positive inflammatory assessment with very high activity indices, with a mean of 4.6. 64.86% of the patients received NSAIDs, of which 11% responded well. 57% were treated with csDMARDs, and 17.86% were treated with biologics. At the time of our study, the mean visual analog scale was 5.84 ± 1.7 out of 10 (2-9). The mean Epworth score was 8.38 ± 5.2 (0-21), 56.1% of patients had no sleep debt, 33.3% had a sleep deficit, and only 10.6% had signs of drowsiness. For the overall Pittsburgh score, the mean was 702.3 ± 3.6 (1-18). The mean of “subjective quality of sleep” was 1.12, “sleep latency” was 1.22, “duration of sleep” was 1.06, “usual sleep efficiency” was 0.74, “Sleep disturbance” of 1.28, “use of a sleep medication” of 0.54, and the average of the component concerning “poor shape during the day” was 1.03 out of 3. The LEQUESNE index went from an average of 6 to 8, which corresponds to an average handicap (P=0.2) over a period of 3 years. 68% of the patients had an alteration in the quality of sleep, starting on average three years after the onset of symptoms. 11% reported having experienced anxiety and depressive symptoms, and reported having used antidepressants or anxiolytics in the past 5 years.

Conclusion: Our study showed the negative impact of SpA on the duration and overall quality of sleep. The degree of pain as well as functional impairment can cause and worsen sleep disturbances in SpA. We have shown that the Pittsburg score increases significantly with the increase of pain. The Lequesne score and the Epworth score increase with disease activity[1].

REFERENCES:

Disclosure of Interests: None declared.

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Table 1. Descriptive output of data

<table>
<thead>
<tr>
<th></th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>82</td>
</tr>
<tr>
<td>Females</td>
<td>29% (23)</td>
</tr>
<tr>
<td>Males</td>
<td>71% (59)</td>
</tr>
<tr>
<td>Age</td>
<td>45.03</td>
</tr>
<tr>
<td>BASDAI</td>
<td>4.33</td>
</tr>
<tr>
<td>BASFI</td>
<td>3.88</td>
</tr>
<tr>
<td>Pen score</td>
<td>3.93</td>
</tr>
<tr>
<td>Sock score</td>
<td>2.88</td>
</tr>
<tr>
<td>Sock score greater</td>
<td>7.2% (6)</td>
</tr>
<tr>
<td>Pen score greater</td>
<td>50% (41)</td>
</tr>
<tr>
<td>Same scores</td>
<td>42.7% (35)</td>
</tr>
</tbody>
</table>

Conclusion: There is a strong positive correlation between sock (question 1) and pen scores (question 2) as captured by the BASFI. It appears that both questions are capturing a similar functional limitation in patients with axSpA. In order to minimise redundancy and improve the relevance of the BASFI our results support the removal of one of these questions to simplify the BASFI. From a practical perspective, putting on socks (question 1) would be a more commonly encountered daily activity than picking up a pen from the floor (question 2). As such, we would suggest removal of question 2 from the BASFI.

Table 1 & Figure 1

Graph 1. Spread of Data points demonstrating a monotonic relationship with no outliers

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POS1000

COMORBIDITIES ASSOCIATED TO SPONDYLOARTHRITIS

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Background: In contrast to other chronic rheumatic diseases such as rheumatoid arthritis, comorbidities associated to spondyloarthritis (SpA) and their impact on disease outcomes are less well studied.

Objectives: The aim of our study was to investigate the prevalence of comorbidities among SpA patients and to determine factors influencing their appearance.

Methods: We conducted a retrospective study including patients meeting the Assessment of SpondyloArthritis International Society (ASAS) criteria between 2000 and 2020.

The following comorbidities were collected: cardiovascular pathologies and their risk factors (smoking, arterial hypertension, diabetes, dyslipidemia and obesity), neoplasms, osteoporosis, depression, infections, gastrointestinal and pulmonary disorders.

