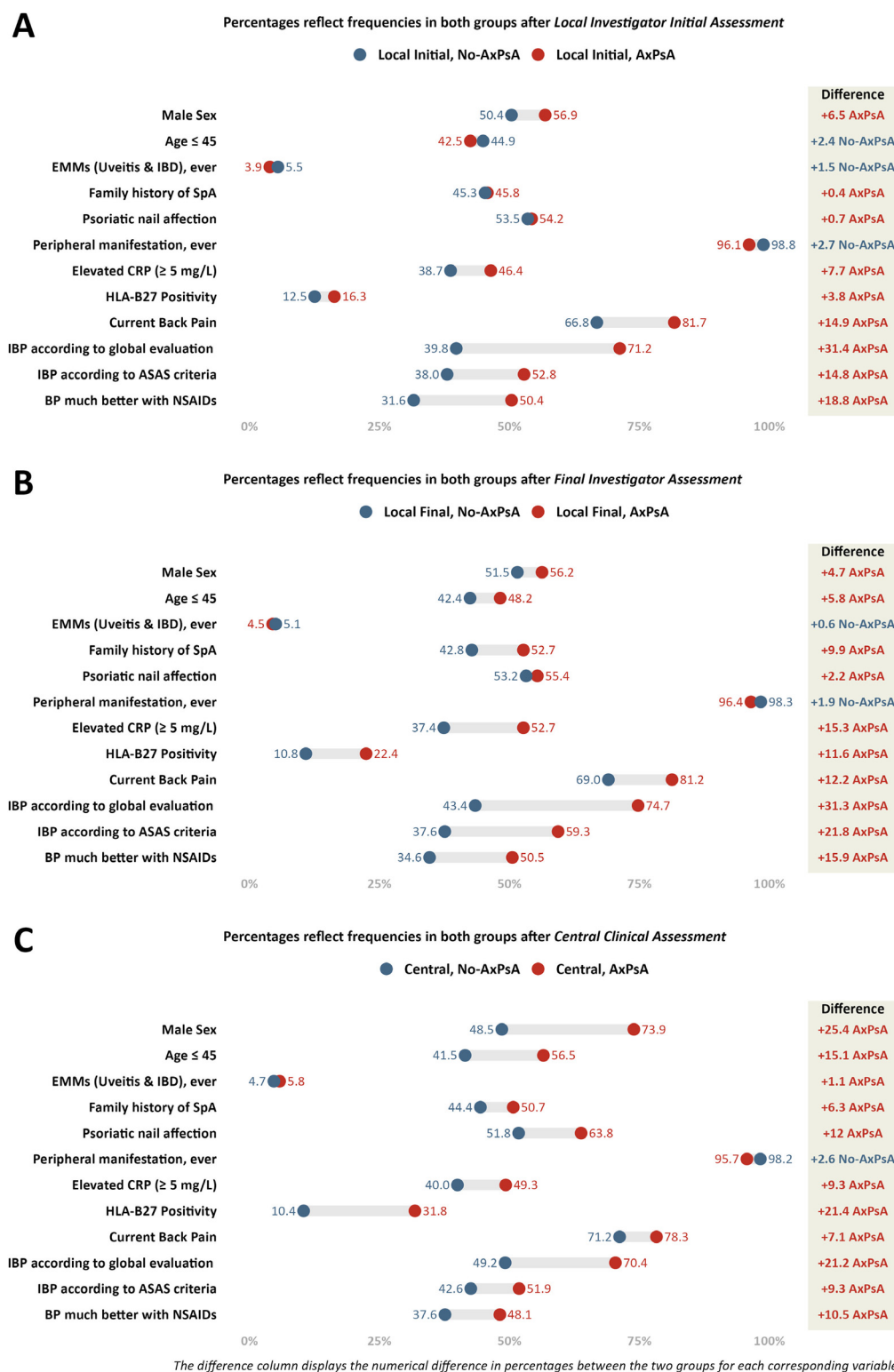


Figure 3. Dumbbell plot highlighting differences in key clinical features between patients with and without axial involvement: (A) at initial assessment by local investigator, (B) at final assessment by local investigator, and (C) at central clinical review. ASAS, Assessment of SpondyloArthritis International Society; AxPsA, Axial Psoriatic Arthritis; BP, back pain; CRP, C-reactive protein; EMM, extramusculoskeletal manifestation; HLA, human leukocyte antigen; IBD, inflammatory bowel disease; IBP, inflammatory back pain; NSAIDs, nonsteroidal anti-inflammatory drugs; SpA, spondyloarthritis.

Disease characteristics, treatments, extra musculoskeletal manifestations, and family history

In total, 96.3% of participants had a history of or a current psoriasis (Table 1). The mean symptom duration of psoriasis and PsA was 14.7 ± 12.7 and 4.1 ± 2.9 years, respectively, and largely comparable between participants with and without

axial involvement. The treatment modalities for psoriasis were comparable, but treatment patterns for PsA differed between groups (Table 1), with participants with axial involvement more frequently treated with nonsteroidal anti-inflammatory drugs (NSAIDs) (88.4% vs 80.1%) and sulfasalazine (22.3% vs 12.1%) and less frequently with methotrexate (55.4% vs 72.1%) compared with those without axial involvement.

According to the central clinical review, similar trends were observed.

Extramusculoskeletal manifestations (EMMs, other than psoriasis) were present in 20 participants (4.9%), predominantly uveitis ($n = 18$), with inflammatory bowel disease being rare ($n = 3$), and these frequencies were similar between groups (Table 1 and Fig 3A,B). To explore whether this observation might reflect HLA-B*27-associated phenotypes, EMM frequencies were also examined by HLA-B*27 status and showed no meaningful difference between HLA-B*27-positive and -negative participants (8.9% vs 4.0%, respectively). A family history of spondyloarthritis (SpA) was higher in participants with axial involvement compared with those without (52.7% vs 42.8%). The distribution of specific SpA entities in the family history is provided in Table 1. In comparisons based on the central clinical review, there were no notable differences observed.

Back pain characteristics

A total of 296 participants (72.3%) reported back pain at the current visit. The age of onset and duration of current back pain were similar between participants with and without axial involvement (Table 2). A higher proportion of participants with axial involvement reported current IBP according to the investigator's global evaluation (74.7% vs 43.4%), more often fulfilled the ASAS criteria for IBP (59.3% vs 37.6%), and had good response to NSAIDs (50.5% vs 34.6%). Similar patterns were observed in the central clinical review. Notably, differences in NSAID response and fulfilment of ASAS IBP criteria were smaller in the central clinical review than in the final investigator assessment. Figure 3 summarises these current back pain characteristics across diagnostic evaluations.

Physical examination, disease activity, and laboratory findings

Table 2 also compares the clinical and laboratory characteristics related to psoriasis and PsA. Psoriatic skin and nail involvement and Psoriasis Area and Severity Index scores were similar between participants with and without axial involvement. Nearly all participants (97.8%) had either a history of or current peripheral musculoskeletal manifestations regardless of axial involvement status. Peripheral manifestations and related counts (tender/swollen joint counts, enthesitis, and dactylitis) were broadly comparable between groups. The physician global assessment was found to be higher in participants with axial involvement than in those without (4.6 ± 2.3 vs 3.7 ± 2.2). However, no differences were observed in the Bath Ankylosing Spondylitis Disease Activity Index (4.7 ± 2.5 vs 4.3 ± 2.5), whereas both the Axial Spondyloarthritis Disease Activity Score (ASDAS) and CRP were higher in participants with axial involvement (ASDAS: 2.9 ± 1.1 vs 2.6 ± 1.0 ; CRP: 9.8 ± 12.3 mg/L vs 6.2 ± 8.7 mg/L). Furthermore, a higher frequency of HLA-B*27 positivity was observed in participants with axial involvement (22.4% vs 10.8%) (Fig 3B).

In comparisons based on the central clinical review, HLA-B*27 positivity (31.8% vs 10.4%) and CRP (11.1 ± 14.2 mg/L vs 6.4 ± 8.6 mg/L) remained higher, and even more markedly so, in participants with axial involvement. However, most remaining clinical variables were similar between groups; the main exception was peripheral enthesitis, which was less frequent in participants with axial involvement (37.7% vs 51.8%) (Table 2 and Fig 3C).

Local investigator imaging interpretation and diagnostic assessment

Figure 4 presents a comparison of the local investigator's imaging assessments according to 3 diagnostic assessment categories. A total of 175 participants (42.8%) had at least 1 positive imaging among 4 modalities (radiographs or MRIs of the SIJ, or spine), indicative of axial involvement. Following the initial assessment, 143 participants (93.5%) with axial involvement exhibited at least 1 positive imaging, whereas 10 participants (6.5%) were diagnosed with axial involvement despite negative imaging findings (Fig 4A).

Looking at the categories of final investigator assessment, 101 of 112 participants (90.2%) with axial involvement had at least 1 positive imaging result according to the local investigator (Fig 4B). Furthermore, 74 of 297 participants (24.9%) without axial involvement exhibited imaging findings according to the local investigator's assessment, but these changes were interpreted as not sufficiently specific to support a diagnosis of axial involvement. Among participants with axial involvement after the final investigator assessment, SIJ-MRI positivity was more frequent than definite radiographic sacroiliitis, whereas positive spinal imaging was observed similarly on both radiographs and MRI (Fig 4B). Structural lesions were more frequent than active inflammatory lesions on SIJ MRI according to local investigator interpretation, but on spinal MRI, active inflammatory and structural lesions had a similar frequency (Fig 4B).

When considering SIJ and spine together, a subgroup of participants showed isolated spinal findings in the absence of SIJ changes: 21 participants (18.8%) with axial involvement had mNY-negative SIJ radiographs but positive spinal radiographs, and 18 participants (16.1%) had no SIJ-MRI findings but typical changes on spinal MRI according to local investigator imaging evaluation (Supplementary Table S1). These patterns highlight that relying on a single anatomical region or modality might potentially miss a subset of participants with imaging findings in another region.

Similar trends were observed in the central-imaging readers' consensus assessments, but with lower proportions across modalities (Fig 4A,B), consistent with the study design. Stratification by central clinical review yielded comparable differences between participants with and without axial involvement (Fig 4C).

