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MUSCULOSKELETAL

Prevalence of inflammatory posterior arch abnormalities on lumbar spine MRI in spondyloarthritis patients compared with low back pain patients

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Abstract

Objectives This study was conducted in order to compare the prevalence of inflammatory posterior arch abnormalities on lumbar spine MRI between axial spondyloarthritis (axSpA) patients and low back pain (LBP) patients.

Methods *Patients*—axSpA patients meeting the 2009 ASAS criteria and chronic LBP patients who had a lumbar spine MRI were selected. *MRI*—STIR and T1 sagittal images up to T8–T9 were reviewed by two experienced rheumatologists blinded to the diagnosis and clinical data to identify inflammatory posterior arch abnormalities. *Analyses*—The prevalence of inflammatory posterior arch abnormalities between axSpA and LBP patients was compared. Clinical data were compared in the axSpA group depending on whether or not inflammatory posterior arch abnormalities were present.

Results Ninety-five patients were enrolled in each group. The prevalence of all inflammatory posterior arch abnormalities was the same in the axSpA and LBP groups (58% in the SpA group versus 70% in the LBP group, $p = 0.1$). However, differences in terms of the prevalence of costotransverse joint arthritis, pedicle oedema above L3 and transverse and spinous process oedema were observed between the two groups (axSpA 27% versus LBP 6%, $p = 0.0004$). Patients with inflammatory posterior arch abnormalities in the axSpA group had a longer disease duration (11 versus 8 years, $p = 0.02$), higher CRP levels (median 11 versus 3 mg/l, $p = 0.0002$) and higher prevalence of radiographic sacroiliitis (84 versus 47%, $p = 0.001$) compared to patients without inflammatory posterior arch abnormalities.

Conclusions Costotransverse arthritis, pedicle oedema and transverse process oedema are more frequent in axSpA patients than LBP patients, on lumbar spine MRI depicting TH9-S1.

Key Points

- *MRI pedicle oedema above L3, transverse process oedema, spinous process oedema or costotransverse arthritis is more frequently observed in axial spondyloarthritis (SpA).*
- *SpA patients with at least one MRI inflammatory lesion on the posterior arch had higher clinical activity scores and biological inflammation.*
- *Facet joint arthritis was more common in patients with chronic low back pain.*

Keywords Spondylitis · Ankylosing · Magnetic resonance imaging · Diagnosis · Differential

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Abbreviations

ASAS	Assessment of Spondyloarthritis International Society
ASDAS	Ankylosing Spondylitis Disease Activity Score
axSpA	Axial spondyloarthritis
BASDAI	Bath Ankylosing Spondylitis Disease Activity Index
BASFI	Bath Ankylosing Spondylitis Functional Index
bDMARD	Biologic disease-modifying antirheumatic drugs
CIL	Vertebral corner inflammatory lesions
CRP	C-reactive protein
CTA	Costotransverse joint arthritis
csDMARD	Conventional synthetic disease-modifying antirheumatic drugs
ESR	Erythrocyte sedimentation rate
FJA	Facet joint arthropathy
IAAA	Inflammatory anterior arch abnormalities
IBD	Inflammatory bowel disease
IPAA	Inflammatory posterior arch abnormality
IQR	Interquartile range
ISE	Interspinous oedema
LBP	Low back pain
MRI	Magnetic resonance imaging
NSAID	Non-steroidal anti-inflammatory drug
PE	Pedicle oedema
SD	Standard deviation
SPE	Spinous process oedema
STIR	Short-TI inversion recovery sequence
TNF	Tumour necrosis factor
TPE	Transverse process oedema
VAS	Visual analogue scale
VFD	Vertebral fat deposition

Axial spondyloarthritis (axSpA) is a chronic inflammatory rheumatic disease affecting the entheses. It causes inflammation-related pain in the spine and sacroiliac joints [1]. The condition is usually diagnosed by rheumatologists and based on several features including clinical, biological and imaging findings and particularly radiographic or MRI sacroiliitis. Inflammatory MRI lesions precede visible structural changes on radiographs [2, 3]. However, the specificity of inflammatory lesions seen on sacroiliac MRI scans has been criticised due to the high prevalence of abnormal sacroiliac findings when MRI is performed on healthy adults [4–7].

The diagnostic value of spinal MRI for axSpA is the subject of ongoing debate. The presence of at least three vertebral corner inflammatory lesions (CIL) is suggestive of axSpA with 45% sensitivity and 88% specificity [8–10]. Sensitivity is 33% and specificity 97% in patients under 50 years of age [11]. When vertebral fat deposition (VFD) is found in at least six locations, the sensitivity and sensitivity for the axSpA diagnosis are 22 and 98%, respectively [12]. Fifteen to 50% of axSpA patients have abnormal vertebral corner signals on spinal MRI scans with no changes in their sacroiliac joints, while 20% of patients with low back pain (LBP) also display these changes on their spinal MRI images [13–15]. Ez-Zaitouni et al [16] concluded that spinal MRI had little diagnostic value for axSpA based on the presence of inflammatory lesions on spinal MRI in the SPACE and DESIR cohorts.

Few studies have focused on inflammatory lesions in the posterior arches of the spine. Their estimated prevalence is 26–29% [8, 12, 17] in axSpA and 12% [13] in degenerative disc disease. However, to date, no study has compared the prevalence of these lesions in axSpA and LBP patients. Given the lack of published evidence, the Assessment of Spondyloarthritis International Society (ASAS) discounted the presence of inflammatory posterior arch abnormalities (IPAA) as a possible contributor to the diagnosis of positive spinal MRI in axSpA [18].

The aims of our study were to determine the prevalence of IPAA on the lumbar spine MRIs of patients with axSpA compared to those of LBP patients and to investigate the relationships between these lesions and the clinical features of axSpA.

Patients and methods

Study design

This was a case-controlled study performed at a single hospital.