Results: We included 138 patients. Sixty-eight per cent of them were males. The mean age was 45.73 ± 12.66 years. The mean age at the disease onset was 28.89 ± 12.54 years. The mean CRP was 33.38 ± 39.65 mg/dL. The mean BASDAI and ASDAS-CRP were 4.21 ± 2.23 and 3.06 ± 1.26, respectively. The mean BASFI was 4.77 ± 2.58.

Sixty patients had at least one comorbidity (43.5%); 53 patients had one comorbidity (38.4%), 21 accumulated two types of comorbidities (15.2%) and 7 patients accumulated three types or more (5%).

Osteoporosis was the most frequent comorbidity, it was present in 23.1% of the cases (n=32), followed by tuberculosis 8.7% (n=12), stomach ulcers 5.1% (n=7), cardiovascular pathologies and their risk factors (smoking, arterial hypertension, diabetes, dyslipidemia and obesity), neoplasms, osteoporosis, depression, infections, gastrointestinal and pulmonary disorders.

Thirty-seven per cent of our patients were smokers.

SpA patients with comorbidities were significantly older than those without (50.2 ± 11.07 versus 42.3 ± 12.8 years, p<0.0001).

The presence of comorbidities was significantly associated to a higher disease activity evaluated by BASDAI (p=0.005) and ASDAS-CRP (p=0.002).

Furthermore, BASFI was significantly higher among patients with comorbidities (5.47 ± 2.38 versus 4.31 ± 2.62, p=0.028).

However, no association was found between presence of comorbidities and smoking or CRP.

Conclusion: Our results show that more than 40% of our SpA patients presented with at least one comorbidity. Remarkably, the presence of comorbidities...
was associated with high disease activity, suggesting that that inflammation might promote comorbidities. For optimal management of SpA, a systemic screening for comorbidities is essential.

Disclosure of Interests: None declared.

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POS001

CLINICAL AND SURGROATE CARDIOVASCULAR RISK ASSESSMENT AND ITS RELATIONSHIP WITH PSORIATIC ARTHRITIS PATHOGENESIS

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Results: Traditional CV risk factors including atherogenic risk, insulin resistance (IR), metabolic syndrome, smoking, obesity, arterial hypertension, apolipoprotein B/A ratio, type 2 diabetes mellitus and the levels of SCORE were significantly increased in PsA patients. The presence of IR was associated with disease activity markers (DAPSA, ESR and CRP). In fact, the HOMA-IR index was related to the CRP persistence. PsA patients with obesity had significantly increased the number of tender and swollen joints, the levels of DAPSA and CRP. Twenty-eight proteins involved in CV disease and six adipocytokines were significantly elevated in the plasma of PsA patients. Several of these cardiovascular molecules were associated with higher levels of DAPSA (CTSD, GAL3, CD163, FABP4, IL6 and LIFRT2), acute phase reactants (GAL3, TNF, Fas), adiponectin, TNF1, IL6 and IL8), affected body surface area (IL2RA, GAL3, CCL15, TRAP, C5B, CD163, OPG and CNTN1) and onychopathy (TRAP, WVF, MCP-1, GAL3, LTBR, TFP1, CH3L1, CTSZ and JAM-A). In addition, the mRNA expression of most of those 28 CV molecules were significantly increased in PBMCs from PsA patients. At intracellular level, the activation of 11 kinases (ERK1/2, AKT, S6 Ribosomal, mTOR, HSP27, Bad, p70 S6 kinase, PAR4, p53 and caspase-3) involved in insulin signaling, inflammation, cell survival and apoptosis were altered in PBMCs. Finally, serum from PsA patients was able to modify the expression of these molecules in adipocytes.

Conclusion: 1) Disease activity and inflammatory burden are closely associated with the presence of metabolic alterations, specifically obesity and IR in patients with PsA. 2) The development of IR is extremely related to the persistence of CRP levels in the previous 5 years. 3) Inflammation is closely associated to the moment of disease progression in PsA and 4) FABP4, CD163 and GAL3 are surrogate CV markers commonly associated with clinical features of PsA, suggesting the role of these molecules linking CVD and PsA pathogenesis.