Supplementary Figure S3A,B illustrates the distribution of local investigators' LoC in their diagnostic assessments of axial involvement. LoC shifted towards higher confidence at the final assessment after central-imaging review (Supplementary Fig S3A,B).

Central-imaging reading consensus assessment for SIJs and spine

Central-imaging readers' consensus assessment of SI radiographs and MRI is summarised in Table 3. Overall, definite radiographic sacroiliitis was present in 27 participants (6.6%). On MRI, 62 (15.2%) had SIJ MRI judged indicative of axial involvement, 31 (7.6%) showed typical active inflammatory lesions, and 29 (7.1%) fulfilled ASAS criteria for sacroiliitis on MRI. Typical active inflammatory lesions represent a stricter subset than the presence of any inflammatory signal; accordingly, bone marrow oedema (BMO) was observed more frequently than typical active lesions overall. Active inflammatory SIJ-MRI lesions were most often BMO, observed in 80 participants (19.6%), whereas other inflammatory features were uncommon (Table 3). Structural lesions were recorded in 56

Table 1
Demographic, participant, and disease characteristics

	Overall	Local investigator initial assessment of axial involvement			Local investigator final assessment of axial involvement			Central clinical review consensus categories of axial involvement		
Variable	N = 409	Yes-Initial N = 153	No-Initial N = 256	P value	Yes-Final N = 112	No-Final N = 297	P value	Yes-Central N = 69	No-Central N = 340	P value
Demographic and participant characteristics										
Male sex	216 (52.8)	87 (56.9)	129 (50.4)	.20	63 (56.3)	153 (51.5)	.39	51 (73.9)	165 (48.5)	<.001
Age (y)	47.0 ± 13.0	47.2 ± 13.6	46.9 ± 12.6	.75	45.2 ± 13.8	47.6 ± 12.6	.10	43.7 ± 13.9	47.6 ± 12.7	.011
Ethnicity				.39			.60			.051
White European	252 (61.6)	102 (66.7)	150 (58.6)		75 (67.0)	177 (59.6)		36 (52.2)	216 (63.5)	
White North American	20 (4.9)	5 (3.3)	15 (5.9)		3 (2.7)	17 (5.7)		1 (1.4)	19 (5.6)	
Asian	81 (19.8)	24 (15.7)	57 (22.3)		19 (17.0)	62 (20.9)		22 (31.9)	59 (17.4)	
White Arabic	7 (1.7)	2 (1.3)	5 (2.0)		1 (0.9)	6 (2.0)		0 (0.0)	7 (2.1)	
Black	2 (0.5)	1 (0.7)	1 (0.4)		0 (0.0)	2 (0.7)		0 (0.0)	2 (0.6)	
Others	47 (11.5)	19 (12.4)	28 (10.9)		14 (12.5)	33 (11.1)		10 (14.5)	37 (10.9)	
Manual occupation	183 (44.7)	71 (46.4)	112 (43.8)	.60	56 (50.0)	127 (42.8)	.19	33 (47.8)	150 (44.1)	.57
Education, college or above	214 (52.3)	76 (49.7)	138 (53.9)	.41	57 (50.9)	157 (52.9)	.72	41 (59.4)	173 (50.9)	.20
Smoking, ever	181 (44.3)	65 (42.5)	116 (45.3)	.58	48 (42.9)	133 (44.8)	.73	24 (34.8)	157 (46.2)	.082
Alcohol, ever	193 (47.2)	78 (51.0)	115 (44.9)	.23	53 (47.3)	140 (47.1)	.97	26 (37.7)	167 (49.1)	.083
Body mass index (kg/m²)	28.3 ± 7.8	28.1 ± 9.9	28.4 ± 6.3	.28	28.1 ± 11.4	28.4 ± 6.0	.17	27.7 ± 14.0	28.4 ± 5.9	.010
Disease characteristics										
Patient's history of psoriasis				.29			.92			.92
Current	337 (82.4)	123 (80.4)	214 (83.6)		91 (81.3)	246 (82.8)		57 (82.6)	280 (82.4)	
Previous	57 (13.9)	26 (17.0)	31 (12.1)		17 (15.2)	40 (13.5)		9 (13.0)	48 (14.1)	
Never	15 (3.7)	4 (2.6)	11 (4.3)		4 (3.6)	11 (3.7)		3 (4.3)	12 (3.5)	
Psoriasis symptom duration, y	14.7 ± 12.7	14.3 ± 11.8	14.9 ± 13.3	.91	13.7 ± 11.5	15.0 ± 13.2	.65	13.3 ± 9.8	15.0 ± 13.3	.95
Any topical treatment, ever	348 (85.1)	131 (85.6)	217 (84.8)	.81	96 (85.7)	252 (84.8)	.83	60 (87.0)	288 (84.7)	.63
Phototherapy, ever	58 (14.2)	20 (13.1)	38 (14.8)	.62	16 (14.3)	42 (14.1)	.97	8 (11.6)	50 (14.7)	.50
Systemic corticosteroid for psoriasis, ever	24 (5.9)	9 (5.9)	15 (5.9)	>.99	7 (6.3)	17 (5.7)	.84	5 (7.2)	19 (5.6)	.58
Systemic retinoid for psoriasis, ever	8 (2.0)	3 (2.0)	5 (2.0)	>.99	4 (3.6)	4 (1.3)	.22	1 (1.4)	7 (2.1)	>.99
Cyclosporine for psoriasis, ever	7 (1.7)	4 (2.6)	3 (1.2)	.43	3 (2.7)	4 (1.3)	.40	2 (2.9)	5 (1.5)	.34
Methotrexate for psoriasis, ever	87 (21.3)	32 (20.9)	55 (21.5)	.89	22 (19.6)	65 (21.9)	.62	12 (17.4)	75 (22.1)	.39
Age of PsA onset, y	42.9 ± 12.9	42.8 ± 13.4	42.9 ± 12.6	.99	41.0 ± 13.8	43.6 ± 12.5	.083	39.2 ± 13.7	43.6 ± 12.6	.006
PsA symptom duration, y	4.1 ± 2.9	4.4 ± 3.0	3.9 ± 2.8	.13	4.2 ± 2.8	4.1 ± 2.9	.57	4.5 ± 2.9	4.0 ± 2.9	.17
Intra-articular corticosteroid for PsA, ever	94 (23.0)	33 (21.6)	61 (23.8)	.60	22 (19.6)	72 (24.2)	.32	12 (17.4)	82 (24.1)	.23
NSAID for PsA, ever	337 (82.4)	133 (86.9)	204 (79.7)	.063	99 (88.4)	238 (80.1)	.051	62 (89.9)	275 (80.9)	.074
Systemic corticosteroid for PsA, ever	114 (27.9)	43 (28.1)	71 (27.7)	.94	34 (30.4)	80 (26.9)	.49	18 (26.1)	96 (28.2)	.72
Methotrexate for PsA, ever	276 (67.5)	88 (57.5)	188 (73.4)	<.001	62 (55.4)	214 (72.1)	.001	44 (63.8)	232 (68.2)	.47
Leflunomide for PsA, ever	43 (10.5)	19 (12.4)	24 (9.4)	.33	14 (12.5)	29 (9.8)	.42	11 (15.9)	32 (9.4)	.11
Sulfasalazine for PsA, ever	61 (14.9)	23 (15.0)	38 (14.8)	.96	25 (22.3)	36 (12.1)	.010	23 (33.3)	38 (11.2)	<.001
Cyclosporine for PsA, ever	9 (2.2)	3 (2.0)	6 (2.3)	>.99	2 (1.8)	7 (2.4)	>.99	3 (4.3)	6 (1.8)	.18
Any csDMARDs for PsA, ever	291 (71.1)	97 (63.4)	194 (75.8)	.007	69 (61.6)	222 (74.7)	.009	49 (71.0)	242 (71.2)	.98
History of any EMMs	20 (4.9)	6 (3.9)	14 (5.5)	.48	5 (4.5)	15 (5.1)	.81	4 (5.8)	16 (4.7)	.76
History of uveitis	18 (4.4)	5 (3.3)	13 (5.1)	.39	5 (4.5)	13 (4.4)	>.99	4 (5.8)	14 (4.1)	.52
History of IBD	3 (0.7)	2 (1.3)	1 (0.4)	.56	0 (0.0)	3 (1.0)	.57	0 (0.0)	3 (0.9)	>.99
Family history of any SpA in accordance with ASAS definition	186 (45.5)	70 (45.8)	116 (45.3)	.93	59 (52.7)	127 (42.8)	.072	35 (50.7)	151 (44.4)	.34
Family history of psoriasis	138 (33.7)	53 (34.6)	85 (33.2)	.77	44 (39.3)	94 (31.6)	.15	22 (31.9)	116 (34.1)	.72
Family history of PsA	37 (9.0)	15 (9.8)	22 (8.6)	.68	12 (10.7)	25 (8.4)	.47	8 (11.6)	29 (8.5)	.42
Family history of AxSpA	17 (4.2)	7 (4.6)	10 (3.9)	.74	7 (6.3)	10 (3.4)	.26	4 (5.8)	13 (3.8)	.50

ASAS, Assessment of SpondyloArthritis International Society; AxSpA, axial spondyloarthritis; csDMARDs, conventional disease-modifying antirheumatic drugs; EMMs, extramusculoskeletal manifestations; IBD, inflammatory bowel disease; NSAIDs, nonsteroidal anti-inflammatory drugs; PsA, psoriatic arthritis; SpA, spondyloarthritis.

The variables are presented as mean ± SD, or as number (%) unless otherwise indicated.

participants (13.7%), most frequently erosions (10.0%) and fat lesions (8.3%), whereas other structural features were uncommon. Across all 3 diagnostic levels, mNY-positive SIJ radiographs, SIJ MRI indicative of axial involvement, and typical active and structural SIJ-MRI lesions were consistently more frequent in participants classified as having axial involvement than in those without, with the exception of enthesitis, and, to a lesser extent, capsulitis, for which differences were small and not clearly discriminative.