Patients

Patients assigned to the axSpA group were selected retrospectively on the basis of electronic medical records. In order to be identified, patients had to meet the ASAS 2009 criteria for axSpA [2]. They were not taking biological disease-modifying antirheumatic drugs (bDMARDs) or had stopped these treatments at least 3 months earlier. They had undergone thoracic and lumbar spine MRI with sagittal T1 and T2 STIR sequences between 2009 and June 2016. Their clinical data were available within 2 months of the MRI examination. Patients with a medical history of vertebral fractures based on medical reports were excluded. Patients assigned to the LBP group were included prospectively during their visit to our radiology unit for a lumbar spine MRI due to common sciatica or low back pain between September 2015 and January 2017. The radiologist or rheumatologist asked several questions. Patients were excluded if they were < 18 years of age, had potential secondary back pain (e.g. infection, tumour, inflammatory disease, fracture or any features that could lead to potential diagnosis of axSpA such as uveitis, psoriasis, inflammatory bowel disease, peripheral arthritis or enthesitis,

a family history of SpA or psoriasis) or if their MRI led to the diagnosis of one of these conditions.

Spinal MRI of the axSpA group could be selected retrospectively since all MRI images already had STIR sequences used to assess inflammatory lesions. However, LBP patients often had only the T1 and T2 sequences, without STIR, and retrospective case selection was not feasible. Low back pain patients were thus selected prospectively and a STIR sequence was systematically included for these patients. This did not overly extend the examination period, which increased by only a few minutes. However, routine assessment of the thoracic spine was not possible as this would have significantly increased the duration of the examination.

The study protocol was approved by the Research Ethics Committee of Toulouse University Hospital.

Clinical data

The following clinical data were collected from the medical records of axSpA patients or through self-administered questionnaires completed on the day on which LBP patients underwent their MRI scan: demographics (gender, age, height and weight), disease characteristics (duration of symptoms, morning stiffness, nighttime awakening, assessment of back pain using the visual analogue scale (VAS), Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), and Bath Ankylosing Spondylitis Functional Index (BASFI)) and NSAID intake. Additional data were collected for the axSpA group: disease characteristics (axial or axial/peripheral), personal history of arthritis, dactylitis, uveitis, inflammatory bowel disease (IBD), psoriasis, enthesitis, family history of spondyloarthritis or psoriasis, treatments (corticosteroids, synthetic DMARDs, bDMARDs), laboratory findings (HLA-B27 positivity, ESR, CRP), disease activity (Ankylosing Spondylitis Disease Activity Score ASDAS-CRP) and imaging findings (presence of radiographic syndesmophytes, sacroiliitis on radiographs (New York definition) or MRI (ASAS definition)).

MRI

All patients underwent sagittal spinal MRI using T1 and T2 STIR sequences at our hospital's radiology unit (1.5 T Phillips Achieva or 3 T Siemens Skyra). For the LBP group, the acquisition was extended to T7–T8 where possible. Images between T7 and T10 could not be acquired in certain LBP patients due to technical limitations of the examination and patient size. Hence, 5 patients in the LBP group were not imaged at and above T9–T10, 18 were not imaged at and above T8–T9 and 31 had no images at T7–T8. Thus, images above T9 were not taken into account for all patients in the analysis to avoid any selection bias. The parameters for various sequences on these two MRI units are provided in the Supplementary materials (Table S1).

All images were read by two trained rheumatologists. Details of the training method and reproducibility assessment are provided in the Supplementary materials. The images were analysed for the presence of IPAA (inflammatory posterior facet joint arthropathy, pedicle oedema, transverse process oedema, spinous process oedema, costotransverse joint arthritis—Fig. 1), inflammatory anterior arch abnormalities (IAAA—CIL or VFD), dehydration (hypointense STIR signal in the disc, which is darker than the vertebral body) or loss of more than 50% of disc height, Modic inflammatory changes (STIR hyperintensity and T1 hypointensity of the mirrored vertebral plates relating to a disc) or fatty changes (STIR and T1 hyperintensity of the mirrored vertebral plates relating to a disc), discitis (intradiscal STIR hyperintensity) and spondylolisthesis (Fig. 2).

Statistical analysis

According to published data, the prevalence of inflammatory lesions is 29% in axSpA patients versus 12% in chronic LBP patients (14). Given a 5% type I error risk and 80% power, 88 cases of axSpA and 88 cases of LBP were needed to demonstrate a significant difference between the groups. As some MRI examinations might not be usable, we decided to enrol 95 patients in each group.

The number of patients having at least one IPAA and the prevalence of each type of IPAA and IAAA in the axSpA group were compared to the LBP group using a bilateral chi-square test with 5% type I error risk. Further details about the statistical analysis are available in the Supplementary material.

All of these analyses were subsequently repeated on the subgroup of patients under 50 years of age.

The statistical analyses were carried out with STATA/IC 12.1 software (STATA Corp.).

Results

Study population

The two patient groups were comparable overall (Table 1). However, the body mass index in the LBP group was higher with a median of 25.3 g/cm^2 (22.5–29.4) versus 22.9 g/cm^2 (19.5–25.9) in the axSpA group ($p = 0.008$). The percentage of patients with morning stiffness lasting more than 30 min was higher in the axSpA ($n = 62$, 70%) than the LBP group ($n = 58$, 62%) ($p < 0.0001$). The percentage of patients who were responsive to NSAIDs was also higher in the axSpA group ($n = 78$, 94%) than in the LBP group ($n = 40$, 60.6%) ($p < 0.0001$). Lastly, the BASDAI was higher in the axSpA group (mean = 47.8, SD ± 19.4) than in the LBP group (mean = 39.9, SD ± 21.7) ($p = 0.02$).

