left HJ (Table 1). There were no statistical differences between MRI symptoms of the impairment HJ and US symptoms of coxitis at baseline and after 2 years (Table 2).

Radiographic progression (BASRI-hip ≥ 1 stage) after 2 years follow-up founded in 7 (31.8%) pts with MRI symptoms of the impairment HJ. There are radiographic progression from normal HJ to bilateral stage 1 in 5 (71.4%) pts, from bilateral stage 1 to bilateral stage 2 in 2 (28.6%) pts. Mean NSAID index in pts with radiographic progression (31.8%) amount 62.2%, while in pts without radiographic progression – 72.5% ($p=0.2$).

Conclusions: 1. In patients with early axial spondyloarthritis in two years of observation radiographic progression observed in 31.7% patients despite on regular intake of NSAIDs. 2. Further studies of the impairment HJ are required in patients with axial SpA.

Disclosure of Interest: None declared

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SAT0395 SIMILARITIES AND DIFFERENCES BETWEEN NON-RADIOGRAPHIC AND RADIOGRAPHIC AXIAL SPONDYLOARTHRITIS IN PROOF COHORT

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Background: Previously, some differences between non-radiographic and radiographic axial spondyloarthritis (axSpA) – such as a higher prevalence of females and lower level of acute phase reactants in non-radiographic axSpA (nr-axSpA) – have been reported in national observational studies, mostly from Europe.

Objectives: To compare demographic and clinical characteristics of patients (pts) with nr-axSpA and radiographic axSpA (ankylosing spondylitis, AS) in a large multinational cohort of pts with recently diagnosed axSpA.

Methods: PROOF is a prospective observational study evaluating clinical and radiographic outcomes in axSpA pts in rheumatology clinical practice in 29 countries. Pts with axSpA fulfilling ASAS classification criteria were eligible if diagnosed ≤ 1 year prior to study enrolment. Investigator's confidence with the diagnosis of axSpA was ascertained on a numeric rating scale (NRS 0–10) at enrolment and end of follow-up. At baseline, demographic and clinical data related to the diagnosis, disease activity, quality of life and work productivity, as well as conventional radiographs of the sacroiliac joints were collected. Classification as nr-axSpA or AS was based on the results of the assessment of sacroiliac radiographs. Available radiographs were assessed first by a local reader and then by a central reader according to the grading system of the modified New York criteria. In the case of a disagreement in the classification (nr-axSpA or AS), the radiograph was evaluated by the 2nd central reader, who was blinded to the previous assessments and the final classification was made based on the decision of 2 out of 3 readers.

Results: Of the 2126 pts enrolled in PROOF, 1281 (60.3%) pts were classified as AS and 845 (39.7%) as nr-axSpA according to investigators. The confidence with the diagnosis of axSpA was 8.7 ± 1.8 . The final classification according to the central assessment of sacroiliac radiographs was confirmed in 1583 pts included in this analysis. A total of 987 pts (62.3%) were classified as AS and 596 (37.7%) as nr-axSpA. AS pts expectedly had longer symptom duration, more frequently had elevated and higher CRP and were more often male and treated with TNF inhibitors (Table). In addition, HLA-B27 positivity was more frequent among AS pts, while pts with nr-axSpA had a significantly higher prevalence of enthesitis, psoriasis, and inflammatory bowel disease (IBD). The prevalence of other SpA features was comparable between the two subgroups of axSpA. Mostly, pt-reported outcomes reflecting burden of disease were comparable between the two subgroups, but BASDAI was significantly higher in the nr-axSpA subgroup (Table).

Conclusions: There were a few differences between nr-axSpA and AS pts in the PROOF cohort. The clinical constellation of female sex, low CRP, enthesitis, psoriasis, and IBD in nr-axSpA pts appears to reflect a phenotype less prone to structural damage in the sacroiliac joints. However, the clinical burden of disease was comparable between the two subgroups of axSpA.

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Table Baseline demographic and clinical characteristics of patients from PROOF cohort.