Central readers judged 35 participants (8.6%) as having spinal radiographs indicative of axial involvement (Table 4). The changes compatible with SpA were uncommon overall but clustered in those classified as having axial involvement. Although marginal syndesmophytes and ossifications were seen in 25 participants (6.1%), other SpA-compatible structural changes were less, and no erosions, endplate syndesmophytes, or facet fusion were identified. Degenerative disc disease was frequent, being reported in 75 participants (18.3%) in the cohort. Spinal MRI

Table 2

Back pain, physical examination, activity, and laboratory characteristics

Variable	Overall	Local investigator initial assessment of axial involvement			Local investigator final assessment of axial involvement			Central clinical review consensus categories of axial involvement		
	N = 409	Yes-Initial N = 153	No-Initial N = 256	P value	Yes-Final N = 112	No-Final N = 297	P value	Yes-Central N = 69	No-Central N = 340	P value
Back pain characteristics										
History of BP				.005			.035			.49
Current	296 (72.4)	125 (81.7)	171 (66.8)		91 (81.3)	205 (69.0)		54 (78.3)	242 (71.2)	
Past	53 (13.0)	13 (8.5)	40 (15.6)		8 (7.1)	45 (15.2)		7 (10.1)	46 (13.5)	
Never	60 (14.7)	15 (9.8)	45 (17.6)		13 (11.6)	47 (15.8)		8 (11.6)	52 (15.3)	
Current BP*	296 (72.4)	125 (81.7)	171 (66.8)	.001	91 (81.3)	205 (69.0)	.014	54 (78.3)	242 (71.2)	.23
Current BP, buttocks/sacroiliac joints*	131 (44.3)	55 (44.0)	76 (44.4)	.94	46 (50.5)	85 (41.5)	.15	25 (46.3)	106 (43.8)	.74
Current BP, cervical spine*	131 (44.3)	56 (44.8)	75 (43.9)	.87	44 (48.4)	87 (42.4)	.34	22 (40.7)	109 (45.0)	.57
Current BP, thoracic spine*	75 (25.3)	39 (31.2)	36 (21.1)	.047	32 (35.2)	43 (21.0)	.010	16 (29.6)	59 (24.4)	.42
Current BP, lumbar spine*	220 (74.3)	92 (73.6)	128 (74.9)	.81	66 (72.5)	154 (75.1)	.64	41 (75.9)	179 (74.0)	.77
Current BP, spine*	277 (93.6)	118 (94.4)	159 (93.0)	.62	86 (94.5)	191 (93.2)	.67	51 (94.4)	226 (93.4)	>.99
Age of current BP onset <45 y*	218 (73.6)	89 (71.2)	129 (75.4)	.41	68 (74.7)	150 (73.2)	.78	41 (75.9)	177 (73.1)	.67
Duration of current BP, y*	12.1 ± 14.7	11.2 ± 14.9	12.7 ± 14.6	.62	10.8 ± 14.5	12.7 ± 14.8	.37	10.7 ± 14.7	12.4 ± 14.7	.33
Current BP, insidious onset*	220 (74.3)	111 (88.8)	109 (63.7)	<.001	82 (90.1)	138 (67.3)	<.001	46 (85.2)	174 (71.9)	.043
Current BP, IBP according to global evaluation of investigator*	157 (53.0)	89 (71.2)	68 (39.8)	<.001	68 (74.7)	89 (43.4)	<.001	38 (70.4)	119 (49.2)	.005
Current BP, IBP according to ASAS criteria*	131 (44.3)	66 (52.8)	65 (38.0)	.011	54 (59.3)	77 (37.6)	<.001	28 (51.9)	103 (42.6)	.21
Current BP is much better with NSAIDs*	117 (39.5)	63 (50.4)	54 (31.6)	.001	46 (50.5)	71 (34.6)	.010	26 (48.1)	91 (37.6)	.15
Physical examination										
Any psoriatic lesion	344 (84.1)	130 (85.0)	214 (83.6)	.71	95 (84.8)	249 (83.8)	.81	61 (88.4)	283 (83.2)	.28
Any psoriatic nail involvement	220 (53.8)	83 (54.2)	137 (53.5)	.89	62 (55.4)	158 (53.2)	.70	44 (63.8)	176 (51.8)	.068
PASI score	2.8 ± 3.8	3.0 ± 4.3	2.7 ± 3.5	.97	3.2 ± 4.3	2.7 ± 3.7	.45	3.6 ± 4.9	2.7 ± 3.6	.050
Peripheral arthritis, current	251 (61.4)	91 (59.5)	160 (62.5)	.54	66 (58.9)	185 (62.3)	.53	37 (53.6)	214 (62.9)	.15
TJC	4.7 ± 7.7	4.9 ± 8.5	4.5 ± 7.3	.80	5.6 ± 9.4	4.3 ± 7.0	.96	3.8 ± 6.4	4.8 ± 8.0	.15
SJC	2.6 ± 5.0	2.8 ± 5.9	2.4 ± 4.4	.73	3.3 ± 9.9	2.3 ± 4.0	.96	2.6 ± 4.6	2.6 ± 5.1	.53
Enthesitis, current	202 (49.4)	74 (48.4)	128 (50.0)	.75	55 (49.1)	147 (49.5)	.94	26 (37.7)	176 (51.8)	.033
SPARCC enthesitis score	1.7 ± 2.8	1.8 ± 3.1	1.7 ± 2.6	.56	1.9 ± 3.3	1.7 ± 2.6	.64	1.0 ± 2.1	1.8 ± 2.9	.014
MASES enthesitis score	1.3 ± 2.4	1.4 ± 2.5	1.3 ± 2.3	.79	1.4 ± 2.4	1.3 ± 2.4	.87	0.8 ± 1.8	1.4 ± 2.5	.050
LEI enthesitis score	0.8 ± 1.3	0.8 ± 1.3	0.7 ± 1.3	.92	0.9 ± 1.5	0.7 ± 1.2	.65	0.4 ± 0.9	0.8 ± 1.3	<.001
Dactylitis, current	66 (16.1)	25 (16.3)	41 (16.0)	.93	19 (17.0)	47 (15.8)	.78	11 (15.9)	55 (16.2)	.96
Dactylitis count	0.4 ± 1.2	0.5 ± 1.6	0.3 ± 0.8	.74	0.6 ± 1.8	0.3 ± 0.7	.54	0.6 ± 1.6	0.3 ± 1.0	.83
Any peripheral manifestation				.20			.27			.17
Current	311 (76.0)	115 (75.2)	196 (76.6)		88 (78.6)	223 (75.1)		48 (69.6)	263 (77.4)	
Previous	89 (21.8)	32 (20.9)	57 (22.3)		20 (17.9)	69 (23.2)		18 (26.1)	71 (20.9)	
Never	9 (2.2)	6 (3.9)	3 (1.2)		4 (3.6)	5 (1.7)		3 (4.3)	6 (1.8)	
Disease activity										
PGA (psoriasis and arthritis), 0–10 NRS	5.3 ± 2.5	5.4 ± 2.5	5.2 ± 2.5	.67	5.6 ± 2.5	5.2 ± 2.5	.18	5.2 ± 2.4	5.3 ± 2.5	.77
Participant reported back pain, 0–10 NRS	4.4 ± 3.1	4.8 ± 3.1	4.2 ± 3.0	.070	4.8 ± 3.1	4.3 ± 3.0	.16	4.3 ± 3.1	4.4 ± 3.1	.80
PhGA (MSK and Skin), 0–10 NRS	3.9 ± 2.2	4.3 ± 2.3	3.7 ± 2.2	.011	4.6 ± 2.3	3.7 ± 2.2	<.001	4.2 ± 2.3	3.9 ± 2.2	.21
DAPSA	18.4 ± 14.3	19.2 ± 15.9	18.0 ± 13.2	.67	20.8 ± 17.7	17.5 ± 12.6	.20	17.7 ± 13.3	18.6 ± 14.5	.66
ASDAS	2.7 ± 1.0	2.8 ± 1.1	2.6 ± 1.0	.083	2.9 ± 1.1	2.6 ± 1.0	.007	2.8 ± 1.1	2.6 ± 1.0	.49
BASDAI	4.4 ± 2.5	4.6 ± 2.5	4.4 ± 2.5	.44	4.7 ± 2.5	4.3 ± 2.5	.25	4.3 ± 2.3	4.5 ± 2.6	.75
Laboratory										
ESR (mm/h)	18.0 ± 16.6	18.2 ± 16.0	17.9 ± 16.9	.40	19.6 ± 16.1	17.5 ± 16.7	.048	18.5 ± 17.7	18.0 ± 16.4	>.99
CRP (mg/L)	7.2 ± 9.9	8.2 ± 11.0	6.6 ± 9.2	.038	9.8 ± 12.3	6.2 ± 8.7	<.001	11.1 ± 14.2	6.4 ± 8.6	.007
Elevated CRP (≥5 mg/L)	170 (41.6)	71 (46.4)	99 (38.7)	.12	59 (52.7)	111 (37.4)	.005	34 (49.3)	136 (40.0)	.15
HLA-B27 positivity (n = 402)	56 (13.9)	24 (16.3)	32 (12.5)	.29	24 (22.4)	32 (10.8)	.003	21 (31.8)	35 (10.4)	<.001

* n = 296; ASAS, Assessment of SpondyloArthritis International Society; ASDAS, Axial Spondyloarthritis Disease Activity Score; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BP, back pain; CRP, C-reactive protein; DAPSA, Disease Activity in Psoriatic Arthritis; ESR, erythrocyte sedimentation rate; HLA, human leukocyte antigen; IBP, inflammatory back pain; LEI, Leeds Enthesitis Index; MASES, Maastricht Ankylosing Spondylitis Enthesitis Score; MSK, musculoskeletal; NRS, Numerical Rating Scale; NSAIDs, nonsteroidal anti-inflammatory drugs; PASI, Psoriasis Area and Severity Index; PGA, Participant Global Assessment of disease activity (psoriasis and arthritis); PhGA, Physician Global Assessment of disease activity (musculoskeletal and skin); SJC, swollen joint count; SPARCC, Spondyloarthritis Research Consortium of Canada; TJC, tender joint count.