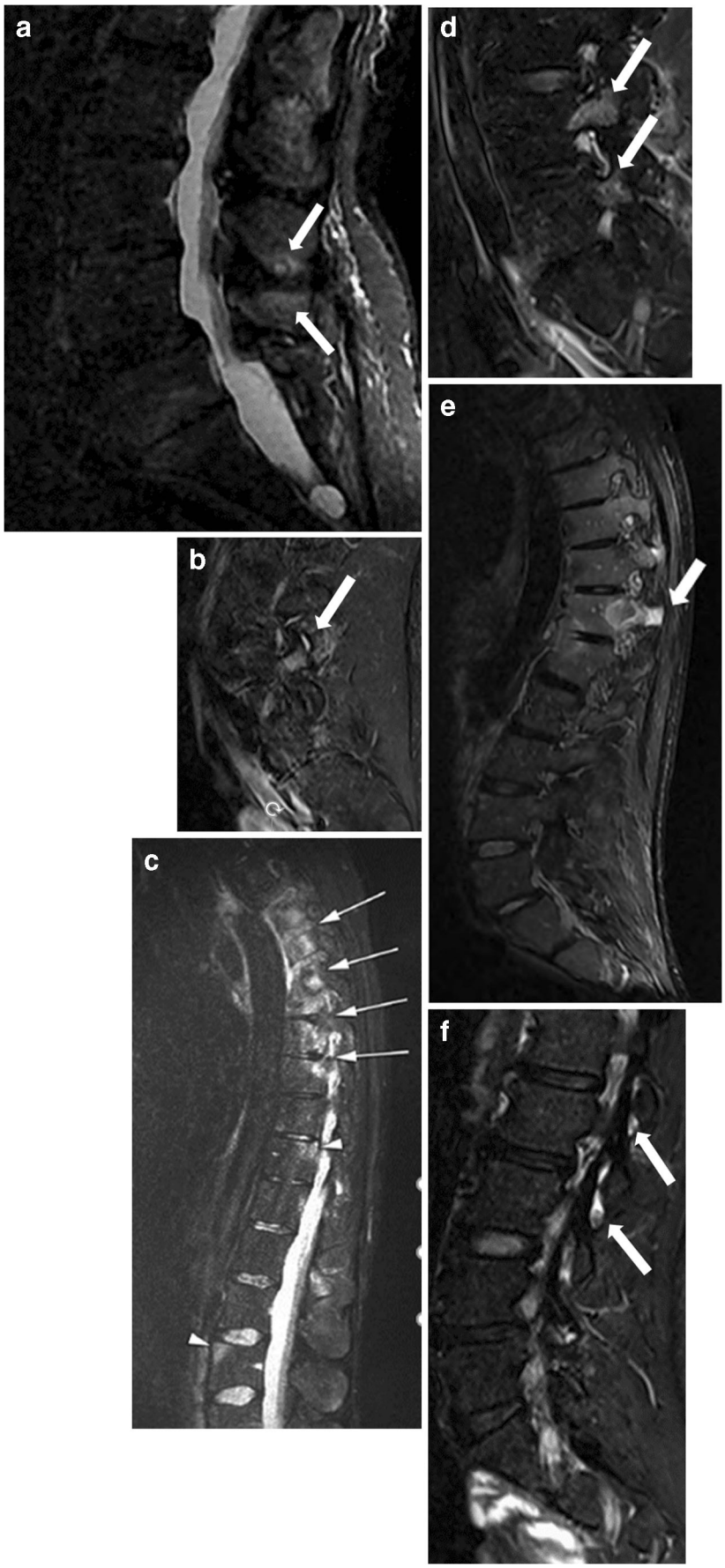

Fig. 1 Inflammatory posterior arch abnormalities on MRI T2-fat sat sequences. **a** Spinous process oedema (hypersignal STIR) (low back pain patient). **b** Zygapophyseal joint arthritis L4–L5 (hypersignal STIR) (low back pain patient). **c** Costovertebral joint arthritis and pedicle oedema from T5 to T9 (hypersignal STIR) (axial spondyloarthritis patient). **d** Pedicle oedema (hypersignal STIR) (low back pain patient). **e** Transverse process oedema (hypersignal STIR) (axial spondyloarthritis patient). **f** Zygapophyseal joint arthritis (hypersignal STIR) (low back pain patient)

