Table. Ranking of the parameters relevant to deciding on the presence of axial involvement in a PsA patient in the opinion of ASAS and GRAPPA members (n=186).

N	Parameters	Median rank	Mean rank
1	Presence of structural damage on an X-ray of SIJ	2	2.8
2	Presence of structural damage on an X-ray of spine	3.5	4.1
3	Presence of subchondral BME / osteitis on MRI of SIJ compatible with SpA	4	4.5
4	Presence of BME / osteitis on MRI of spine compatible with SpA	4	5
5	History or current presence of back pain	5.5	5.8
6	History of or current presence of inflammatory back pain	5.5	6
7	Good response of back pain to non-steroidal anti-inflammatory drugs	8	7.8
8	HLA-B27	8	8.1
9	Family history for SpA	9.5	9
10	Elevated C-reactive protein	10	9.3
11	Presence of peripheral arthritis and/or enthesitis and/or dactylitis	10	9.4
12	Presence of anterior uveitis	10	9.5
13	Presence of inflammatory bowel disease	10	9.6

BME=bone marrow edema, MRI=magnetic resonance imaging, SIJ=sacroiliac joints, SpA=spondyloarthritis

Figure. An example of a survey question with two clinical scenarios comparing two pairs of parameters to determine part-worth utilities of the parameters.

In a patient with diagnosed psoriatic arthritis fulfilling the CASPAR criteria, which of these 2 (hypothetical) profiles would be indicative of the presence of axial involvement? (all else being equal)

History of or current presence of inflammatory back pain (IBP)
Presence of IBP

Presence of structural damage on an X-ray of sacroiliac joints
No definite sacroiliitis (grade 0 to 1)

this one

this combination is impossible

OR

History of or current presence of inflammatory back pain (IBP)
No IBP

Presence of structural damage on an X-ray of sacroiliac joints
Radiographic sacroiliitis of at least grade 2 bilaterally or grade 3 unilaterally (grading system of the mNY criteria for ankylosing spondylitis)

this one

this combination is impossible

they are equal

skip this question for now >

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AB0825 TIME-COURSE CHANGE IN AXIAL MOBILITY IN PSORIATIC ARTHRITIS PATIENTS UNDER bDMARD

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Background: Spinal mobility is assessed frequently in patients with psoriatic arthritis (PsA) using *Bath Ankylosing Spondylitis Metrology Index* (BASMI) to provide baseline measurement, monitor changes over time and to assess the impact of clinical interventions. BASMI comprises 4 measures of spinal mobility (cervical rotation, tragus-to-wall distance, modified Schober's test and lumbar lateral flexion) and one hip mobility measurement (intermalleolar distance).

Objectives: The aim of this study is to investigate the time-course change of BASMI in PsA patients after 6 months of *Biologic Disease-modifying Antirheumatic Drug* (bDMARD) therapy. The authors also pretend to evaluate, at baseline and after 6 months of treatment, the association between BASMI, disease activity scores and physical function.

Methods: An observational retrospective study was performed in patients with PsA under bDMARD followed in the Rheumatology department of a tertiary university hospital. Were included patients treated with only one bDMARD. Demographic and clinical data were collected from the Rheumatic Diseases Portuguese Register. For spinal mobility calculation BASMI was used. Disease activity was evaluated with *Ankylosing Spondylitis Disease Activity Score* (ASDAS) and *Bath Ankylosing Spondylitis Activity Index* (BASDAI). Physical function was assessed with *Bath Functional Index* (BASFI). The variation of BASMI, ASDAS, BASDAI and BASFI was calculated as the difference between values registered at 6 months and at baseline and presented as Δ . Correlations between Δ BASMI, Δ ASDAS and Δ BASFI was calculated using Pearson test.

Results: A total of 55 patients were included. Thirty patients were males (54.5%). The mean age at diagnosis was 44.6 ± 12.6 years and the median disease duration at start of bDMARD was 5.4 years (min: 0.30; max: 25.5). In total, 19 (34.5%) patients had predominant axial involvement, 36 (65.5%) peripheral and 36 (65.5%) enthesopathic. Almost all patients fulfilled the CASPAR criteria for PsA (n=50, 90.9%). According to ASDAS criteria, at the baseline 20 patients (36.4%) had high disease activity and 34 (61.8%) very high. The most used bDMARD was etanercept (n=21, 38.3%) followed by golimumab (n=19, 34.5%) and adalimumab (n=8, 14.5%). Three patients were treated with infliximab, two with certolizumab and other two with secukinumab. Forty-one patients (75.9%) were concomitantly treated with conventional synthetic DMARDs. Axial PsA patients had more limitations in spinal mobility (BASMI mean 4.5 ± 1.5) and more functional limitation (BASFI mean 6.8 ± 1.9) than patients with predominant peripheral involvement (BASMI mean 3.3 ± 1.2 , $p=0.004$; BASFI mean 5.4 ± 3 , $p=0.0048$). Statistically significant differences in ASDAS and BASDAI in these two groups were not observed ($p=0.332$ and $p=0.605$, respectively). For all patients, BASMI did not vary significantly ($p=0.691$) at baseline (mean 3.7 ± 1.4) and after 6 months (mean 3.8 ± 1.3) of treatment. Although the Δ BASMI for etanercept was negative (mean -0.12 ± 0.9) and for golimumab positive (0.14 ± 0.8), it was not statistically significant. At baseline there is a significant positive association between BASMI and ASDAS ($r=0.435$, $p=0.001$), BASMI and BASDAI ($r=0.567$, $p<0.001$) and BASMI and BASFI ($r=0.510$, $p<0.001$). However, there was not a statistically significant association between Δ BASMI and: Δ ASDAS, Δ BASDAI and Δ BASFI ($r=0.158$; $p=0.269$, $r=0.019$; $p=0.096$ and $r=0.121$; $p=0.397$, respectively).