The variables are presented as mean ± SD, or as number (%) unless otherwise indicated.

Table 3
Central-imaging readers' consensus assessment of sacroiliac radiograph and MRI

Variable	Overall	Local investigator initial assessment of axial involvement			Local investigator final assessment of axial involvement			Central clinical review consensus categories of axial involvement		
	N = 409	Yes-Initial N = 153	No-Initial N = 256	P value	Yes-Final N = 112	No-Final N = 297	P value	Yes-Central N = 69	No-Central N = 340	P value
mNY-positive SIJ-radiograph	27 (6.6)	26 (17.0)	1 (0.4)	<.001	26 (23.2)	1 (0.3)	<.001	24 (34.8)	3 (0.9)	<.001
SIJ-MRI indicative of axial involvement	62 (15.2)	53 (34.6)	9 (3.5)	<.001	55 (49.1)	7 (2.4)	<.001	56 (81.2)	6 (1.8)	<.001
Typical active lesions in SIJ-MRI	31 (7.6)	30 (19.6)	1 (0.4)	<.001	30 (26.8)	1 (0.3)	<.001	29 (42.0)	2 (0.6)	<.001
ASAS criteria for SpA	29 (7.1)	28 (18.3)	1 (0.4)	<.001	28 (25.0)	1 (0.3)	<.001	27 (39.1)	2 (0.6)	<.001
Bone marrow oedema	80 (19.6)	53 (34.6)	27 (10.5)	<.001	43 (38.4)	37 (12.5)	<.001	33 (47.8)	47 (13.8)	<.001
Inflammation at the site of erosion cavity	4 (1.0)	4 (2.6)	0 (0.0)	.019	4 (3.6)	0 (0.0)	.005	4 (5.8)	0 (0.0)	<.001
Enthesitis	1 (0.2)	1 (0.7)	0 (0.0)	.37	1 (0.9)	0 (0.0)	.27	1 (1.4)	0 (0.0)	.17
Capsulitis	2 (0.5)	2 (1.3)	0 (0.0)	.14	2 (1.8)	0 (0.0)	.075	2 (2.9)	0 (0.0)	.028
Typical structural lesions in SIJ-MRI	56 (13.7)	46 (30.1)	10 (3.9)	<.001	48 (42.9)	8 (2.7)	<.001	50 (72.5)	6 (1.8)	<.001
Erosion	41 (10.0)	32 (20.9)	9 (3.5)	<.001	32 (28.6)	9 (3.0)	<.001	32 (46.4)	9 (2.6)	<.001
Fat lesion	34 (8.3)	27 (17.6)	7 (2.7)	<.001	28 (25.0)	6 (2.0)	<.001	27 (39.1)	7 (2.1)	<.001
Backfill	15 (3.7)	12 (7.8)	3 (1.2)	<.001	14 (12.5)	1 (0.3)	<.001	14 (20.3)	1 (0.3)	<.001
Sclerosis	30 (7.3)	18 (11.8)	12 (4.7)	.008	14 (12.5)	16 (5.4)	.014	10 (14.5)	20 (5.9)	.012
Bone bud	1 (0.2)	1 (0.7)	0 (0.0)	.37	1 (0.9)	0 (0.0)	.27	1 (1.4)	0 (0.0)	.17
Ankylosis	11 (2.7)	10 (6.5)	1 (0.4)	<.001	11 (9.8)	0 (0.0)	<.001	11 (15.9)	0 (0.0)	<.001

ASAS, Assessment of SpondyloArthritis International Society; mNY, Modified New York Criteria; MRI, magnetic resonance imaging; SIJ, sacroiliac joint; SpA, spondyloarthritis; STIR, Short Tau Inversion Recovery.

The variables are presented as number (%).

Typical acute/active inflammatory lesions in the SIJ are bone oedema/contrast medium enhancement within/adjacent to the SI joints or at enthesal sites; STIR and/or T1 + Gd sequences, or equivalent, are required. Typical structural lesions in the SIJ refer to the clear presence of typical findings such as sclerosis, erosions, backfill, fat lesions, bony bridges, or ankylosis.

was considered indicative of axial involvement in 49 participants (12.0%) (Table 4). Typical active spinal MRI lesions were present in 41 participants (10.0%), with BMO being the most frequent active lesion (20.3%). As for SIJ MRI, BMO reflects any inflammatory signal and was therefore more frequent than the subset of typical active lesions. Typical structural spinal MRI lesions were present in 28 participants (6.8%). Fat lesions were the most common structural change (14.7%), whereas erosions and new bone formation were infrequent. Across diagnostic categories, radiographs and active and structural spinal MRI lesions were more frequent among participants evaluated as having axial involvement than among those without.

When evaluating radiographic changes in the SIJ and spine together based on central-imaging reviewers' reports, we found that 7 participants (6.3%) with axial involvement had mNY-negative SIJ radiographs but positive spinal radiographs. We observed a similar pattern in the global evaluation of SIJ and spinal MRIs: 8 participants (7.1%) had no SIJ-MRI findings but typical changes on spinal MRI (Supplementary Table S2). These lower numbers compared with the local interpretation were consistent with the central-imaging interpretation.

DISCUSSION

The identification and inclusion of individuals with a specific disease profile in clinical trials or population/cohort studies are crucial to ensure accurate case selection and enhance the validity of results. This objective can be primarily achieved through a precise definition of the condition, especially in the absence of specific biomarkers. The AXIS study, therefore, contributes to ongoing efforts aimed at more clearly defining axial involvement in PsA by providing comprehensive data.

The results of our study underscore the variability in diagnosing axial involvement, depending on the diagnostic approach taken. Local investigators identified axial involvement in 37.6% of participants at the initial assessment and 27.3% at the final

assessment, which incorporated the central-imaging review, whereas the central clinical review provided a more conservative estimate and identified axial involvement in 16.9%. This variability highlights a critical insight of the study: local assessments, while sensitive and informative, may overestimate axial involvement compared with central expert evaluations. At the same time, the central clinical reviewers' evaluation might have underestimated the frequency of axial disease by assessing participants in a 'paper-based' way, focusing mainly on objective signs such as CRP, HLA-B*27, and imaging. Overall, this variability illustrates different scenarios for patient inclusion in clinical studies, including the impact of central-imaging evaluation.

Various definitions of axial involvement in PsA are heterogeneous and still evolving [4]. Although both PsA and axSpA fall under the broader SpA spectrum, the distinctive characteristics of the axial form of PsA result in differences that are not fully captured by the axSpA framework. In contrast to axSpA, where back pain is a *sine qua non* and is importantly associated with HLA-B*27 positivity, axial involvement in PsA shows a weaker association with HLA-B*27 [6,7]. On the other hand, a strong correlation has been identified between the presence of the HLA-B*27 allele and more severe PsA, with a higher frequency observed in those presenting with axial involvement [21–23]. In addition, previous studies have suggested that other HLA-B alleles, particularly HLA-B*08, may be associated with specific axial phenotypes. However, such genotype-phenotype relationships could not be evaluated in AXIS, as extended HLA genotyping was unavailable at the time. Our findings also demonstrated that HLA-B*27 positivity was less prevalent (13.9% in the entire cohort) than historically observed in axSpA [24], yet our findings suggest it still plays a role in the diagnosis of a subset of patients with axial involvement, particularly as an important determining factor in the assessment of central clinical reviewers. Furthermore, previous publications suggest that IBP is less prevalent in axial PsA, and the IBP definitions developed for axSpA may not perform as well in this form of PsA, further

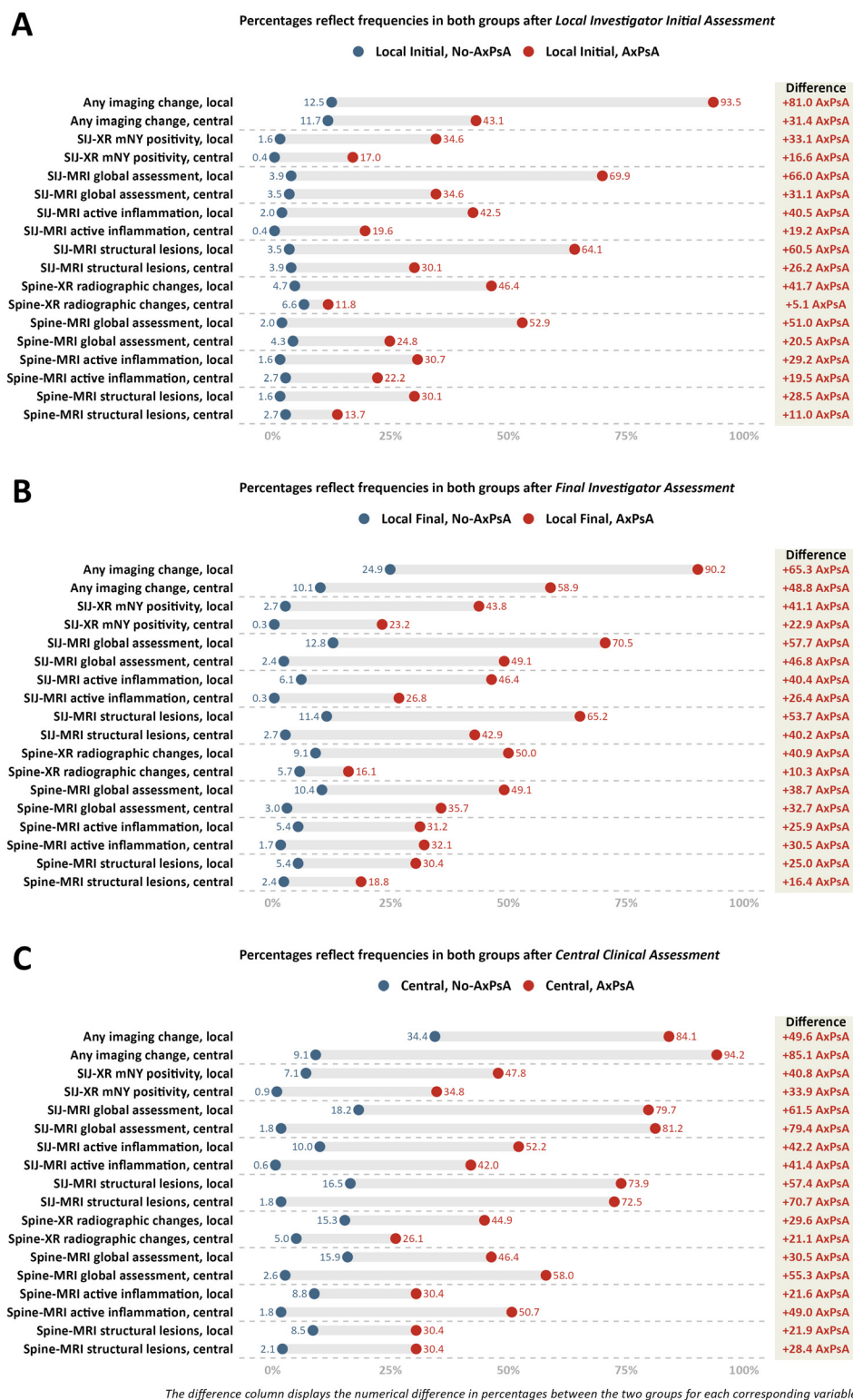


Figure 4. Dumbbell plot highlighting differences in imaging characteristics between patients with and without axial involvement: (A) at initial assessment by local investigator, (B) at final assessment by local investigator, and (C) at central clinical review. AxPsA, Axial Psoriatic Arthritis; mNY, modified New York Criteria; MRI, magnetic resonance imaging; SIJ, sacroiliac Joint; XR, X-ray.