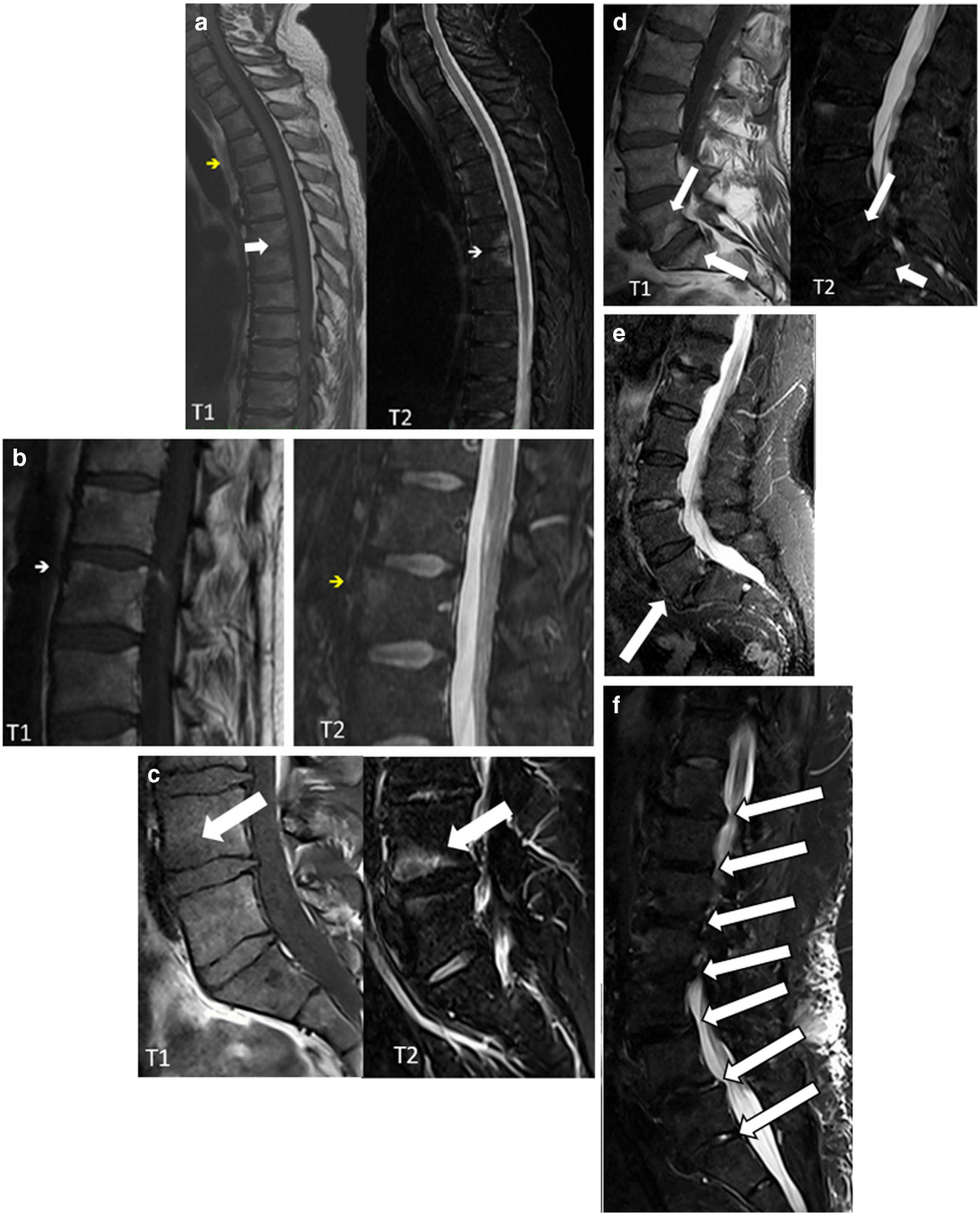

Fig. 2 Inflammatory and structural anterior arch abnormalities on MRI T1- and T2-fat sat sequences. **a** Inflammatory vertebral corner (T1 and STIR) (axial spondyloarthritis patient). **b** Fatty vertebral corner (T1 and STIR) (axial spondyloarthritis patient). **c** Modic I (inflammatory) (A: T1; B: STIR) (low back pain patient). **d** Modic II (fatty) (A: T1; B: STIR) (low back pain patient). **e** Spondylolesthesis (STIR) (low back pain patient). **f** Disc degeneration (loss of disc height, disc dehydration, STIR) (low back pain patient)

Table 1 Study population

	Spondyloarthritis ($n = 95$)	Low back pain ($n = 95$)	p value
Age (years), median (IQR)	43 (35–52)	47 (34–60)	0.052
Gender, males (%)	52 (55)	42 (44)	0.2
BMI (kg/m^2), median (IQR)	22.9 (19.5–25.9)	25.3 (22.5–29.4)	0.008
Disease duration (years), median (IQR)	10 (5–15)	5 (2–18)	0.1
Peripheral features, number (%)	58 (61.1)	0	–
Medical history of:			
– Arthritis, number (%)	19 (20)	0	–
– Dactylitis, number (%)	4 (4.2)	0	–
– Uveitis, number (%)	15 (15.8)	0	–
– IBD, number (%)	8 (8.4)	0	–
– Family history of SpA, number (%)	10 (10.5)	0	–
– Personal psoriasis, number (%)	14 (14.7)	0	–
– Family history of psoriasis, number (%)	7 (7.4)	0	–
– Inflammatory heel pain, number (%)	24 (25.3)	0	–
ASDAS CRP, mean ± SD	2.82 ± 1.13	NA	–
HLA-B27 positivity, number (%)	59 (62.1)	NA	–
ESR (mm/h), median (IQR)	8 (5–16)	NA	–
CRP (mg/l), median (IQR)	3.5 (1.2–10.4)	NA	–
Imaging signs:			
– Sacroiliitis (NY criteria), number (%)	53 (55.8)	NA	–
– At least 1 syndesmophyte, number (%)	21 (22.1)	NA	–
– MRI sacroiliitis, number (%)	59 (62.1)	NA	–
Associated treatments:			
– NSAID intake, number (%)	59 (63)	65 (72)	0.17
– NSAID good response, number (%)	78 (94)	40 (61)	< 0.0001
– csDMARD past intake, number (%)	29 (30.5)	NA	–
– csDMARD current intake, number (%)	11 (11.6)	NA	–
– TNF inhibitor past intake, number (%)	19 (20)	NA	–
Night awakening, number (%)	62 (70)	58 (62)	0.3
Morning stiffness > 30 min, number (%)	63 (72)	30 (32)	< 0.0001
VAS for pain, mean ± SD	52.7 ± 16.3	53.6 ± 21.9	0.9
BASDAI, mean ± SD	47.8 ± 19.4	39.9 ± 21.7	0.02
BASFI, median (IQR)	40 (27.5–61)	34 (16–54)	0.054

IBD, inflammatory bowel disease; *IQR*, interquartile range; *SD*, standard deviation; *VAS*, visual analogue scale; *BASDAI*, Bath Ankylosing Spondylitis Disease Activity index; *BASFI*, Bath Ankylosing Spondylitis Functional Index; *NA*, not available; *NY criteria*, sacroiliitis according to the modified New York criteria; NSAID, non-steroidal anti-inflammatory drug; *csDMARD*, conventional synthetic disease-modifying antirheumatic drugs including methotrexate, sulfasalazine and leflunomide; *TNF*, tumour necrosis factor; *CRP*, C-reactive protein; *ASDAS*, Ankylosing Spondylitis Disease Activity Score

Prevalence of IPAA

Taking all types of IPAA lesions into account, there was no significant difference between the two groups: 55 axSpA patients (57.9%) and 66 LBP patients (69.5%) had at least one IPAA ($p = 0.1$) (Table 2).

