AB0840

INFLUENCE OF PSORIATIC ARTHRITIS (PSA) ON BONE LOSS AND ANALYSIS BETWEEN AXIAL AND PERIPHERAL PSA IN JAPANESE PATIENTS

S. Tsuji¹, T. Tomita², M. Higashiyama^{3,4}, T. Noguchi^{1,1}, T. Mouri^{3,4}, J. Hashimoto¹. ¹Osaka minami medical center, Department of Orthopedics/ Rheumatology, Osaka, Japan; ²Department of Orthopedic Biomaterial Science, Osaka University Graduate School of Medicine, Osaka, Japan; ³Nippon Life Hospital, Osaka, Japan; ⁴Nippon Life Hospital, Osaka, Japan

Background: Osteoporosis is one of the major comorbidities in patients with psoriasis and psoriatic arthritis (PsA). It has been reported that PsA induces fragility bone structure¹ and high risk of osteoporosis². However, there is no report about relationship between psoriatic arthritis and osteoporosis in Japanese patients and its mechanism has not been elucidated.

Objectives: The objective of this study is to investigate influence of PsA on bone mineral density (BMD) and its mechanism including analysis between axial and peripheral PsA in Japanese patients.

Methods: This study was retrospective study. We examined 58 cases of PsA and 29 cases of RA that underwent DXA tests at our facility from January 2017 to July 2019 (Table 1). The axial PsA was classified as axial SpA using the ASAS classification criteria. First, we investigated influence of PsA containing both axial (n=30, 19 males, 11 females, mean age: 50.6 years) and peripheral (n=28, 19 males, 9 females, mean age: 58.0 years) subtypes on BMD measured by dual-energy X-ray absorptiometry. Second, we measured serum bone metabolism markers (P1NP: type 1 procollagen-N-propeptide, TRACP-5b: tartrate-resistant acid phosphatase 5b) and bone remodeling effector molecules (Dkk1: Dickkopf1, sclerostin, 25(OH)D: 25-hydroxyvitamin D) to elucidate differences in BMD between axial and peripheral PsA. Furthermore, rheumatoid arthritis (RA) (n=29, 2 males, 27 females, mean age: 66.2 years), as a reference disease, was also evaluated for comparison with axial and peripheral PsA. Osteoporosis and Osteopenia were defined as T-score ≤ -2.5 or %YAM $\leq 70\%$, -1.0< T-score >-2.5 or 80<%YAM $>70\%$ respectively.

Results: 58 patients with PsA indicated low T-score, Z-score and %YAM in both lumbar spine and proximal femur (Table 1). Axial PsA and peripheral PsA showed osteoporosis in 16.7% and 35.7%, and osteopenia in 20.0% and 32.1%, respectively, despite the fact that there were many middle-aged men. Comparison between axial and peripheral PsA, axial PsA showed higher BMD than peripheral PsA. In bone remodeling makers, P1NP in both PsA were almost same, but TRACP-5b, bone resorption marker, in axial PsA was lower than that in peripheral PsA (Table 2). In bone remodeling influencer molecules, Dkk1, and sclerostin in axial PsA was slightly higher than those in peripheral PsA, whereas 25(OH)D is almost same as the both PsA. On the other hand, RA also indicated low T-score and %YAM in both lumbar spine. P1NP in RA showed slightly lower, but TRACP-5b and Homocysteine in RA higher than those in axial and peripheral PsA. Dkk1 and sclerostin in RA were slightly lower than those in both PsA.

Conclusion: Peripheral PsA indicated more severe bone loss than axial PsA in our study. There were some differences in bone remodeling markers and bone remodeling effector molecules between axial and peripheral PsA, but the relationships between BMD and these parameters were not confirmed. Further studies are needed to elucidate bone loss mechanism in these PsA.