Conclusion: In PsA patients treated with bDMARDs, at least in short-term follow-up, BASMI does not improve with time. Changes in BASMI did not correlate with changes in activity disease and in functional outcome. Studies with longer follow-up and with more patients are needed to better evaluate these associations.

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AB0826

PROBABILITY OF SURVIVAL OF USTEKINUMAB IN PSORIATIC ARTHRITIS: A REAL CLINICAL PRACTICE COHORT COMPOSED OF 64 PATIENTS

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Background: Psoriatic arthritis (PsA) is an inflammatory disorder of unknown etiology. Several domains are affected as peripheral or axial joints, enthesitis, dactylitis, nails as well as skin. Diverse cytokines have been described in the pathology of PsA as TNF α , IL-17 and IL-23. Ustekinumab (UST) is a fully human IgG1 κ monoclonal antibody to interleukin 12/23. Its efficacy and safety have been tested in several clinical trials and registries. Nevertheless data from real word evidence studies is needed to understand the effectiveness, safety and behavior of UST in a different population of patients from randomized controlled trials

Objectives: Analyze the persistence of UST 45 and 90mg along 52 weeks of treatment

Methods: Drug survival, effectiveness and security of UST were studied in a population of 64 PsA patients treated in the period between August 2014 to October 2019. Drug survival was defined as the time from initiation to discontinuation (stop/switch) of bDMARDs. For the determination of drug survival, Kaplan-Meier survival curves and Cox-regression analyses were used. Effectiveness was described as a reduction in the use of corticosteroids and in the levels of CRP along the study. All adverse events were recorded during the study

Results: 64 patients were included with a mean follow-up of 57,2 weeks. At baseline the mean age was 47,8 years (8,9). 54,7% of patients were women and 45,3% were male. 31,3% were obese. Mean disease duration was 7,9 (5,0) years. 45,3% presented peripheric arthritis; 32,8% axial involvement; 31,3% enthesitis; 80% psoriasis. Patients were 45% bDMARDs-naïve; had a previous bDMARDs in 20,3% and $\geq$ than two bDMARDs in 34,4%. 30% of the patients had co-therapy with methotrexate and 29,7% of patients received corticosteroid therapy. Mean CRP was 7,9 (12,7) mg/L. The global probability of survival for UST was 96%, 83,9% and 60% at week 12, 24 and 52 respectively. High UST dosage was associated with favorable drug survival (at 52W: UST 45 mg=40,1%; UST 90 mg=75,8%; UST 45 to 90 mg*=88,9%) ($p=0,008$). The bDMARDs-naïve population also correlated with favorable UST survival (at 52W: bDMARDs-naïve=66,1% vs bDMARDs-experienced=56,7%), however no statistical significance was found ($p=0,196$). No difference in survival was observed among patients with or without axial involvement (W52: axial=58,2% vs non-axial=61,6; $p=0,869$). UST produced a reduction in the use of corticosteroids (30% vs 16%) and CRP levels (8,7 vs 7,7). Differences were greater in patients treated more than 28 weeks (maximum efficiency described for UST) (corticosteroids: 26% vs 16%; CRP levels: 8,5 vs 4,4). 4,9% of the patients suffered an AE. Most of them were non-serious AE: infections (3,3%) or headache (1,6%). The main cause of treatment discontinuation was lack of efficacy (30%), followed by primary failure (9,4%) and just a 3% due to AE

Conclusion: The persistence of UST was dose-dependent and greater for the UST 90mg dosage and for the population of bDMARD-naïve patients. Drug survival of UST in the population of patients with axial involvement seems similar to the population of patients without axial affection which provide evidence of the efficacy of the IL23 inhibition in the axial domain of PsA. UST decreased the use of corticosteroids and CRP levels along treatment. The security profile of UST was to the drug. Only few non-serious AE reported during this study