complicating its identification [11,25]. The AXIS study showed that 25% to 30% of participants with axial involvement had no IBP according to the investigators' clinical judgement, which is close to the data reported for axSpA [26]. Of note, enthesitis was observed less frequently among participants with axial involvement by the central clinical review. This pattern was seen only with this definition of axial involvement, which relied

on recorded data and emphasised axial imaging findings, and may therefore have selected a more axial-dominant phenotype with relatively less peripheral enthesal disease. Therefore, this observation should be interpreted with caution.

The present findings underscore the important role of imaging in diagnosing axial involvement in PsA. The higher rates of axial involvement identified in local assessments suggest that

Table 4

Central-imaging readers' consensus assessment of spinal radiograph and MRI

Variable	Overall	Local investigator initial assessment of axial involvement			Local investigator final assessment of axial involvement			Central clinical review consensus categories of axial involvement		
	N = 409	Yes-Initial N = 153	No-Initial N = 256	P value	Yes-Final N = 112	No-Final N = 297	P value	Yes-Central N = 69	No-Central N = 340	P value
Spine radiograph indicative of axial involvement	35 (8.6)	18 (11.8)	17 (6.6)	.073	18 (16.1)	17 (5.7)	<.001	18 (26.1)	17 (5.0)	<.001
Erosion	0 (0.0)	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99
Squaring	3 (0.7)	3 (2.0)	0 (0.0)	.052	3 (2.7)	0 (0.0)	.020	3 (4.3)	0 (0.0)	.005
Sclerosis	1 (0.2)	1 (0.7)	0 (0.0)	.37	0 (0.0)	1 (0.3)	>.99	1 (1.4)	0 (0.0)	.17
Marginal syndesmophyte	25 (6.1)	11 (7.2)	14 (5.5)	.48	9 (8.0)	16 (5.4)	.32	9 (13.0)	16 (4.7)	.022
Endplate syndesmophyte	0 (0.0)	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99
Nonmarginal syndesmophyte	17 (4.2)	7 (4.6)	10 (3.9)	.74	5 (4.5)	12 (4.0)	.79	3 (4.3)	14 (4.1)	>.99
Ossification	25 (6.1)	6 (3.9)	19 (7.4)	.15	5 (4.5)	20 (6.7)	.39	3 (4.3)	22 (6.5)	.78
Paravertebral ossification	2 (0.5)	0 (0.0)	2 (0.8)	.53	0 (0.0)	2 (0.7)	>.99	0 (0.0)	2 (0.6)	>.99
Ankylosis	8 (2.0)	5 (3.3)	3 (1.2)	.16	5 (4.5)	3 (1.0)	.038	4 (5.8)	4 (1.2)	.031
Paravertebral ankylosis	2 (0.5)	0 (0.0)	2 (0.8)	.53	1 (0.9)	1 (0.3)	.47	1 (1.4)	1 (0.3)	.31
Endplate ankylosis	1 (0.2)	1 (0.7)	0 (0.0)	.37	1 (0.9)	0 (0.0)	.27	1 (1.4)	0 (0.0)	.17
Facet fusion	0 (0.0)	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99	0 (0.0)	0 (0.0)	>.99
Degenerative disc disease	75 (18.3)	30 (19.6)	45 (17.6)	.61	15 (13.4)	60 (20.2)	.11	5 (7.2)	70 (20.6)	.009
Spine-MRI indicative of axial involvement	49 (12.0)	38 (24.8)	11 (4.3)	<.001	40 (35.7)	9 (3.0)	<.001	40 (58.0)	9 (2.6)	<.001
Typical active lesions in spine-MRI	41 (10.0)	34 (22.2)	7 (2.7)	<.001	36 (32.1)	5 (1.7)	<.001	35 (50.7)	6 (1.8)	<.001
Any bone marrow oedema	83 (20.3)	47 (30.7)	36 (14.1)	<.001	43 (38.4)	40 (13.5)	<.001	30 (43.5)	53 (15.6)	<.001
Typical structural lesions in spine-MRI	28 (6.8)	21 (13.7)	7 (2.7)	<.001	21 (18.8)	7 (2.4)	<.001	21 (30.4)	7 (2.1)	<.001
Any fat lesion	60 (14.7)	36 (23.5)	24 (9.4)	<.001	28 (25.0)	32 (10.8)	<.001	23 (33.3)	37 (10.9)	<.001
Any erosion	3 (0.7)	3 (2.0)	0 (0.0)	.052	3 (2.7)	0 (0.0)	.020	3 (4.3)	0 (0.0)	.005
Any new bone formation (spurs, ankylosis, syndesmophyte)	13 (3.2)	8 (5.2)	5 (2.0)	.083	6 (5.4)	7 (2.4)	.20	6 (8.7)	7 (2.1)	.012

MRI, magnetic resonance imaging.

The variables are presented as number (%).

Typical acute/active inflammatory lesions in the spine are bone marrow oedema/osteitis involving vertebral bodies and/or posterior structures (facet joints, costo-vertebral, costotransversal joints, and spinal processes). Typical structural lesions in the spine refer to the clear presence of typical findings such as fat lesions in the bone marrow, erosions, and syndesmophytes (bone proliferation)/ankylosis.

radiographs and MRIs, although valuable, are prone to subjective interpretation—a phenomenon, which is also well known in axSpA. The central-imaging reviewers, composed of experienced radiologists and rheumatologists, likely provided a more conservative view of axial involvement in the absence of clinical information. Furthermore, the lack of an established definition for the spinal radiographic changes for axial involvement in PsA contributed to even more subjectivity in the interpretation of this imaging study. As a result, we noted a significant discrepancy between the assessments of local and central-imaging readers regarding spinal radiographs. The discrepancy regarding positive imaging findings in participants without axial involvement further emphasises the need for a standardised definition for axial involvement. Conversely, the AXIS study demonstrated that, despite imaging being integral to diagnosing axial involvement, nearly 10% of the participants diagnosed with axial involvement did not have any imaging evidence, suggesting that clinical assessment remains an important component of the diagnosis. Similarly, in 28% of the participants with negative central imaging, the investigator maintained the diagnosis of axial involvement. These observations underline the importance of clinical evaluation, particularly back pain and HLA-B*27 positivity, and they deserve detailed consideration in the future. MRI was emphasised in the AXIS as a critical tool for identifying active inflammation and structural damage. However, the challenge of accurately interpreting MRI findings—especially distinguishing inflammatory changes from mechanical or degenerative issues—remains. Recently, McGonagle et al [27] suggested that axial PsA may involve ligament-centric soft-tissue pathology, which can be called ‘ligamentitis’, unlike the bone-centric changes in axSpA. Further research is needed to confirm this theory and explore its mechanisms, but this theory might explain some discrepancies between the clinical judgement and findings of MRI, which might not be sensitive enough to detect soft-tissue inflammation in the absence of bone involvement. A detailed analysis of the images generated in AXIS might be able to shed some light on the imaging manifestations of PsA in the spine. In AXIS, central-imaging readers performed detailed radiographic and MRI assessments of both the spine and SIJs, including structural features beyond the mSASSS (modified Stoke Ankylosing Spondylitis Spinal Score) for spinal radiographs and detailed inflammatory and structural lesions in MRIs. Taken together, the central-imaging evaluations confirm that imaging remains a cornerstone for identifying axial involvement in PsA. At a more granular level, lesion-based scoring showed that SIJ and spinal MRI patterns were discriminative at the group level, especially when active and structural abnormalities were considered together, but this was also true for individual lesion levels at MRIs. In contrast, specific lesions on spinal radiographs, although numerically more frequent in participants with axial involvement, were not sufficiently discriminative when considered in isolation. These observations may suggest that proposed soft-tissue-dominated mechanisms of axial PsA require further dedicated imaging studies with targeted protocols/imaging modalities, and that, for now, axial disease in PsA is best approached through an overall integrated interpretation of radiographic and MRI findings rather than reliance on individual features.