(Table 1) Characteristic DATA

	Axial-PsA	Peripheral-PsA	RA
n	30	28	29
male (%)	63.3%	67.9%	6.9%
ave.age (y.o.)	50.6 $\pm$ 11.1	58.0 $\pm$ 10.1	66.2 $\pm$ 12.4
Osteoporosis (%)	16.70%	35.70%	41.40%
Osteopenia (%)	20.00%	32.10%	13.80%
DXA			
Lumbar spine			
T-score	-0.77 $\pm$ 1.39	-1.95 $\pm$ 1.04	-1.84 $\pm$ 1.31
Z-score	-0.24 $\pm$ 1.63	-0.97 $\pm$ 1.36	-0.31 $\pm$ 1.24
%YAM	91.9 $\pm$ 14.4	79.3 $\pm$ 11.2	79.9 $\pm$ 14.6
Proximal femur			
T-score	-0.81 $\pm$ 1.13	-1.14 $\pm$ 0.64	-1.20 $\pm$ 1.32
Z-score	-0.07 $\pm$ 1.01	-0.19 $\pm$ 0.81	0.36 $\pm$ 0.94
%YAM	89.3 $\pm$ 15.1	84.9 $\pm$ 8.57	84.5 $\pm$ 16.9
Fracture			
vertebral (n)	2	3	3
Hip (n)	2	0	0

(Table 2) Laboratory DATA and Treatment Drugs

	Axial PsA (n=30)	Peripheral PsA (n=28)	RA (n=29)
Laboratory DATA (ave.)			
Homocysteine, g/mol	13.364 $\pm$ 4.095	14.525 $\pm$ 4.986	368.098 $\pm$ 1900.195
Dkk-1	3782.11 $\pm$ 1575.975	3427.29 $\pm$ 880.759	3093.52 $\pm$ 1427.96
SOST/Sclerostin	153.06 $\pm$ 69.38	134.52 $\pm$ 106	89.68 $\pm$ 62.96
TRACP-5b, mU/dL	283.5 $\pm$ 113.7	355 $\pm$ 136.7	410.6 $\pm$ 179.1
ucOC, ng/mL	2.01 $\pm$ 1.23	4.91 $\pm$ 4.85	3.33 $\pm$ 3.08
25(OH)D, ng/mL	16.08 $\pm$ 6.94	17.04 $\pm$ 6.06	13.90 $\pm$ 5.34
total P1NP, ng/mL	50.62 $\pm$ 19.85	52.74 $\pm$ 33.97	39.03 $\pm$ 22.73
CRP, mg/L	8.20 $\pm$ 16.4	6.6 $\pm$ 20.9	6.6 $\pm$ 10.2
Treatment Drugs			
TNFInhibitor (n)	6	2	3
IL-17inhibitor (n)	2	1	0
IL-12/23inhibitor (n)	2	1	0
IL-6inhibitor (n)	0	0	2
Bisphosphonate (n)	3	6	15
Vitamin D (n)	4	4	15
Teriparatide (n)	0	1	0
Prednisolone use (n,mg/d)	0, -	1, ave.3.0mg/d	10, ave.4.0mg/d

References:

- [1] Zhu TY, et al. Osteoporosis Int. 2015; 26:261–272.
- [2] Kathuria R, et al. J Am Acad Dermatol. 2017;76:1045-53.

Disclosure of Interests: Shigeyoshi Tsuji Grant/research support from: Eli Lilly, Speakers bureau: AbbVie, Asahi Kasei, Chugai, Daiichi Sankyo, Eli Lilly, Eisai, Mitsubishi Tanabe, Celgene, and Novartis Pharma K.K., Tetsuya Tomita Consultant of: Eli Lilly and Company, Mari Higashiyama: None declared, Takaaki Noguchi: None declared, Toshikazu Mouri: None declared, Jun Hashimoto Speakers bureau: AbbVie, Asahi Kasei, Chugai, Daiichi Sankyo, Eli Lilly, Eisai, Mitsubishi Tanabe, Celgene, and Novartis Pharma K.K.

DOI: 10.1136/annrheumdis-2020-eular.2826

AB0841

TARGET OUTCOMES IN PSA: SIMULTANEOUS ACHIEVEMENT OF ACR50-PASI100 AND BEYOND: INSIGHTS FROM SPIRIT-H2H AT WEEK 24

J. S. Smolen¹, F. Behrens², S. Liu Leage³, C. Sapin³, I. De La Torre³, G. Meszaros³, G. Schett⁴, L. Gossec⁵, A. Ostor⁶, B. Combe⁷, F. Van den Bosch⁸. ¹Medical University of Vienna, Vienna, Austria; ²Goethe University and Fraunhofer IME-TMP, Frankfurt, Germany; ³Eli Lilly and Company, Indianapolis, United States of America; ⁴Friedrich-Alexander-Universität Erlangen-Nuremberg, Erlangen, Germany; ⁵Sorbonne Université, Paris, France; ⁶Cabrini Medical Center, Monash University, Malvern, Australia; ⁷Montpellier University, Montpellier, France; ⁸Ghent University Hospital, Ghent, Belgium