Taking each type of IPAA individually, there was a significantly higher prevalence of CT arthritis and pedicle oedema at T9–L2 in the axSpA group ($n = 16$ (17%) and $n = 12$ (13%), respectively) than in the LBP group ($n = 0$ and $n = 1$ (1.4%), respectively) ($p = 0.007$). Conversely, an isolated analysis of posterior facet joint arthropathy revealed a higher prevalence in the LBP group ($n = 45$, 63%) compared to the axSpA group ($n = 38$, 40%) ($p = 0.005$), especially at the two lowest lumbar levels (Table 2).

The presence of at least one IPAA such as pedicle oedema, spinous process oedema (regardless of level), transverse process oedema or CT arthritis was detected in 27 axSpA patients (29%) and 8 LBP patients (11%) ($p = 0.006$). Considering only pedicle oedema above L3, which is more specific to axSpA, at

Table 2 Comparison of the prevalence of inflammatory posterior arch abnormalities (IPAA) between axial spondyloarthritis and low back pain patients

IPAA		Spondyloarthritis ($n = 95$)	Low back pain ($n = 95$)	p
FJA	Patients with at least 1 lesion, *n* (%):	38 (40)	45 (63)	0.005
	Number of lesions per patient, median (IQR)	0 (0–1)	1 (0–2)	0.002
ISE	Patients with at least 1 lesion, *n* (%):	20 (21)	23 (32)	0.1
	Number of lesions per patient, median (IQR)	0 (0–0)	0 (0–1)	0.2
PE	Patients with at least 1 lesion, *n* (%):	16 (17)	7 (10)	0.2
	Patients with at least 1 lesion above L3, *n* (%):	12 (12)	1 (1)	0.007
	Number of lesions per patient, median (IQR)	0 (0–0)	0 (0–0)	0.2
SPE	Patients with at least 1 lesion, *n* (%):	10 (10.6)	3 (4.2)	0.2
	Number of lesions per patient, median (IQR)	0 (0–0)	0 (0–0)	0.1
TPE	Patients with at least 1 lesion, *n* (%):	3 (3)	0 (0)	0.3
	Number of lesions per patient, median (IQR)	0 (0–0)	0 (0–0)	0.1
CTA	Patients with at least 1 lesion, *n* (%):	16 (17)	0 (0)	< 0.0001
	Number of lesions per patient, median (IQR)	0 (0–0)	0 (0–0)	0.0002
All IPAA	Patients with at least 1 lesion, *n* (%):	55 (58)	66 (70)	0.1
	Number of lesions per patient, median (IQR)	1 (0–3)	2 (0–3)	0.4
PE + SPE + TPE + CTA	Patients with at least 1 lesion, *n* (%):	27 (29)	8 (11)	0.006
	Number of lesions per patient, median (IQR)	0 (0–1)	0 (0–0)	0.004
PE above L3 + SPE + TPE + CTA	Patients with at least 1 lesion, *n* (%):	25 (27)	4 (6)	0.0004
	Number of lesions per patient, median (IQR)	0 (0–1)	0 (0–0)	0.0003

CTA, costotransverse joint arthritis; *FJA*, facet joint arthropathy; *ISE*, interspinous oedema; *PE*, pedicle oedema; *SPE*, spinous process oedema; *TPE*, transverse process oedema; *n*, number; *IQR*, interquartile range

least one of the aforementioned IPAA was present in 25 axSpA patients (27%) and 4 LBP patients (6%) ($p = 0.0004$) (Table 2).

Prevalence of IAAA

Vertebral CILs were found in more patients in the axSpA group ($n = 39$, 41.5%) than in the LBP group ($n = 16$, 22.2%) ($p = 0.009$), particularly at the thoracic level (axSpA: $n = 30$, 31.9% versus LBP: $n = 8$, 11.1%) ($p = 0.002$) (Table 3). There was no significant difference between the groups in terms of VFD, inflammatory or fatty Modic changes, discitis or spondylolisthesis. Conversely, signs of disc degeneration were more common in LBP patients ($n = 56$, 77.8%) than axSpA patients ($n = 51$, 54.3%) (comparison: $p = 0.002$).

There were more CILs in patients with IPAA than in those without IPAA (52 versus 18%, $p = 0.02$). Furthermore, in 36.6% of cases, IPAA was apparent, while no CILs could be identified (42.2 and 30% at thoracic and lumbar spine, respectively).

Clinical factors associated with IPAA in axSpA

Given the high prevalence of posterior facet joint arthropathy and interspinous oedema in LBP patients, we defined a patient as having an IPAA when they had at least one lesion such as pedicle oedema, spinous process oedema, transverse process oedema or CT arthritis (Table 4).

Patients with at least one of these lesions had longer disease duration (11 versus 8 years, $p = 0.02$), were more likely to have a history of IBD or psoriasis (19 versus 4.4%, $p = 0.04$, and 27 versus 10.2%, $p = 0.04$, respectively) and had a lower Schöber index (2.6 versus 3.8 cm, $p = 0.0002$) and a higher ASDAS CRP level (4.1 versus 2.28, $p = 0.01$) along with a lower prevalence of peripheral arthritis (15 versus 36%, $p = 0.04$) but used NSAIDs less often (42 versus 69%, $p = 0.01$). The two groups did not differ significantly in terms of the BASDAI, BASFI and pain levels.

The percentage of HLA-B27-positive patients was comparable between groups (61 and 62%, $p = 0.99$). However, median ESR and CRP values were higher in the IPAA group (11 versus 7 mm/h, $p = 0.02$, and 11 versus 3 mg/l, $p = 0.0002$, respectively). Patients in the IPAA group had more radiographic syndesmophytes at the thoracolumbar junction (52 versus 12%, $p < 0.001$). However, the proportion of sacroiliitis found on MRI was similar.