This study’s strengths include its multicentre and multinational design, which provides a large, internationally generalisable PsA cohort assessed with a standardised clinical and imaging protocol. AXIS is the first study to combine systematic evaluation of 4 axial imaging modalities (radiographs and MRIs of the SIJs and spine) with staged local and central review,

enabling direct quantification of how the determination of axial involvement and the associated phenotype shift across diagnostic layers. Beyond quantifying variability across diagnostic layers, AXIS provides several concrete empirical insights that were previously unavailable. First, the study establishes benchmark frequencies of axial involvement under 3 clearly defined evaluation strategies, demonstrating that the estimated prevalence can differ by almost 2-fold depending on how clinical and imaging information are integrated. Second, AXIS identifies specific patterns driving reclassification, including cases with clinical features suggestive of axial disease but negative central imaging, as well as participants with imaging abnormalities interpreted as nonspecific in the absence of a convincing clinical context. Third, the systematic and parallel assessment of SIJs and spine confirms that a measurable subset of participants exhibits spinal imaging abnormalities in the absence of SI changes, a finding that has implications for future definition work and argues against restricting imaging assessment to a single anatomical region. Together, these observations move the field from heterogeneous prevalence estimates towards a structured understanding of how case definition strategies shape cohort composition and phenotype. The data further suggest that different entry strategies for clinical studies—such as reliance on local assessment alone vs mandatory central-imaging confirmation—are likely to yield populations with distinct clinical and imaging profiles. This may have implications for the interpretation and comparability of therapeutic trials targeting axial manifestations in PsA.

Although the AXIS study provides comprehensive data using a standardised international protocol, several limitations should be considered. First, the cross-sectional design precludes conclusions regarding the temporal evolution of axial involvement, the progression of imaging findings, or the stability of diagnostic classification over time. AXIS was conceived as a descriptive foundation for definition development; longitudinal validation will be required to determine prognostic relevance and reproducibility of the proposed diagnostic constructs. Second, although central-imaging review enhances standardisation, it is performed without direct clinical context, whereas the central clinical review is necessarily ‘paper-based’. Both approaches may weigh objective findings differently than bedside assessment by the treating physician. Conversely, local investigators integrate clinical judgement with real-time patient interaction but may be influenced by pre-existing diagnostic impressions. These structural differences between evaluation layers are intrinsic to the study design and likely contribute to the observed shifts across assessments. Third, in the absence of a validated reference standard for axial involvement in PsA, no assessment strategy can be considered a formal gold standard. The final local investigator assessment was used as a pragmatic midpoint integrating clinical and centrally reviewed imaging information. However, several features evaluated in the study—such as HLA-B*27 status, IBP, and imaging abnormalities—are also elements considered during diagnostic judgement. This overlap introduces the possibility of circular reasoning and limits the ability to assess independent diagnostic performance. Fourth, imaging interpretation remains challenging. Despite standardised protocols and expert central reading, MRI findings—particularly BMO and structural changes at the SIJs and spine—may be influenced by mechanical or degenerative changes. Pregnancy history was not systematically collected, which may be relevant when interpreting SI MRI findings in women [28,29]. Furthermore, although central reading reduced variability, discrepancies between local and central-imaging

interpretations highlight the inherent subjectivity of current imaging definitions, particularly for spinal radiographic changes, where no consensus criteria specific to axial PsA exist. Fifth, the cohort was restricted to patients with musculoskeletal symptom duration ≤ 10 years and without prior exposure to biologic or targeted synthetic DMARDs. Although this design minimises treatment-related confounding and reflects an earlier disease spectrum, it may limit generalisability to long-standing or previously treated populations, in whom structural damage patterns and phenotype distribution may differ. Finally, although AXIS systematically evaluated both SIJs and spine using 4 imaging modalities, the study was not designed to establish diagnostic cut-offs or validate specific lesion-based scoring systems. The findings, therefore, inform, but do not by themselves define, a consensus definition of axial involvement.

In conclusion, the AXIS study provides an important contribution toward improving the understanding of axial disease in PsA, whereas acknowledging that important challenges remain in fully characterising the spectrum of axial involvement. Presence of back pain (including IBP), HLA-B*27 positivity, higher levels of CRP, and the presence of active inflammatory and structural changes in the SIJ and spine were associated with the final assessments for the presence of axial involvement, both by the local investigator and the central clinical reviewers, reflecting current understanding of axial PsA, likely influenced by existing knowledge from conventional axSpA. This is supported by the staged evaluations, which highlight that the determination of axial involvement and associated phenotype varies depending on how clinical information and imaging are weighted, reinforcing the need for an explicit and standardised definition. Although the direction of these shifts across evaluation settings is anticipated, AXIS offers a standardised framework for quantification and delineates specific clinical-imaging combinations.

As efforts continue to refine the definition of axial involvement in PsA, the findings from the AXIS study will be instrumental in shaping future diagnostic frameworks and treatment strategies. A logical subsequent step, which is already underway, is to translate these findings into a consensus definition and a unified nomenclature for axial involvement in PsA. Concurrently, these findings define a clear research agenda, including the development of explicit rules for integrating clinical and imaging data, more rigorous evaluation of imaging specificity, and longitudinal validation after a consensus definition has been established. The objective of this endeavour is to enable the identification of a homogeneous subgroup for research and clinical trials. Ultimately, these advances will help improve outcomes for patients with this challenging form of PsA. Going forward, further research utilising advanced imaging techniques and high-throughput molecular technologies is needed to comprehensively evaluate the full spectrum of axial involvement in PsA.

CRediT authorship contribution statement

Murat Torgutalp: Writing – review & editing, Writing – original draft, Visualization, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Raquel Almodovar:** Data curation. **Valderio F Azevedo:** Data curation. **Xenofon Baraliakos:** Methodology, Investigation, Data curation, Conceptualization. **Filip Van den Bosch:** Writing – review & editing, Methodology, Investigation, Data curation, Conceptualization. **Jürgen Braun:** Methodology, Conceptualization. **Vinod Chandran:** Writing –

review & editing, Methodology, Conceptualization. **Laura C Coates:** Writing – review & editing, Methodology, Conceptualization. **Atul Deodhar:** Writing – review & editing, Data curation. **Torsten Diekhoff:** Methodology, Data curation, Conceptualization. **Filippo Fagni:** Writing – review & editing, Data curation. **Alberto Floris:** Data curation. **Floris A van Gaalen:** Methodology, Data curation, Conceptualization. **Rodrigo Garcia-Salinas:** Data curation. **Lianne S Gensler:** Methodology, Data curation, Conceptualization. **Niti Goel:** Writing – review & editing, Methodology, Conceptualization. **Alice B Gottlieb:** Conceptualization. **Désirée van der Heijde:** Methodology, Conceptualization. **Philip S Helliwell:** Methodology, Conceptualization. **Kay Geert A Hermann:** Methodology, Data curation, Conceptualization. **Umut Kalyoncu:** Data curation. **Uta Kiltz:** Data curation. **Francis Kynaston-Pearson:** Data curation. **Robert GW Lambert:** Methodology, Data curation, Conceptualization. **Ramasharan Laxminarayan:** Data curation. **Ying Ying Leung:** Data curation. **Maria Llop:** Data curation. **Clementina López-Medina:** Data curation. **Alejandra López-Rodríguez:** Data curation. **Michele M Luchetti Gentiloni:** Data curation. **Miranda van Lunteren:** Data curation. **Marina Magrey:** Data curation. **Ajesh B Maharaj:** Data curation. **Hernán Maldonado-Ficco:** Data curation. **Walter P Maksymowych:** Methodology, Data curation, Conceptualization. **Helena Marzo-Ortega:** Data curation. **Marco Massarotti:** Data curation. **Ashish J Mathew:** Data curation. **Philip Mease:** Methodology, Data curation, Conceptualization. **Peter Nash:** Methodology, Conceptualization. **Victoria Navarro-Compán:** Data curation. **Mikkel Østergaard:** Methodology, Data curation, Conceptualization. **Fabian Proft:** Methodology, Data curation, Conceptualization. **Mikhail Protopopov:** Methodology, Conceptualization. **Roberto Ranza:** Data curation. **Sherry Rohekar:** Data curation. **Carlo Salvarani:** Data curation. **Ruxandra E Schiotis:** Data curation. **Nicholas Shenker:** Data curation. **Joachim Sieper:** Methodology, Conceptualization. **Dilek Solmaz:** Data curation. **Enrique R Soriano:** Data curation. **Lai-Shan Tam:** Data curation. **Ricardo Acayaba de Toledo:** Data curation. **James CC Wei:** Data curation. **Nelly Ziade:** Data curation. **Dafna D Gladman:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Denis Poddubnyy:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization.

Acknowledgements

We would like to express our heartfelt gratitude to Deepak Jadon for his invaluable contributions to this study. His dedication and expertise greatly enriched our work, and his passing during the recruitment period was a profound loss to the scientific community and to us personally. We honour his memory and the impact he made in the field.

We thank all investigators, site personnel, and patients participating in the study. We gratefully acknowledge the team of CARE Arthritis for their valuable technical support in developing the eCRF and management of imaging data in the collaboration of this research. The views expressed are those of the authors and not necessarily those of the (UK) National Health Service (NHS), the NIHR, or the (UK) Department of Health. Preliminary findings from this study were delivered as an invited lecture at EULAR 2024 and subsequently presented at ACR 2024 (Abstract #2234).

Contributors

XB, FvdB, JB, VC, LCC, TD, FAvG, LSG, NG, ABG, DvdH, PSH, KGAH, RGWL, WPM, PM, PN, FP, MP, JS, MT, DDG, and DP conceptualised the study. MT performed statistical analyses. XB, WPM, MØ, TD, KGAH, and RGWL performed imaging data interpretation. FvdB, DDG, PM, and DP performed central clinical review. MT, DP, and DDG wrote the first draft of the manuscript. DP and DDG are guarantors. All authors curated the data, contributed to data interpretation, critically reviewed the manuscript for important intellectual content, and approved the final version.

Funding

This study is conducted under the umbrella of ASAS and GRAPPA by unrestricted research grants from AbbVie, Galapagos BV/Alfasigma S.p.A, Johnson & Johnson, Eli Lilly, Novartis, Pfizer, and UCB.