*UST 45 to 90mg, patients who change from UST 45 to UST 90mg dosage

References:

- [1] Ritchlin C. Efficacy and safety of the anti-IL-12/23 p40 monoclonal antibody, ustekinumab, in patients with active psoriatic arthritis despite conventional non-biological and biological anti-tumour necrosis factor therapy: 6-month and 1-year results of the phase 3. *Ann Rheum Dis* 2014;73:990–9
- [2] McInnes IB. Efficacy and safety of ustekinumab in patients with active psoriatic arthritis: 1 year results of the phase 3, multicentre, double-blind, placebo-controlled PSUMMIT 1 trial. *Lancet* 2013;382:780–9

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AB0827

IMPACT OF BASELINE BODY MASS INDEX ON THE EFFICACY AND SAFETY OF TOFACITINIB IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Obesity is highly prevalent in PsA (~45%)¹ and is associated with a reduced response to TNF inhibitors.² Tofacitinib is an oral Janus kinase inhibitor for the treatment of PsA.

Objectives: This post hoc analysis assessed tofacitinib efficacy and safety in patients (pts) with PsA by baseline (BL) body mass index (BMI) category.

Methods: Data were pooled from two placebo (PBO)-controlled, double-blind, Phase 3 studies in pts with active PsA and an inadequate response to $\geq$ 1 conventional synthetic DMARD (OPAL Broaden [12 months; NCT01877668]) or to $\geq$ 1 TNF inhibitor (OPAL Beyond [6 months; NCT01882439]).^{3,4} This analysis included pts randomised to tofacitinib 5 mg twice daily (BID), tofacitinib 10 mg BID or PBO, stratified by BL BMI: <25 kg/m², ≥ 25 – <30 kg/m², ≥ 30 – <35 kg/m², or ≥ 35 kg/m². Efficacy and safety were reported to Month (M)3. M3 efficacy outcomes included ACR20/50/70 and HAQ-DI responses, dactylitis and enthesitis resolution rates and changes from BL in HAQ-DI, Short Form-36 Version 2 (SF-36v2) Physical (PCS) and Mental Component Summary (MCS) scores, and Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F) scores. Safety outcomes included adverse events (AEs), such as cardiovascular (CV) events and changes in lipid levels and liver function tests (LFTs).

Results: This analysis included 710 pts; 43.8% were obese (BMI ≥ 30). At BL, 161 (22.7%) pts had a BMI <25 , 238 (33.5%) had a BMI ≥ 25 – <30 , 186 (26.2%) had a BMI ≥ 30 – <35 and 125 (17.6%) had a BMI ≥ 35 . Most pts were white (92.5–96.8%), middle-aged (mean: 44.5–51.2 yrs) and female (49.5–65.6%). Greater proportions of obese pts were from Russia/Eastern Europe (35.0%) and USA/Canada (31.8%), vs the rest of world. At BL, higher BMI correlated with an increased prevalence of metabolic syndrome (4.3% in BMI <25 to 76.0% in BMI ≥ 35) and CRP levels >2.87 mg/L (49.1% in BMI <25 to 84.0% in BMI ≥ 35). Higher proportions of pts (42.5–47.9%) in BL BMI categories <35 reported no prior biologic DMARD use, vs pts with a BL BMI ≥ 35 (33.6%). At M3, efficacy improvements were greater in tofacitinib-treated pts vs PBO-treated pts (Figure 1). In pts with a BL BMI ≥ 35 , a trend towards fewer pts responding was observed (Figure 1) and mean changes from baseline in SF-36v2 PCS and MCS and FACIT-F generally appeared lower (Figure 2) vs pts in lower BL BMI categories. Up to M3, the proportions of pts with AEs, and percentage change from BL in lipid levels and LFTs, were generally similar across all BL BMI categories. Three CV events were reported: non-fatal cerebrovascular accident, transient ischemic attack (both tofacitinib 5 mg BID, BMI ≥ 30 – <35) and coronary artery revascularisation (PBO; BMI ≥ 35). Limitations include the 3-month observation time, particularly for safety findings, thus longer observation times are warranted.

Conclusion: Regardless of BL BMI, tofacitinib demonstrated greater efficacy than PBO at M3 in pts with PsA. Similar to other advanced therapies,² reduced efficacy was generally observed in tofacitinib and PBO pts with a BL BMI ≥ 35 . Tofacitinib safety appeared consistent across all BL BMI categories.

References:

- [1] Labitigan et al. *Arthritis Care Res (Hoboken)* 2014;66:600-07.
- [2] Singh et al. *PLoS One* 2018;13:e0195123.
- [3] Mease et al. *NEJM* 2017;377:1537-50.
- [4] Gladman et al. *NEJM* 2017;377:1525-36.