Of 143 patients with complete data, glucocorticoid doses were increased or maintained after sampling in 45 and decreased or not prescribed in 98. Increased/maintained glucocorticoids were associated with higher IFN Score A ($p=0.003$, $n=113$), IFN Score B ($p=0.013$, $n=113$) and tetherin ($p=0.043$, $n=84$).

After adjustment for age, gender, pre and post-sampling glucocorticoid dose, substantive associations with flare remained for IFN Score A (OR=1.44/unit, 95% CI: 0.998 to 2.082, $p=0.051$) and tetherin (OR=3.135/1000 units, 95% CI: 1.179 to 8.335, $p=0.022$).

Conclusions: SLE patients in low disease activity with high IFN Score A, Score B and tetherin levels have an increased risk of disease activity and/or glucocorticoid exposure in the months that follow biomarker testing. There is a potential role for use of these IFN assays as predictive biomarkers in SLE in the future.

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THURSDAY, 14 JUNE 2018

The 'A-B-C' of PsA (Assessment, Biologicals, Co-morbidities)

OP0125

DAPSA AND ULTRASOUND SHOW DIFFERENT PERSPECTIVES OF PSORIATIC ARTHRITIS ACTIVITY: RESULTS FROM A LONGITUDINAL OBSERVATIONAL STUDY OF PATIENTS STARTING TREATMENT WITH BIOLOGIC DMARDS

S. Puksic, J. Sexton, P. Bolton-King, B. Michelsen, T.K. Kvien, H.B. Hammer. *Dept. of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway*

Background: Clinical disease activity in patients with psoriatic arthritis (PsA) may be assessed by DAPSA- a sum score of tender (of 68) and swollen (of 66) joints, CRP (mg/dl), patient's global assessment of disease activity (PGA) and patient's pain (0–10 VAS for both). Ultrasound (US) with grey scale (GS) and power Doppler (PD) is used to detect inflammation in joints as well as extra-articular structures. Discrepancies have previously been described between DAPSA and US.¹

Objectives: To explore the associations between DAPSA and US assessments of disease activity during follow-up of patients with PsA starting bDMARD treatment.

Methods: Patients with PsA were included when initiating bDMARDs, and examined at 0, 3, 6, 9 and 12 months including 66 swollen/68 tender joint counts (SJC/TJC), examiner's global assessment VAS (EGA), patient reported outcome measures (PROMs; PGA, patient's pain, RAID, MHAQ), laboratory measures (CRP, ESR, calprotectin) and US (46 joints, 8 flexor tendons, 14 entheses and 4 bursae with calculated GS and PD sum scores). DAPSA was calculated. PD sum score zero was defined as US remission. Generalised Estimating Equations (GEE) was used for longitudinal analysis.

Abstract OP0125 – Table 1 Median (IQR) of clinical, laboratory and US assessments during bDMARD treatment

	Baseline (n=47)	3 months (n=42)	6 months (n=36)	9 months (n=33)	12 months (n=31)
DAPSA	19.8 (13.9–30.9)	13.1 (4.9–20.9)	10.5 (2.6–18.7)	10.2 (3.5–20.5)	9.0 (2.4–20.6)
TJC (of 68)	7 (2–15)	2 (0–11)	1 (0–9)	2 (0–9)	2 (0–11)
SJC (of 66)	1 (0–3)	0 (0–1)	0 (0–1)	0 (0–0)	0 (0–0)
PGA (VAS 0–10)	5.2 (3.3–6.9)	2.8 (1.0–5.1)	2.0 (0.9–4.3)	3.3 (0.5–5.4)	2.7 (0.5–4.9)
PAIN (VAS 0–10)	5.4 (3.0–6.8)	3.1 (1.0–4.1)	2.8 (0.9–5.8)	2.6 (1.1–5.4)	2.2 (0.7–5.4)
MHAQ	0.50 (0.25–0.88)	0.25 (0.00–0.50)	0.13 (0.00–0.44)	0.25 (0.00–0.44)	0.13 (0.00–0.50)
RAID (0–10)	5.1 (4.0–6.0)	2.9 (0.9–5.1)	2.3 (1.3–4.7)	3.0 (0.9–5.2)	2.6 (1.1–4.9)
CRP mg/L	3 (1–7)	1 (1–3)	2 (1–4)	2 (1–3)	2 (1–3)
ESR mm/h	11 (6–17)	6 (3–12)	7 (3–11)	9 (3–13)	7 (4–14)
Calprotectin µg/L	826 (487–1202)	511 (307–741)	431 (305–669)	459 (267–1005)	432 (294–768)
EGA (VAS 0–10)	2.4 (2.0–2.8)	1.5 (1.0–2.2)	1.2 (0.8–1.6)	1.2 (0.8–1.6)	1.0 (0.5–1.2)
Sum score GS	4 (2–9)	2 (0–5)	2 (0–3)	3 (0–6)	4 (1–8)
Sum score PD	1 (0–4)	0 (0–2)	0 (0–2)	0 (0–2)	0 (0–2)