Competing interests

MT has received research support from UCB and Janssen. RA has received consulting and/or speaker fees from Almirall, Amgen, Bristol Myers Squibb, Galápagos, Gebro, Johnson & Johnson, Lilly, MSD, Nordic, Novartis, Pfizer, and UCB. VFA has received honoraria or travel support from Organon, Roche, Abbott, Fresenius Kabi, Sandoz, and Celltrion; honoraria from Organon, Celltrion, AstraZeneca, Amgen, Fresenius Kabi; support attending to meetings from Organon and Fresenius Kabi; and has participated on a Data Safety Monitoring Board or Advisory Board of Fresenius Kabi, Organon, and Abbott. XB is a consultant and member of the Scientific Advisory Board of AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Cestas, Celltrion, Clarivate, Galapagos, Greywolf, J&J, Lilly, Moonlake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB, and Zuellig and has received research grants from: AbbVie, Celltrion, Janssen, Moonlake, Novartis. Non-commercial disclosures: ASAS Past President, EULAR President. FvdB has received consulting fees from AbbVie, Alfasigma, Celltrion, Eli Lilly, Fresenius Kabi, Greywolf Therapeutics, Janssen, Novartis, UCB, and Xencor. VC is supported by the Dr. Dafna D. Gladman Chair in Psoriatic Arthritis Research, a joint Hospital-University Named Chair between the University of Toronto, the University Health Network, and the UHN Foundation. VC has received research grants from AbbVie, Amgen, and Eli Lilly and has received honoraria for advisory board member roles from AbbVie, BMS, Eli Lilly, Fresenius Kabi, Janssen, Novartis, and UCB. His spouse is an employee of AstraZeneca. LCC is supported by the Oxford NIHR BRC. LCC has received grants/research support from AbbVie, Amgen, Janssen, and UCB; worked as a paid consultant for AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Enlivex, Janssen, Moonlake, Novartis, Pfizer, Takeda, and UCB; and has been paid as a speaker for AbbVie, Amgen, Eli Lilly, Janssen, Novartis, Pfizer, and UCB. AD has received consulting and/or Research Grants from BMS, Eli Lilly, J&J, MoonLake, Novartis, Pfizer, and UCB. TD has received speaker honoraria from Novartis, MSD, UCB, Janssen, Eli Lilly, Canon MS, Berlinflame, Bracco, and Advisory Board: Eli Lilly, AbbVie, and UCB. FF has received speaker honoraria, consulting fees, or research support from Novartis, AbbVie, UCB, J&J, and AstraZeneca. AF has received speaker or advisory board honoraria from AbbVie, Eli Lilly, and UCB. FAvG has received grants and/or consulting fees from, AbbVie, ASAS, BMS, Galapagos, Janssen, Lilly, Novartis, Pfizer, and UCB (all paid to the LUMC). RG-S has received consultancy/speaker/research grants from AbbVie, BMS, Janssen, Eli Lilly,

Novartis, Pfizer, Roche, UCB, GSK, Biogen, Amgen, Raffo, and Adium. LSG has received consultancy/research grants from Ace-lyrin, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB. NG is a minority shareholder in Abcuro and UCB. ABG has received research/educational grants from Avalo Therapeutics, Bristol Myers Squibb, Janssen, Moonlake, and UCB (all paid to Mount Sinai School of Medicine), and honoraria as an advisory board member and consultant for Amgen, Eli Lilly, Highlights Therapeutics, Janssen, Novartis, Sanofi, SunPharma, Takeda, Teva, UCB, and Xbiotech (stock options for RA). DvdH has received consulting fees from AbbVie, Alfasigma, ArgenX, BMS, Eli Lilly, Greywolf Therapeutics, Janssen, Novartis, Pfizer, Takeda, and UCB Pharma. Associate editor *Annals of the Rheumatic Diseases*, editorial board member *Journal of Rheumatology*, and director of *Imaging Rheumatology* bv. PSH has received a consulting fee from Amgen, and a speaker fee from Novartis and Janssen. KGAH has received consulting fees from AbbVie, Lecture fees from Novartis, Pfizer, and UCB. PSH is a Cofounder of BerlinFlame GmbH. UK has received consulting fees AbbVie, Novartis, Pfizer, and UCB. UK has received grant and a research support and consultancy fees from AbbVie, Amgen, Biocad, Biogen, Chugai, Eli Lilly, Fresenius, Gilead, Grünenthal, GSK, Janssen, MSD, Novartis, Pfizer, Roche, and UCB. FK-P has received support for attending meetings and/or travel from Novartis, AbbVie, Eli Lilly, and Pfizer. RGWL has received consulting fees from AbbVie and CARE Arthritis. RL has received a speaker fee from Novartis. YYL is supported by the Singapore National Medical Council. YYL has received a speaker fee from AbbVie, DKSH, Janssen, Novartis, and Pfizer. ML has received consulting fees and/or speaker fee from AbbVie, Novartis, Pfizer, Eli Lilly, Janssen, Amgen, and UCB. CL-M has received speaker honoraria, consulting fees, or research support from AbbVie, Eli Lilly, Janssen, MSD, Novartis, and UCB. AL-R has received research support from BMS and Novartis; consulting fees from Janssen, Novartis, Eli Lilly, and GSK; and speaker fees from Novartis, Eli Lilly, Janssen, BMS, and GSK. MML-G has received research support from AbbVie; consulting fees from AbbVie, Janssen, Novartis, and Pfizer; and speaker fees from AbbVie, Amgen, Eli Lilly, Janssen, Novartis, and Pfizer. Marina Magrey has received consulting fees from Novartis, UCB, Janssen, Pfizer, and AbbVie. SPARTAN Board member, ABM has received grant support and/or consulting fees and or speaker fees from AbbVie, Amgen, Novartis, Roche Pharmaceuticals, Eli Lilly, Janssen, Pfizer, AstraZeneca, and Servier Laboratories. HM-F has received consulting and/or speaker fees from AbbVie, BMS, Celgene, Eli Lilly, Janssen, Novartis, Pfizer, and Roche. WPM has received grant/research support from AbbVie, Novartis, Pfizer, and UCB Pharma; consulting fees, speaking fees, and/or honoraria fees from AbbVie, BMS, Celgene, Galapagos, Janssen, Eli Lilly, Medscape, Novartis, Peervoice, Pfizer, and UCB Pharma; is Chief Medical Officer of CARE Arthritis Limited; Editorial Board member of *Journal of Rheumatology*, *RMD Open*, *Clinical and Experimental Rheumatology*; and has received royalties or licences from the University of British Columbia for the 14-3-3eta diagnostic biomarker. HM-O is supported by the National Institute for Health Research (NIHR) Leeds Biomedical Research Centre (BRC). HM-O has received grant support from Janssen, Novartis, Pfizer, and UCB. HM-O has received honoraria and/or speaker fees from AbbVie, Amgen, Biogen, Eli Lilly, Janssen, Moonlake, Novartis, Pfizer, Takeda, and UCB. Marco Massarotti has received consulting/speaker fees and/or support for attending meetings from AbbVie, Gilead, UCB, and Novartis. AJM has received grant support from Novartis, honoraria, and/or speaker fees from CIPLA, Janssen and Abbott. PM has received grant support, consulting, and/or speaker fees from AbbVie, Amgen, ArgenX, BMS, Century, Cullinan, Eli Lilly, Johnson &

Johnson, Merck, Moonlake Pharma, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB. PN has received grant support, consulting, payment or honoraria, and/or speaker fees from AbbVie, Amgen, BMS, Lilly, Janssen, UCB, Servatus, Novartis, and Arrowtex. He serves as a member of the steering committee of GRAPPA and IMID Forum. VN-C has received research grants/honoraria from AbbVie, Alfasigma, Fresenius Kabi, Galapagos, Eli Lilly, Janssen, Novartis, Pfizer, UCB, and ASAS. VN-C is a member of the ASAS Executive Committee and currently ASAS President. VN-C is an associate editor of *Seminars Arthritis and Rheumatism*. MØ has received research grants from AbbVie, Amgen, BMS, Merck, Celgene, Eli Lilly, Novartis, and UCB; speaker fees from AbbVie, BMS, Boehringer Ingelheim, Celgene, Eli Lilly, Galapagos, Gilead, Hospira, Janssen, MEDAC, Merck, Novartis, Novo, Orion, Pfizer, Regeneron, Roche, Sandoz, Sanofi, and UCB; and consultancy fees from AbbVie, BMS, Boehringer Ingelheim, Celgene, Eli Lilly, Galapagos, Gilead, Hospira, Janssen, MEDAC, Merck, Novartis, Novo, Orion, Pfizer, Regeneron, Roche, Sandoz, Sanofi, and UCB. FP has received grants and personal fees from Novartis, Eli Lilly, and UCB and personal fees from AbbVie, AMGEN, BMS, Celgene, Janssen, Hexal, Medscape, Moonlake, MSD, Pfizer, and Roche outside the presented work. MP has received consulting/speaker fees from Janssen and Novartis; and support for attending meetings and/or travel from Novartis, AbbVie, Janssen, and UCB. RR has received grant support and/or consulting and/or speaker fees from AbbVie, BMS, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB. SR has received honoraria and/or participated in advisory boards from AbbVie, Amgen, BioJAMP, BMS, Celgene, Celltrion, Eli Lilly, Fresenius Kabi, Gilead, Janssen, Merck, Novartis, Organon, Pfizer, Roche, Sandoz, UCB, and Viatrix and a research grant from UCB. RES has received speaker honoraria from AbbVie, MSD, Novartis, and Pfizer. NS is supported by the Cambridge NIHR BRC. NS has received grant support from Gilead, GRAPPA, GSK, UCB, Kirin, AbbVie, MRC, Versus Arthritis, APDP, and Addenbrooke's Charitable Trust. NS has received speaker/conference fees from Eli Lilly and Roche. JS has received honoraria and/or speaker fees from Merck and Novartis. DS has received speaker honoraria, consulting fees, or research support from AbbVie, Novartis, and UCB. ERS has received grant support and/or consulting and/or speaker fees and/or travelling support from AbbVie, Amgen, AstraZeneca, Elea, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, and UCB. L-ST has received grant support and/or consulting and/or speaker fees and/or travelling support from AbbVie, Amgen, AstraZeneca, BMS, Eli Lilly, Euroimmun, Fresenius Kabi, Janssen, Novartis, and Pfizer. RADT has received speaker fees from AbbVie, Novartis, Janssen, UCB, and GSK. JCCW has received grant support from Pfizer, Abbott, AbbVie, Bristol Myers Squibb, Eli Lilly, JNJ, UCB, MSD, GSK, Chugai, Roche, Celgene, Sanofi-Aventis, and Novartis and has received honoraria and/or speaker fees from Pfizer, AbbVie, Eli Lilly, JNJ, UCB, MSD, GSK, Chugai, and Novartis. NZ has received speaker fees (Roche, AbbVie, Eli Lilly, Pfizer, Janssen, Novartis, Pierre Fabre, Apotex, Pharmaline, and Sandoz), internal workshops (AbbVie, Eli Lilly, Sanofi-Aventis, Pfizer, and Janssen), advisory boards (Roche, AbbVie, Eli Lilly, Pfizer, Janssen, Novartis, Gilead, and NewBridge), and research grants (AbbVie, NewBridge, Pfizer, and Algorithm/Celgene). DDG has received grant support and/or consulting fees from AbbVie, Amgen, BMS, Eli Lilly, Fresenius Kabi, Johnson & Johnson, Novartis, Pfizer, and UCB. DDG is a member of the Steering Committee of GRAPPA. DP has received research support from AbbVie, Eli Lilly, Janssen, Novartis, Pfizer, and UCB; consulting fees from AbbVie, Eli Lilly, Greywolf Therapeutics, Janssen, Merck, Moonlake, Novartis, Pfizer, and UCB; and speaker fees from AbbVie, Canon, Eli Lilly, Janssen, Medscape, Novartis, Peervoice, Pfizer, and UCB. He

serves as a member of the executive committee of ASAS and a member of the steering committee of GRAPPA. All other authors declare they have no competing interests.