Characteristic	nr-axSpA (N=644)	AS (N=1039)	P-value*
Age, years, mean $\pm$ SD	35.5 $\pm$ 9.8	34.5 $\pm$ 11.1	.070
Duration since back pain onset, months, mean $\pm$ SD	48.7 $\pm$ 69.2	62.4 $\pm$ 90.9	.001
Duration since diagnosis, months, mean $\pm$ SD	2.8 $\pm$ 5.6	4.0 $\pm$ 20.2	.119
Male sex, n (%)	264 (48.5)	737 (71.0)	<.001
HLA-B27 (+), n (%)	254 (55.3) ^b	591 (69.0) ^c	<.001
Inflammatory back pain, n (%)	512 (94.1)	991 (95.4)	.279
Peripheral arthritis, n (%)	171 (31.4)	343 (33.0)	.535
Enthesitis (heel), n (%)	214 (39.3)	348 (33.5)	.023
Dactylitis, n (%)	32 (5.9)	57 (5.5)	.732
Uveitis, n (%)	49 (9.0)	106 (10.2)	.477
Psoriasis, n (%)	54 (9.9)	59 (5.7)	.003
IBD, n (%)	23 (4.2)	18 (1.7)	.004
Good response to NSAIDs, n (%)	324 (59.6)	636 (61.2)	.551
Family history of SpA, n (%)	101 (18.6)	196 (18.9)	.946
Elevated CRP, n (%)	178 (32.7)	555 (53.4)	<.001
Number of positive SpA parameters, mean $\pm$ SD	3.5 $\pm$ 1.4	3.8 $\pm$ 1.4	.001
CRP, mg/l, mean $\pm$ SD	11.5 $\pm$ 19.5	17.6 $\pm$ 24.3	<.001
ASDAS-CRP, mean $\pm$ SD	2.8 $\pm$ 1.1	3.0 $\pm$ 1.1	.004
BASDAI, points NRS (0-10), mean $\pm$ SD	4.8 $\pm$ 2.4	4.3 $\pm$ 2.3	<.001
Patient global, points NRS (0-10), mean $\pm$ SD	5.0 $\pm$ 4.8	4.8 $\pm$ 4.6	.183
BASFI, points NRS (0-10), mean $\pm$ SD	3.4 $\pm$ 2.5	3.3 $\pm$ 2.5	.815
SF-12v2, physical component score, mean $\pm$ SD	40.9 $\pm$ 8.9	41.0 $\pm$ 8.8	.698
SF-12v2, mental component score, mean $\pm$ SD	42.9 $\pm$ 10.9	43.7 $\pm$ 10.4	.166
WPAI-SHP – total activity impairment, mean $\pm$ SD	44.9 $\pm$ 28.1	43.1 $\pm$ 27.4	.208
NSAIDs, n (%)	428 (78.7)	800 (77.0)	.485
Methotrexate, n (%)	40 (7.4)	63 (6.1)	.335
Sulfasalazine, n (%)	117 (21.5)	253 (24.4)	.212
Steroids, n (%)	40 (7.4)	85 (8.2)	.624
Analgesics, n (%)	98 (18.0)	144 (13.9)	.033
TNF α inhibitors, n (%)	48 (8.8)	165 (15.9)	<.001

*P-values from two-sided t-test for scale variables and Fisher's exact test for categorical variables.

^bN = 459, ^cN = 656.

nr-axSpA = non-radiographic axial spondyloarthritis; AS = Ankylosing spondylitis; SD = standard deviation; SpA = spondyloarthritis; HLA-B27 = human leukocyte antigen B27; IBD = inflammatory bowel disease; NSAIDs = non-steroidal anti-inflammatory drugs; CRP = C-reactive protein; ASDAS-CRP = Ankylosing Spondylitis Disease Activity Score containing CRP; BASDAI = Bath Ankylosing Spondylitis Disease Activity Index; NRS = numeric rating scale; BASFI = Bath Ankylosing Spondylitis Functional Index; SF-12v2=Short form 12-Item health survey; WPAI-SHP = Work productivity impairment Questionnaire-specific health problem; TNF = tumor necrosis factor.

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SAT0396 INVESTIGATION OF IRON DEFICIENCY ANEMIA IN ANKYLOSING SPONDYLITIS PATIENTS

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Background: It is reported that subclinic intestinal inflammation may occur in Ankylosing spondylitis (AS) patients, besides, using NSAIDs cause peptic and duodenal ulcers. %50–60 of AS patients have asymptomatic ileal and colonic mucosal inflammation. It is reported that inflammatory bowel disease (IBD) is found in 5–10% of AS patients and 4–10% of IBD patients have concomitant findings with AS. These conditions may cause iron deficiency anemia (IDA).

Objectives: It is well known that chronic disease anemia is a frequent finding in AS patients. But there is no study in the literature about relationship between AS patients and IDA. In this particular study we aimed to assess frequency of IDA in AS patients and to investigate the etiologies of IDA.

Methods: Ninety four consecutive AS patients who meet 2012 ASAS/EULAR criteria, who were followed Cukurova University Romatology Clinic, were included. We investigated the etiologies of IDA in anemic patients. Twenty six AS patients were diagnosed as IDA. Twenty six patients without anemia were assigned as a control group. Hepcidin, soluble transferrin receptor1 (sTfR1) and anemia parameters were tested in both groups. Findings were analyzed with SPSS version 23.

Results: Twenty six of 94 AS patients were diagnosed as IDA (%27). Frequency of IDA in our AS patients was higher when compared to the IDA prevalence in the society (%1–2). Endoscopy and colonoscopy were performed for searching etiology of IDA. Mucosal inflammation was found in 62% of patients by endoscopy and 11% of patients by colonoscopy. One patient was diagnosed as Crohn's disease and one patient was diagnosed as Coeliac disease histopathologically. Hepcidin was found to be significantly lower in IDA patients ($p<0.01$). We found sTfR1 levels significantly higher in IDA patients ($p<0.01$). BASDAI (Bath Ankylosing Spondylitis Disease Activity Index) and sedimentation values were found to be higher in IDA patients statistically ($p<0.01$ and $p=0.01$ respectively). Although we found C- reactive protein (CRP) values were higher when compared to the non- anemic patients; however it was not statistically significant ($p>0.05$).

Conclusions: We found higher frequency of IDA when compared to the normal