Abstract OP0125 – Figure 1. Baseline correlations with DAPSA

Results: 47 patients (mean (SD) age 48.4 (12.8) years, disease duration 12.7 (10.9) years, 60% women) were included (68% biologic-naïve). The figure depicts high correlation between DAPSA and subjective assessments at baseline, while PD sum score was more strongly associated with SJC ($r=0.49$, $p<0.001$), EGA ($r=0.29$, $p=0.05$) and DAS28 ($r=0.35$, $p=0.015$), but not with DAPSA ($r=0.21$, $p=0.16$) or PROMs. There was significant improvement at 3 months in all variables ($p=0.046$ – <0.001) (table 1). Longitudinally, TJC and patient's pain had strongest association with DAPSA (standardised β 0.86 vs 0.52, both $p<0.001$). DAPSA remission was obtained in 27%–33% of the patients and PD remission in 60%–70%, but their association was weak and not statistically significant (OR 1.4, 95% CI: 0.69 to 2.95, $p=0.33$).

Conclusions: In our observational PsA cohort DAPSA was mostly influenced by TJC and patient's pain and had no significant association with a comprehensive US examination. This finding indicates possible limitations of using DAPSA in T2T strategy in PsA.

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OP0126

EVALUATING LOW DISEASE ACTIVITY DEFINITIONS IN PSORIATIC ARTHRITIS USING ULTRASOUND

P. Bosch¹, R. Husic¹, A. Ficjan¹, J. Gretler¹, A. Lackner¹, W. Graninger¹, C. Duftner², J. Hermann¹, C. Dejaco^{1,3}. ¹Department of Rheumatology and Immunology, Medical University Graz, Graz; ²Department of Internal Medicine VI, Medical University Innsbruck, Innsbruck, Austria; ³Rheumatology Service, Hospital of Bruneck, Bruneck, Italy

Background: Cut offs for low disease activity (LDA) using psoriatic arthritis (PsA) specific composite scores have recently been proposed.^{1–3} Whether these definitions adequately reflect the absence of inflammation is unknown.

Objectives: To evaluate these definitions against a low level of activity according to ultrasound examination.

Methods: We performed a prospective study on 83 PsA patients undergoing clinical and ultrasound examinations at two study visits scheduled 6 months apart. LDA was assessed using the Disease Activity index for Psoriatic Arthritis (DAPSA ≤ 14), the Psoriatic Arthritis Disease Activity Score (PASDAS ≤ 3.2), the Composite Psoriatic Disease Activity Index (CPDAI ≤ 4), the Disease Activity Score 28 CRP (DAS28-CRP ≤ 2.8) and the Minimal Disease Activity criteria (MDA).

Ultrasound (US) evaluation was performed at 68 joints (evaluating synovia, peritendinous tissue, tendons and bony changes) and 14 entheses. Minimal ultrasound disease activity (MUDA) was defined as a Power Doppler (PD) score ≤ 1 , respectively at joints, peritendinous tissue, tendons and entheses.