Background: Psoriatic Arthritis (PsA) treatment should aim to achieve robust improvement of arthritis as well as control of extra-articular manifestations like the skin. SPIRIT-H2H evaluated the efficacy of ixekizumab (IXE) and adalimumab (ADA) in patients with active PsA and psoriasis, and naïve to biologic Disease-Modifying Anti-Rheumatic Drugs (bDMARDs). At week 24 (W24), IXE showed superiority to ADA in simultaneous achievement of ACR50 and PASI100 as well as significant improvement of treat-to-target and other extra-articular outcomes.

Objectives: To examine and to compare PsA efficacy outcomes in patients beyond achievement of the primary endpoint of the SPIRIT-H2H trial at W24, irrespective of treatment allocation.

Methods: All patients recruited had active PsA (defined as tender joint count $\geq 3/68$, swollen joint count $\geq 3/66$ and body surface area [BSA] $\geq 3\%$), and inadequate response to conventional synthetic (cs)-DMARDs. Patients were randomised 1:1 to open-label, assessor-blinded IXE or ADA. We conducted post-hoc analysis of SPIRIT-H2H (NCT03151551), categorizing patients into four independent groups based on the achievement of the primary outcome (ACR50 & PASI100), ACR50 only, PASI100 only or none of them after 24 weeks of treatment. Statistical analyses consisted of mixed model for repeated measurement and logistic regression models using non-response imputation.

Results: At week 24, patients reaching simultaneously ACR50 and PASI100 had a statistically significant higher response in most treat-to-target endpoints than those meeting ACR50 only ($p<0.05$). In this latter group, a high response rate was observed in ACR70, MDA, DAPSA remission and PASI90 response (48.9%, 60.6%, 35.1%, 36.2%, respectively). In patients that did not achieve either ACR50 or PASI100, up to 1/3 of the patients did achieve ACR20, DAPSA score ≤ 14 , or no physical impairment.

Table. Efficacy Endpoints at W24

	ACR50 & PASI100	ACR50 only	PASI100 only	Neither ACR50 nor PASI100
n=181	n=94	n=121	n=170	
ACR20	100.0 ^{a,b,c}	100.0	53.7	34.7
ACR70	64.6 ^{a,b,c}	48.9	0.0	0.0
MDA	75.7 ^{a,b,c}	60.6	23.1	12.4
VLDA	32.6 ^{a,b,c}	13.8	3.3	1.8
DAPSA LDA or Remission (≤ 14)	92.3 ^{a,b,c}	81.9	43.0	28.8
DAPSA Remission (≤ 4)	44.8 ^{b,c}	35.1	6.6	2.4
HAQ-DI score ≤ 0.5	75.7 ^{b,c}	64.9	30.6	27.4
PASI75	100.0 ^{a,c}	60.6	100.0	37.1
PASI90	100.0 ^{a,c}	36.2	100.0	14.7
SF-36 PCS change from baseline [§]	12.3 $\pm$ 0.53 ^{b,c}	12.3 $\pm$ 0.74	5.4 $\pm$ 0.66	4.0 $\pm$ 0.55

Data are presented as %; [§]mean $\pm$ standard error.

^a $p<0.05$ vs. ACR50 only; ^b $p<0.05$ vs. PASI100only; ^c $p<0.05$ vs. Neither ACR50 nor PASI100. ACR, American College of Rheumatology; DAPSA, Disease Activity in Psoriatic Arthritis; HAQ-DI, Health Assessment Questionnaire Disability Index; LDA, Low Disease Activity; MDA, Minimal Disease Activity; PASI, Psoriasis Area Severity Index; VLDA, Very Low Disease Activity.

Nine patients with active PsO and BSA $\geq 3\%$ were assessed as PASI=0 at baseline, a medical inconsistency that was resolved using medical judgement. These patients were considered PASI100 responders if PASI=0 and BSA=0 at post baseline visits.