Patient consent for publication

Not applicable.

Ethics approval

The study was performed according to the ethical principles of the Declaration of Helsinki and International Council for Harmonisation Good Clinical Practice guidelines. The ethics committee of the coordinating centre of the study, located at the Charité-Universitätsmedizin Berlin, Germany, approved the conduction of the study (the approval number: EA4/021/21). Thereafter, the study protocol was approved by the individual Independent Ethics Committee (IEC)/Institutional Review Board (IRB) of each participating countries/centres.

Provenance and peer review

Not commissioned; externally peer reviewed.

Patient and public involvement

Patient research partner (NG) has been involved in the design of the study, contributed to data interpretation, and critically reviewed the manuscript for important intellectual content.

Data availability statement

Data are available on reasonable request. The ethics committee limits the sharing of patient-level data. Summary data may be shared upon reasonable request to the corresponding author.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the first author used DeepL, DeepL-Write, and Grammarly to improve clarity and grammar of the text. After using this tool/service, the author reviewed and edited the content as needed and takes full responsibility for the content of the publication.

ORCID iD

Murat Torgutalp <http://orcid.org/0000-0003-4600-9484>

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at [doi:10.1016/j.ard.2026.02.025](https://doi.org/10.1016/j.ard.2026.02.025).

Orcid

Murat Torgutalp: <http://orcid.org/0000-0003-4600-9484>

REFERENCES

- [1] Ritchlin CT, Colbert RA, Gladman DD. Psoriatic arthritis. *N Engl J Med* 2017;376(10):957–70.
- [2] Alinaghi F, Calov M, Kristensen LE, Gladman DD, Coates LC, Jullien D, et al. Prevalence of psoriatic arthritis in patients with psoriasis: a systematic

- review and meta-analysis of observational and clinical studies. *J Am Acad Dermatol* 2019;80(1):251–65 e19.
- [3] Feld J, Chandran V, Haroon N, Inman R, Gladman D. Axial disease in psoriatic arthritis and ankylosing spondylitis: a critical comparison. *Nat Rev Rheumatol* 2018;14(6):363–71.
 - [4] Poddubnyy D, Jadon DR, Van den Bosch F, Mease PJ, Gladman DD. Axial involvement in psoriatic arthritis: An update for rheumatologists. *Semin Arthritis Rheum* 2021;51(4):880–7.
 - [5] Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK. Psoriatic arthritis (PSA)—an analysis of 220 patients. *Q J Med* 1987;62(238):127–41.
 - [6] Jadon DR, Sengupta R, Nightingale A, Lindsay M, Korendowych E, Robinson G, et al. Axial Disease in Psoriatic Arthritis study: defining the clinical and radiographic phenotype of psoriatic spondyloarthritis. *Ann Rheum Dis* 2017;76(4):701–7.
 - [7] Feld J, Ye JY, Chandran V, Inman RD, Haroon N, Cook R, et al. Is axial psoriatic arthritis distinct from ankylosing spondylitis with and without concomitant psoriasis? *Rheumatology (Oxford)* 2020;59(6):1340–6.
 - [8] Regierer AC, Weiß A, Proft F, Baraliakos X, Behrens F, Poddubnyy D, et al. Comparison of patients with axial PsA and patients with axSpA and concomitant psoriasis: an analysis of the German register RABBIT-SpA. *RMD Open* 2023;9(1):e002837.
 - [9] Gentiloni MML, Paci V, Cimaroli I, Agostinelli A, Giannoni M, Campanati A, et al. The ATTRACT study: screening for the early identification of axial psoriatic arthritis in a cohort of Italian psoriatic patients. *Rheumatology (Oxford)* 2023;63(8):2152–61.
 - [10] Mease PJ, Palmer JB, Liu M, Kavanaugh A, Pandurengan R, Ritchlin CT, et al. Influence of axial involvement on clinical characteristics of psoriatic arthritis: analysis from the Corrona Psoriatic Arthritis/Spondyloarthritis Registry. *J Rheumatol* 2018;45(10):1389–96.
 - [11] Yap KS, Ye JY, Li S, Gladman DD, Chandran V. Back pain in psoriatic arthritis: defining prevalence, characteristics and performance of inflammatory back pain criteria in psoriatic arthritis. *Ann Rheum Dis* 2018;77(11):1573–7.
 - [12] Chandran V, Bull SB, Pellett FJ, Ayeart R, Rahman P, Gladman DD. Human leukocyte antigen alleles and susceptibility to psoriatic arthritis. *Human immunol* 2013;74(10):1333–8.
 - [13] Baraliakos X, Gossec L, Pournara E, Jeka S, Mera-Varela A, D'Angelo S, et al. Secukinumab in patients with psoriatic arthritis and axial manifestations: results from the double-blind, randomised, phase 3 MAXIMISE trial. *Ann Rheum Dis* 2021;80(5):582–90.
 - [14] Helliwell PS, Gladman DD, Chakravarty SD, Kafka S, Karyekar CS, You Y, et al. Effects of ustekinumab on spondylitis-associated endpoints in TNFi-naïve active psoriatic arthritis patients with physician-reported spondylitis: pooled results from two phase 3, randomised, controlled trials. *RMD Open* 2020;6(1):e001149.
 - [15] Mease PJ, Helliwell PS, Gladman DD, Poddubnyy D, Baraliakos X, Chakravarty SD, et al. Efficacy of guselkumab on axial involvement in patients with active psoriatic arthritis and sacroiliitis: a post-hoc analysis of the phase 3 DISCOVER-1 and DISCOVER-2 studies. *Lancet Rheumatol* 2021;3(10):e715–e23.
 - [16] Poddubnyy D, Baraliakos X, Van den Bosch F, Braun J, Coates LC, Chandran V, et al. Axial Involvement in Psoriatic Arthritis cohort (AXIS): the protocol of a joint project of the Assessment of SpondyloArthritis international Society (ASAS) and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA). *Ther Adv Musculoskelet Dis* 2021;13:1759720x211057975.
 - [17] Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H, et al. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum* 2006;54(8):2665–73.
 - [18] van der Linden S, Valkenburg HA, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis. A proposal for modification of the New York criteria. *Arthritis Rheum* 1984;27(4):361–8.
 - [19] Team RC. R: A language and environment for statistical computing. R Foundation for Statistical Computing. (No Title) 2013. <https://www.r-project.org/>. Accessed March 23, 2026.
 - [20] Sjoberg DD, Whiting K, Curry M, Lavery JA, Larmarange J. Reproducible summary tables with the gtsummary package. *The R Journal* 2021;13(1):570–80.
 - [21] Chandran V, Tuluso DC, Cook RJ, Gladman DD. Risk factors for axial inflammatory arthritis in patients with psoriatic arthritis. *J Rheumatol* 2010;37(4):809–15.
 - [22] Queiro R, Morante I, Cabezas I, Acasuso B. HLA-B27 and psoriatic disease: a modern view of an old relationship. *Rheumatology (Oxford)* 2016;55(2):221–9.
 - [23] Coates LC, Baraliakos X, Blanco FJ, Blanco-Morales EA, Braun J, Chandran V, et al. The phenotype of axial spondyloarthritis: is it dependent on HLA-B27 status? *Arthritis Care Res (Hoboken)* 2021;73(6):856–60.
 - [24] Navarro-Compán V, Sepriano A, Capelusnik D, Baraliakos X. Axial spondyloarthritis. *Lancet* 2025;405(10473):159–72.
 - [25] Haroon M, Gallagher P, FitzGerald O. Inflammatory back pain criteria perform well in subset of patients with active axial psoriatic arthritis but not among patients with established axial disease. *Ann Rheum Dis* 2019;78(7):1003–4.
 - [26] Poddubnyy D, Callhoff J, Spiller I, Listing J, Braun J, Sieper J, et al. Diagnostic accuracy of inflammatory back pain for axial spondyloarthritis in rheumatological care. *RMD Open* 2018;4(2):e000825.
 - [27] McGonagle D, David P, Macleod T, Watad A. Predominant ligament-centric soft-tissue involvement differentiates axial psoriatic arthritis from ankylosing spondylitis. *Nat Rev Rheumatol* 2023;19(12):818–27.
 - [28] de Winter J, de Hooge M, van de Sande M, de Jong H, van Hoven L, de Koning A, et al. Magnetic resonance imaging of the sacroiliac joints indicating sacroiliitis according to the assessment of spondyloarthritis international society definition in healthy individuals, runners, and women with postpartum back pain. *Arthritis Rheumatol* 2018;70(7):1042–8.
 - [29] Renson T, Depicker A, De Craemer AS, Deroo L, Varkas G, de Hooge M, et al. High prevalence of spondyloarthritis-like MRI lesions in postpartum women: a prospective analysis in relation to maternal, child and birth characteristics. *Ann Rheum Dis* 2020;79(7):929–34.