Disclosure of Interests: None declared.

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POS002

BASELINE CALPROTECTIN AND VISFATIN LEVELS PREDICT RADIOGRAPHIC SPINAL PROGRESSION AFTER 2 YEARS IN ASYMMETRIC SPONDYLITIS PATIENTS ON TNF INHIBITOR THERAPY

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Background: Radiographic spinal progression determines functional status and mobility in ankylosing spondylitis (AS).

Objectives: To analyse whether biomarker of inflammation, bone turnover and adipokines at baseline or their change after 3 months or 2 years can predict spinal radiographic progression after 2 years in AS patients treated with TNF-α inhibitors (TNFi).

Methods: Consecutive AS patients from the Groningen Leeuwarden Axial Spondyloarthritis (GLAS) cohort starting TNFi between 2004 and 2012 were included. The following serum biomarkers were measured at baseline, 3 months and 2 years of follow-up with ELISA:

- Markers of inflammation: calprotectin, matrix metalloproteinas-3 (MMP-3), vascular endothelial growth factor (VEGF)
- Markers of bone turnover: bone-specific alkaline phosphatase (BALP), serum C-terminal telopeptide (sCTX), osteocalcin (OC), osteoprotegerin (OPG), procollagen type I and II N-terminal propeptide (PINP; PITP), sclerostin.
- Adipokines: high molecular weight (HMW) adiponectin, leptin, visfatin

Two independent readers assessed spinal radiographs at baseline and 2 years of follow-up according to the modified Stoke Ankylosing Spondylitis Spine Score (mSASSS). Radiographic spinal progression was defined as mSASSS change ≥2 units or the formation of ≥1 new syndesmophyte over 2 years. Logistic regression was performed to examine the association between biomarker values at baseline, their change after 3 months and 2 years and radiographic spinal progression. Multivariable models for each biomarker were adjusted for mSASSS or syndesmophytes at baseline, elevated CRP (≥5mg/l), smoking status, male gender, symptom duration, BMI, and baseline biomarker level (the latter only in models with biomarker change).

Results: Of the 137 included AS patients, 72% were male, 79% HLAB27+; mean age at baseline was 42 years (SD 10.8), ASDAScrp 3.8 (0.8) and mSASSS 10.6 (16.1). After 2 years of follow-up, 33% showed mSASSS change ≥2 units and 24% had developed ≥1 new syndesmophyte. Serum levels of biomarkers of inflammation and bone formation showed significant changes under TNFi therapy, whereas adipokine levels were not altered from baseline (Figure 1).

Univariable logistic regression revealed a significant association of baseline visfatin (odds ratio OR [95% confidence interval] 1.106 [1.007-1.215]) and sclerostin serum levels (OR 1.006 [1.001-1.011]) with mSASSS progression after 2 years. Baseline sclerostin levels were also associated with syndesmophyte progression (OR 1.001 [1.000-1.013]). In multivariable logistic analysis, only baseline visfatin level remained significantly associated (OR 1.465 [1.137-1.880]) with mSASSS progression. Furthermore, baseline calprotectin showed a positive association with both, mSASSS (OR 1.195 [1.055-1.355]) and syndesmophyte progression (OR 1.107 [1.001-1.225]) when adjusting for known risk factors for radiographic progression.

Univariable logistic regression showed that change of visfatin after 3 months was associated with syndesmophytes progression (OR 1.007 [1.000-1.015]). However, those associations were lost in multivariable analysis.

Conclusion: Independent of known risk factors, baseline calprotectin and visfatin levels were associated with radiographic spinal progression after 2 years of TNFi. Although biomarkers of inflammation and bone formation showed significant changes under TNFi therapy, these changes were not significantly related to radiographic spinal progression in our cohort of AS patients.

REFERENCES:

[1] Podubnyi et al 2018