Results: LDA was present in 33.7%–65.0% of patients at baseline and in 44.3%–80.6% at follow-up examination, depending on the criteria used. MUDA was observed in 16.9% at baseline and in 30% at follow-up.

At baseline only the DAPSA-LDA definition was useful to identify MUDA patients (78.6% of patients identified correctly), whereas at follow up >80% of MUDA

patients were correctly classified as LDA according to DAPSA, PASDAS, CPDAI and DAS28-CRP.

Only DAPSA (Sensitivity (S)=88.2%, Specificity (Sp)=40.5, $p=0.033$), PASDAS (S=88.2%, Sp=55.0%, $p=0.002$) and the MDA criteria (S=71.4%, Sp=67.3%, $p=0.003$) were able to discriminate patients with and without MUDA at follow-up. A global ultrasound inflammation subscore for joints and entheses (GUIS-j/e), containing the above mentioned US variables, was significantly higher in patients with active disease versus patients in LDA according to DAPSA ($p=0.002$) and PASDAS ($p=0.013$) at baseline and DAPSA ($p=0.007$), PASDAS ($p=0.001$), CPDAI ($p=0.021$) and the MDA criteria ($p<0.001$) at follow-up.

Conclusions: Of all tested LDA definitions, DAPSA was overall the most efficacious in differentiating between high and low ultrasound scores and better identified patients with MUDA as compared to the other tested scores.

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OP0127

CAN ACHIEVING MINIMAL DISEASE ACTIVITY (MDA) PREVENT PROGRESSION OF SUBCLINICAL ATHEROSCLEROSIS AND ARTERIAL STIFFNESS? A TWO-YEAR PROSPECTIVE COHORT STUDY IN PSORIATIC ARTHRITIS

T.H. Cheng¹, Q. Shang¹, E. K. M. Li¹, P.C.H. Wong¹, L.H.P. Tam¹, T.Y. Zhu², M. M. Chang¹, J.J.W. Lee³, C.K. Wong⁴, A.P.W. Lee¹, L.S. Tam¹. ¹Department of Medicine and Therapeutics; ²Department of Orthopaedics and Traumatology; ³School of Public Health; ⁴Department of Chemical Pathology, The Chinese University of Hong Kong, Hong Kong, Hong Kong

Background: PsA patients have higher CVD risk due to underlying inflammation. While achieving MDA was associated with articular benefits, its effect on CVD risk remained uncertain

Objectives: To investigate effect of achieving MDA on subclinical atherosclerosis and arterial stiffness

Methods: Subjects without CVD were recruited and received protocolised treatment aiming at MDA for 2 years. High-resolution ultrasound (for subclinical atherosclerosis) and arterial stiffness were assessed yearly. The primary objective was to investigate the effect of achieving MDA (MDA group) at 12 months on the progression of subclinical atherosclerosis (carotid intima-media thickness [IMT] and plaque) over 2 years. Secondary objectives were to compare 1) changes in arterial stiffness (branchial-ankle pulse wave velocity [PWV]) and augmentation index (AIX) over 2 years between MDA and non-MDA group; 2) changes in vascular outcomes in patients who could or could not achieve sustained MDA (sMDA, defined as achieving MDA from month 12 to 24). Carotid plaque progression was defined as an increase in number/region harbouring plaque compared with baseline

Results: 90 patients [male:52(58%); age: 50±11] were included. At 24 months, 62 (69%) were on csDMARDs, 20 (22%) were on anti-TNF- α and 8 (9%) were on Secukinumab. Proportion of patient achieved MDA increased significantly (baseline: 17%; 12 months: 64%; 24 months: 69%) after intensive treatment. Vascular outcomes were similar between MDA and non-MDA group (figure 1). 41 (46%) patients achieved sMDA. At baseline, a higher prevalence of subjects in the non-sMDA group were smokers, treated with NSAIDs and csDMARDs; fewer subjects were on bDMARDs, and they had higher disease activity compared with the sMDA group. 34 (38%) of them had plaque progression and prevalence was numerically higher in the non-sMDA group [22 (45%) vs 12 (29%), $p=0.13$]. Using multivariate analysis, achieving sMDA had protective effect on plaque progression [OR=0.27, 95% CI: 0.09 to 0.84, $p=0.03$] after adjustment of baseline difference (table 1). Achieving sMDA was also related to less progression of total plaque area (TPA), mean and maxIMT, PWV and AIX (table 1)