Conclusion: Reflecting the complexity of PsA, different degrees of improvement were observed across all treat-to-target outcomes with greater improvements in patients that met ACR50 response regardless of skin resolution. These findings at week 24 need to be confirmed with a longer duration of treatment.

Disclosure of Interests: Josef S. Smolen Grant/research support from: AbbVie, AstraZeneca, Celgene, Celltrion, Chugai, Eli Lilly, Gilead, ILTOO, Janssen, Novartis-Sandoz, Pfizer Inc, Samsung, Sanofi, Consultant of: AbbVie, AstraZeneca, Celgene, Celltrion, Chugai, Eli Lilly, Gilead, ILTOO, Janssen, Novartis-Sandoz, Pfizer Inc, Samsung, Sanofi, Frank Behrens Grant/research support from: Pfizer, Janssen, Chugai, Celgene, Lilly and Roche, Consultant of: Pfizer, AbbVie, Sanofi, Lilly, Novartis, Genzyme, Boehringer, Janssen, MSD, Celgene, Roche and Chugai, Soyi Liu League Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Christophe Sapin Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Inmaculada De La Torre Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Gabriella Meszaros Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, Georg Schett Speakers bureau: AbbVie, BMS, Celgene, Janssen, Eli Lilly, Novartis, Roche and UCB, Laure Gossec Grant/research support from: Lilly, Mylan, Pfizer, Sandoz, Consultant of: AbbVie, Amgen, Biogen, Celgene, Janssen, Lilly, Novartis, Pfizer, Sandoz, Sanofi-Aventis, UCB, Andrew Ostor Consultant of: MSD, Pfizer, Lilly, Abbvie, Novartis, Roche, Gilead and BMS, Speakers bureau: MSD, Pfizer, Lilly, Abbvie, Novartis, Roche, Gilead and BMS, Bernard Combe Grant/research support from: Novartis, Pfizer, Roche-Chugai, Consultant of: AbbVie; Gilead Sciences, Inc.; Janssen; Eli Lilly and Company; Pfizer; Roche-Chugai; Sanofi, Speakers bureau: Bristol-Myers Squibb; Gilead Sciences, Inc.; Eli Lilly and Company; Merck Sharp & Dohme; Pfizer; Roche-Chugai; UCB, Filip van den Bosch Consultant of: AbbVie, Celgene Corporation, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer, and UCB, Speakers bureau: AbbVie, Celgene Corporation, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer, and UCB

DOI: 10.1136/annrheumdis-2020-eular.2884

AB0842

DISCOVERY OF ARTHRITIS IN PSORIASIS FOR EARLY RHEUMATOLOGIC REFERRAL (DAPPER): A CROSS-SECTIONAL STUDY

T. Van Hal¹, M. Mulder¹, M. Wenink¹, M. Pasch², J. Van den Reek², E. De Jong². ¹Sint Maartenskliniek, Rheumatology, Nijmegen, Netherlands; ²Radboudumc, Dermatology, Nijmegen, Netherlands

Background: One in three patients with psoriasis (Pso) will develop psoriatic arthritis (PsA) (1). When untreated, this can lead to disability and irreversible joint damage (2). Current screening methods are mostly based on questionnaires. These lack specificity and sensitivity (3,4). Thus, a significant portion of PsA patients remains undetected.

Objectives: Our main objective is to ascertain the prevalence of PsA in a cohort of Pso patient, treated at a dermatology outpatient clinic. Secondary, we wish to make a referral tool for dermatologist to detect patients suspected of PsA.

Methods: A sample of 300 patients, stratified for current skin therapy (topical, systemic non-biologic, biologic), will be screened by a rheumatology resident for PsA signs and symptoms. When PsA is suspected, patients are referred to a rheumatologist for confirmation. We gather information about demography, treatment (past and current) and comorbidity. On top of that, we gather data on disease specifics (age of onset, disease duration, severity). We store biomaterials and DNA. Eventually, all these data will be used to form a more specific prediction model which can be used at the dermatology department for more efficient referral.