Prevalence of IPAA, IAAA and clinical factors associated with IPAA in axSpA patients under 50 years of age

Given the increased prevalence of abnormal findings on spinal MRI with increasing age and the lower specificity of these lesions in older subjects (12), we repeated the analysis in a

Table 3 Comparison of the prevalence of inflammatory anterior arch abnormalities (IAAA) between axial spondyloarthritis and low back pain patients

IAAA		Spondyloarthritis ($n = 95$)	Low back pain ($n = 95$)	p
Inflammatory lesions				
Sacroiliitis				
• Entire spine	Patients with at least 1 lesion, *n* (%):	39 (42)	16 (22)	0.009
	Number of lesions per patients, median (IQR)	1 (0–4)	0 (0–3)	0.05
• Thoracic	Patients with at least 1 lesion, *n* (%):	30 (31.9)	8 (11.1)	0.002
	Number of lesions per patients, median (IQR)	0 (0–2)	0 (0–0)	0.003
• Lumbar	Patients with at least 1 lesion, *n* (%):	16 (16.8)	17 (17.9)	0.9
	Number of lesions per patients, median (IQR)	0 (0–2)	0 (0–1)	0.5
Discitis	Patients with at least 1 lesion, *n* (%):	3 (3.2)	3(4.2)	1.0
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.74
Structural lesions				
Vertebral fat deposition				
• Entire spine	Patients with at least 1 lesion, *n* (%):	22 (23)	15 (21)	0.7
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.7
• Thoracic	Patients with at least 1 lesion, *n* (%):	15 (16)	8 (11)	0.4
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.4
• Lumbar	Patients with at least 1 lesion, n (%):	20 (21)	14 (15)	0.3
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.3
MODIC I	Patients with at least 1 lesion, *n* (%):	14 (14.9)	16 (22.2)	0.2
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.2
MODIC II	Patients with at least 1 lesion, *n* (%):	7 (7)	8 (11)	0.4
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.7
MODIC I and/or II	Patients with at least 1 lesion, *n* (%):	17 (18)	22 (31)	0.06
	Number of lesions per patients, median (IQR)	1 (1–2)	1 (1–2)	0.3
Loss ≥ 50% disc height	Patients with at least 1 lesion, *n* (%):	49 (52.1)	48 (66.7)	0.06
	Number of lesions per patients, median (IQR)	1 (0–2)	1 (0–2.5)	0.01
Disc degeneration	Patients with at least 1 lesion, *n* (%):	51 (54.3)	56 (77.8)	0.002
	Number of lesions per patients, median (IQR)	1 (0–2)	1 (1–3)	0.0007
Spondylolisthesis	Patients with at least 1 lesion, *n* (%):	2 (2.1)	2 (2.8)	1.0
	Number of lesions per patients, median (IQR)	0 (0–0)	0 (0–0)	0.79

n, number; *IQR*, interquartile range

subpopulation of patients under 50 years of age: 67 with axSpA and 36 with LBP. This additional analysis did not alter the pattern of the findings and is provided in the Supplementary material (Tables S2 and S3).

Discussion

In this study, we found no evidence of differences in the combined prevalence of all types of IPAA between axSpA and LBP patients. Conversely, the presence of at least one IPAA—pedicle oedema above L3, transverse process oedema, spinous process oedema or CT arthritis—was much higher in the axSpA group. Patients with at least one IPAA in the axSpA group had longer disease duration with greater clinical activity and biological inflammation. Psoriasis was also more prevalent.

In the study of Bennett et al [13], severe posterior arch lesions (pedicle oedema, spinous process oedema and posterior facet joint arthropathy) were found in 29% of axSpA patients and 12% of patients with disc degeneration, resulting in a sensitivity of 16% and specificity of 88% for the diagnosis of axSpA. In the study of Weber et al [11], the sensitivity was 9% and the specificity was 100%. These lesions were less common than CILs in axSpA patients [13] but appeared earlier than IAAA in the course of the disease [19]. Of the 32 patients with axSpA in the study of Maksymowych et al [20], 87.5% had at least one posterior arch lesion—of which 46.9% were thoracic—including 84.4% transverse or spinous process oedema, 68.8% posterior facet joint arthropathy and 62.5% pedicle oedema. Among the 372 patients with LBP in the study of Lakadamyali et al [21], 85.5% had posterior facet joint arthropathy and 80.6% had interspinous oedema at L4–

Table 4 Comparison of disease features in 95 axSpA patients based on whether at least one of the following IPAA was present: pedicle oedema, transverse process oedema, spinous process oedema or CT arthritis

	SpA with IPAA $n = 26$ (27%)	SpA without IPAA $n = 69$ (73%)	p value
Age, mean ± SD	46.2 ± 10.9	41.7 ± 11.3	0.09
Men, *n* (%)	18 (69)	34 (49)	0.08
Body mass index, median (IQR)	21.4 (19.6–26.8)	23.5 (19.5–25)	0.9
Number of patients with peripheral features (%)	12 (46)	46 (67)	0.07
Disease duration (years), median (IQR)	11 (10–20)	8 (4–15)	0.02
Medical history of:			
– Arthritis, *n* (%)	4 (15)	15 (36)	0.04
– Dactylitis, *n* (%)	0	4 (6)	0.6
– Uveitis, *n* (%)	6 (23)	9 (13)	0.2
– IBD, *n* (%)	5 (19)	3 (4.4)	0.04
– Family history of SpA, *n* (%)	3 (12)	7 (5.9)	0.4
– Personal psoriasis, *n* (%)	7 (27)	7 (10.2)	0.04
– Family history of psoriasis, *n* (%)	2 (8)	5 (7.4)	1.000
– Inflammatory heel pain, *n* (%)	4 (15)	20 (29)	0.2
Night awakening, *n* (%)	17 (68)	45 (70)	0.9
Morning stiffness > 30mn, *n* (%)	18 (78)	45 (70)	0.5
BASDAI, mean ± SD	43.5 ± 22.3	49.8 ± 18	0.2
BASFI, mean ± SD	46.7 ± 11.5	42.9 ± 24	0.9
ASDAS-CRP, mean ± SD	4.1 ± 0.8	2.3 ± 0.8	0.01
Axial pain on VAS (0–100), mean ± SD	45 ± 12.9	55 ± 17.9	0.5
Modified Schöber index, mean ± SD	2.6 ± 1.6	3.8 ± 1.1	0.0002
HLA-B27 positivity, *n* (%)	16 (61)	43 (62)	1.0
ESR, median (IQR)	11 (7–23)	7(4–13)	0.02
CRP, median (IQR)	11 (5–17)	3(1–5.7)	0.0002
Imaging signs:			
– Sacroiliitis (NY criteria), *n* (%)	22 (84)	31 (47)	0.001
– At least 1 syndesmophyte, *n* (%)	13 (52)	8 (12)	< 0.0001
– Sacroiliitis on MRI, *n* (%)	16 (64)	43 (67)	0.7
Associated treatment:			
– NSAID, *n* (%)	11 (42)	48 (69)	0.01
– Good NSAID response, *n* (%)	20 (91)	58 (95)	0.6
– csDMARD past intake, *n* (%)	6 (23)	23 (33)	0.3
– csDMARD current intake, *n* (%)	3 (11)	8 (12)	1.0
– TNF inhibitor past intake, *n* (%)	5 (19)	14 (20)	0.9