Conclusions: Effective suppression of inflammation by achieving sustained MDA may prevent subclinical atherosclerosis and arterial stiffness progression in PsA patients

Abstract OP0127 – Table 1. Multivariate analysis on vascular outcomes

Outcome	Baseline covariates	OR/ β	95% CI	p
Plaque progression	Age	1.07	1.01–1.13	0.02
	VAS Physician Global	0.96	0.93–0.99	0.02
	LDL-C	2.63	1.31–5.49	0.01
	bDMARDs use	0.11	0.01–0.65	0.02
TPA change	sMDA	0.27	0.09–0.85	0.02
	BMI	−0.43	−0.76, −0.10	0.01
	LDL-C	2.83	0.72–4.93	0.01
meanIMT change	sMDA	−3.92	−7.18, −0.66	0.02
	bDMARDs use	−0.03	−0.07, −0.00	0.03
	sMDA	−0.04	−0.07, −0.01	0.01
maxIMT change	Any plaque progression	−0.07	−0.13, −0.01	0.02
	sMDA	−0.07	−0.13, −0.01	0.03
PWV change	Age	4.95	0.92–8.99	0.02
	PWV	−0.48	−0.68, −0.29	<0.001
	sMDA	−71.4	−143.2, 0.23	0.05
			0.23	
AIX change	Sex	3.32	0.11–6.56	0.04
	sMDA	−3.06	−6.07, −0.05	0.05

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OP0128

THE PHENOTYPE OF AXIAL PSORIATIC ARTHRITIS: IS IT DEPENDENT ON HLA-B27 STATUS?

L. Coates¹, E. Alonso², X. Baraliakos³, F. Blanco Garcia⁴, J. Braun³, V. Chandran⁵, J.L. Fernandez-Sueiro⁶, O. FitzGerald⁶, P. Gallagher⁷, D. Gladman⁸, E. Gubar⁸, R. Korotaeva⁸, E. Loginova⁸, E. Lubrano⁹, J. Mulero¹⁰, J. Pinto-Tasende², T. Queiro¹¹, J. Sanz Sanz¹², A. Szentpetery⁷, P. Helliwell¹³. ¹NDORMS, University of Oxford, Oxford, UK; ²INIBIC, Complejo Hospitalario Universitario de A Coruña, A Coruña, Spain; ³Ruhr-University Bochum, Bochum, Germany; ⁴Department of Rheumatology, INIBIC, Complejo Hospitalario Universitario de A Coruña, A Coruña, Spain; ⁵Toronto Western Hospital, Toronto, Canada; ⁶Dept Rheumatology; ⁷St Vincent's University Hospital, Dublin, Ireland; ⁸Nasonova Research Institute of Rheumatology, Moscow, Russian Federation; ⁹Università degli Studi del Molise, Campobasso, Italy; ¹⁰Hospital Puerta de Hierro, Majadahonda, Madrid; ¹¹Hospital Universitario Central de Asturias, Oviedo; ¹²Hospital Universitario Puerta de Hierro, Majadahonda, Madrid, Spain; ¹³University of Leeds, Leeds, UK

Background: Up to 50% of patients with psoriatic arthritis (PsA) have associated spinal involvement similar to ankylosing spondylitis (AS). HLA-B27 positivity is lower in PsA compared to other SpA which may affect the disease phenotype.

Objectives: Our aim was to compare axial phenotype in PsA patients with and without HLA-B27 with AS patients.