VAS, visual analogue scale; *ESR*, erythrocyte sedimentation rate; *SD*, standard deviation; *n*, number; *IQR*, interquartile range; *NSAID*, non-steroidal anti-inflammatory drug; *csDMARD*, conventional synthetic disease-modifying antirheumatic drugs including methotrexate, sulfasalazine and leflunomide; *TNF*, tumour necrosis factor; *CRP*, C-reactive protein

L5 and 79.8% at L5–S1. This prevalence was significantly greater than in the control group of asymptomatic subjects. The prevalence of IPAA was higher in our study than in the study of Bennett et al [13], but lower than in the studies of Maksymowych et al [20] and Lakadamyali et al [21]. A recent study assessed the association between facet inflammation and disease activity in 53 AS patients [17]. As facet inflammation seemed to be less specific of SpA, we did not include these abnormalities in the analyses of the association between IPAA and clinical disease activity.

In the anterior arches, axSpA patients have more CILs. The latter increased when no disc degeneration was present above it [13]. In the study of Weber et al [11], the sensitivity was 66% and the specificity was 94%. In the study of Maksymowych et al [20], 87.5% of patients in their axSpA cohort had at least one anterior arch lesion occurring at the same level as any IPAA present. Finally, a recent study identified several sites on the posterior arch (transverse processes, spinous processes and facet joints) with inflammatory lesions in 3–6% of patients at the

lumbar stage which disappeared in 50–75% of cases after 24 weeks of adalimumab [22].

One of the strengths of our study is that a standardised analysis was carried out with double assessment of abnormal MRI findings in two populations large enough to facilitate statistical comparisons. The MRI acquisition protocol was standardised for all patients. All of the MRI examinations could be used. The clinical data were collected in a standardised manner for all patients.

Nevertheless, this study has several limitations. While the two populations were similar regarding age and gender ratio, the BMI was higher in the LBP group. Patients in the LBP group were older but not to any significant extent. This led to subgroup analyses with patients under 50 years of age. However, the same results were obtained.

We could not evaluate all thoracic levels in both groups due to limitations of the examination technique. Since these patients were being treated according to current practice, the MRI scan requested was typically for the lumbar spine only. The presence of lower thoracic levels on the images depended on the patient's size, coil length and good imaging resolution. Increasing the field of view would have reduced the image quality, thereby increasing the risk of missing a lesion in the relevant areas (lumbar region in this study). Furthermore, adding slices at the thoracic level would also have increased the duration and cost of the examination procedure since our centre did not have the kit to perform the whole spine MRI on the 1.5-T machine. We nevertheless assume that the comparison of inflammatory abnormalities at the lumbar level between axSpA and LBC is relevant since these lesions can be identified in an LBP patient undergoing lumbar spine MRI with suspected degenerative disorders. Furthermore, such findings at the lumbar level could be indicative of axSpA diagnosis.

The analysis of posterior facet joints is controversial. Oedema must be distinguished from effusion, with the former being more common in axSpA and the latter in LBP. In some patients, evidence of effusion at the L4–L5 and L5–S1 joints can be considered physiological, hence the difficulty of defining a pathological threshold. Oedema characteristic of posterior facet joint arthritis sometimes extends to the pedicles. Therefore, the presence of pedicle oedema is more specific to the diagnosis of axSpA when it extends above L3.

Finally, the fact that SpA patients were retrospectively selected on the inclusion of a lumbar MRI scan in their medical records could lead to selection bias. However, most of the axSpA patients in our centre have at least one spinal MRI to address inflammatory lesions indicative of axSpA and could be selected for enrolment in this study. We selected patients who met ASAS criteria, not based solely on the rheumatologist's opinion. False positive cases could potentially be selected, while ASAS-negative axSpA patients were not enrolled in this study. Patients with axSpA were selected if the relevant clinical data were available in their medical records and if they had undergone MRI in the 2-month period before or after the visit when clinical data were collected. As no patients enrolled in this study received biological therapy, we assumed that the MRI lesions would not have changed over a period of 2 months. Finally, MRIs were performed on two different MRI (1.5 and 3 T) varying in terms of performance and image quality. However, patients in both groups could have had an MRI scan on either scanner, and we assumed that this would not affect group comparison. This study assessed the diagnostic value of analysing IPAA for axSpA when lumbar spine MRI is requested in a patient with chronic LBP. Posterior facet joint arthritis, interspinous oedema and L3–L5 pedicle oedema lack specificity for an axSpA diagnosis. The presence of spinous process oedema, transverse process oedema, pedicle oedema above L3 and CT arthritis in the lower lumber levels seems to be more specific to the diagnosis of axSpA. Moreover, the presence of these lesions might be taken into account in addition to conventional clinical, biological and imaging features when considering disease activity of axial SpA.

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Informed consent Written informed consent was obtained from all subjects (patients) in this study.

Ethical approval Institutional Review Board approval was obtained.

Methodology
- Case–control study
- Cross-sectional study
- Diagnostic study
- Observational
- Performed at one institution

References

1. Benjamin M, Rufai A, Ralphs JR (2000) The mechanism of formation of bony spurs (enthesophytes) in the achilles tendon. Arthritis Rheumatol 43:576–583
2. Rudwaleit M, van der Heijde D, Landewé R et al (2009) The development of Assessment of SpondyloArthritis International

Society classification criteria for axial spondyloarthritis (part II): validation and final selection. Ann Rheum Dis 68:777–783
3. Weber U, Lambert RG, Østergaard M, Hodler J, Pedersen SJ, Maksymowych WP (2010) The diagnostic utility of magnetic resonance imaging in spondylarthritis: an international multicenter evaluation of one hundred eighty-seven subjects. Arthritis Rheumatol 62:3048–3058
4. Arnbak B, Jensen TS, Egund N et al (2016) Prevalence of degenerative and spondyloarthritis-related magnetic resonance imaging findings in the spine and sacroiliac joints in patients with persistent low back pain. Eur Radiol 26:1191–1203
5. Lambert RG, Bakker PA, van der Heijde D et al (2016) Defining active sacroiliitis on MRI for classification of axial spondyloarthritis: update by the ASAS MRI working group. Ann Rheum Dis 75:1958–1963
6. Weber U, Zubler V, Pedersen SJ et al (2013) Development and validation of a magnetic resonance imaging reference criterion for defining a positive sacroiliac joint magnetic resonance imaging finding in spondyloarthritis. Arthritis Care Res (Hoboken) 65: 977–985
7. Wendling D, Claudepierre P, Pham T, Loeuille D, Prati C (2015) MRI in axial spondyloarthritis: from light to shadow? Joint Bone Spine 82:302–304
8. Althoff CE, Sieper J, Song IH et al (2013) Active inflammation and structural change in early active axial spondyloarthritis as detected by whole-body MRI. Ann Rheum Dis 72:967–973
9. Baraliakos X, Landewé R, Hermann KG et al (2005) Inflammation in ankylosing spondylitis: a systematic description of the extent and frequency of acute spinal changes using magnetic resonance imaging. Ann Rheum Dis 64:730–734
10. Weber U, Pfirrmann CW, Kissling RO, Hodler J, Zanetti M (2007) Whole body MR imaging in ankylosing spondylitis: a descriptive pilot study in patients with suspected early and active confirmed ankylosing spondylitis. BMC Musculoskelet Disord 8:20
11. Weber U, Hodler J, Kubik RA et al (2009) Sensitivity and specificity of spinal inflammatory lesions assessed by whole-body magnetic resonance imaging in patients with ankylosing spondylitis or recent-onset inflammatory back pain. Arthritis Rheumatol 61: 900–908
12. Bennett AN, Rehman A, Hensor EM, Marzo-Ortega H, Emery P, McGonagle D (2010) The fatty Romanus lesion: a non-inflammatory spinal MRI lesion specific for axial spondyloarthropathy. Ann Rheum Dis 69:891–894
13. Bennett AN, Rehman A, Hensor EM, Marzo-Ortega H, Emery P, McGonagle D (2009) Evaluation of the diagnostic utility of spinal magnetic resonance imaging in axial spondylarthritis. Arthritis Rheumatol 60:1331–1341
14. van der Heijde D, Sieper J, Maksymowych WP et al (2014) Spinal inflammation in the absence of sacroiliac joint inflammation on magnetic resonance imaging in patients with active nonradiographic axial spondyloarthritis. Arthritis Rheumatol 66:667–673
15. Weber U, Zubler V, Zhao Z et al (2015) Does spinal MRI add incremental diagnostic value to MRI of the sacroiliac joints alone in patients with non-radiographic axial spondyloarthritis? Ann Rheum Dis 74:985–992
16. Ez-Zaitouni Z, Bakker PA, van Lunteren M et al (2017) The yield of a positive MRI of the spine as imaging criterion in the ASAS classification criteria for axial spondyloarthritis: results from the SPACE and DESIR cohorts. Ann Rheum Dis 76:1731–1736
17. Lee S, Lee JY, Hwang JH, Shin JH, Kim TH, Kim SK (2016) Clinical importance of inflammatory facet joints of the spine in ankylosing spondylitis: a magnetic resonance imaging study. Scand J Rheumatol 45:491–498
18. Hermann KG, Baraliakos X, van der Heijde DM et al (2012) Descriptions of spinal MRI lesions and definition of a positive MRI of the spine in axial spondyloarthritis: a consensual approach by the ASAS/OMERACT MRI study group. Ann Rheum Dis 71: 1278–1288
19. Bochkova AG, Levshakova AV, Bunchuk NV, Braun J (2010) Spinal inflammation lesions as detected by magnetic resonance imaging in patients with early ankylosing spondylitis are more often observed in posterior structures of the spine. Rheumatology (Oxford) 49:749–755
20. Maksymowych WP, Crowther SM, Dhillon SS, Conner-Spady B, Lambert RG (2010) Systematic assessment of inflammation by magnetic resonance imaging in the posterior elements of the spine in ankylosing spondylitis. Arthritis Care Res (Hoboken) 62:4–10
21. Lakadamyali H, Tarhan NC, Ergun T, Cakir B, Agildere AM (2008) STIR sequence for depiction of degenerative changes in posterior stabilizing elements in patients with lower back pain. AJR Am J Roentgenol 191:973–979
22. Krabbe S, Sørensen IJ, Jensen B et al (2018) Inflammatory and structural changes in vertebral bodies and posterior elements of the spine in axial spondyloarthritis: construct validity, responsiveness and discriminatory ability of the anatomy-based CANDEN scoring system in a randomised placebo-controlled trial. RMD Open 4:e000624