Results: We will present preliminary data of the first 100 patients. In this cohort, we found 14 patients with known PsA. 10 patients were suspected of (previously undiagnosed) PsA, and were referred to a rheumatology clinic. Three cases were confirmed, and 4 are still under analysis. This makes the prevalence of PsA in Pso 17-21%. Of these three new cases, one was treated with topical therapy only, one was treated with a biologic, and one received targeted therapy. In the patients with PsA, we found a higher amount of men. On top of that, we found a trend towards more intensive therapy. This may be due to indication bias, were the presence of arthritis may lead to a more aggressive treatment. Interestingly, 2 of the 3 previously undiagnosed PsA patients were treated with a biologic for their skin symptoms.

Conclusion: Preliminary data of the DAPPER study reveal that the prevalence of confirmed PsA in Pso patients is 17%. If all suspected PsA are confirmed, this rises to 21%. Even under systemic biologic treatment, arthritis can still be active.

References:

- [1] Mease PJ, Gladman DD, Papp KA et al. Prevalence of rheumatologist-diagnosed psoriatic arthritis in patients with psoriasis in European/North American dermatology clinics. *J Am Acad Dermatol* 2013;69(5):729-735.
- [2] Kane D, Stafford L, Bresnihan B, FitzGerald O. A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. *Rheumatology (Oxford)* 2003;42(12):1460-1468.
- [3] Coates LC, Aslam T, Al BF et al. Comparison of three screening tools to detect psoriatic arthritis in patients with psoriasis (CONTEST study). *Br J Dermatol* 2013;168(4):802-807.
- [4] Haroon M, Kirby B, FitzGerald O. High prevalence of psoriatic arthritis in patients with severe psoriasis with suboptimal performance of screening questionnaires. *Ann Rheum Dis* 2013;72(5):736-740.

Disclosure of Interests: Tamara van Hal Speakers bureau: Lilly Eli, Michelle Mulder: None declared, Mark Wenink: None declared, Marcel Pasch: None declared, Juul Van den Reek Speakers bureau: Abbvie, Eli Lilly, Elke De Jong Grant/research support from: Abbvie, Janssen Pharmaceutica, Consultant of: AbbVie, Janssen Pharmaceutica, Novartis, Eli Lilly and Company, Celgene, and Leo Pharma., Speakers bureau: AbbVie, Janssen Pharmaceutica, Novartis, Eli Lilly and Company, Celgene, and Leo Pharma.

DOI: 10.1136/annrheumdis-2020-eular.4745

AB0843

PSORIATIC ARTHRITIS PATIENTS ACHIEVING DAPSA REMISSION/LOW DISEASE ACTIVITY HAVE BETTER LONG-TERM FUNCTIONAL OUTCOMES

L. Vargas Cruz¹, J. Boechat Farani², J. Rabello Costa³, F. Menegat⁴, J. V. Andrade Águas⁵, B. Ruschel⁶, A. A. Gasparin², C. Brenol², C. Kohem², P. Palominos² on behalf of Rheumatology Department. Hospital de Clínicas de Porto Alegre, Brazil Universidade Federal do Rio Grande do Sul, Brazil. ¹Hospital de Clínicas de Porto Alegre, Rheumatology, Porto Alegre, Brazil; ²Hospital de Clínicas de Porto Alegre, Rheumatology, Porto Alegre, Brazil; ³Universidade Federal do Rio Grande do Sul, Medicine, Porto Alegre, Brazil; ⁴Hospital de Clínicas de Porto Alegre, Pesquisa Clínica, Porto Alegre, Brazil; ⁵Universidade Federal do Rio Grande do Sul, Medicine, Porto Alegre, Brazil; ⁶Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil

Background: Patients with psoriatic arthritis (PsA) experience substantial functional impairment, which impacts on health-related quality of life.¹ Evidence from randomized clinical trials (RCTs) suggests better patient-reported functional outcomes when lower disease activity is achieved.^{2,3,4}

Objectives: To evaluate the impact of achieving DAPSA remission (REM) or low disease activity (LDA) on long term function measured by HAQ-DI. To verify predictors of achieving a minimum clinically important difference (MCID) in HAQ-DI (≤ -0.35).

Methods: This is a longitudinal analysis of a real-life retrospective cohort. Inclusion criteria were adult patients fulfilling CASPAR criteria for PsA with at least 4 years of follow-up in the PsA Clinic. Demographic and clinical data were extracted from electronic medical records. Comparison of HAQ-DI variation between patients with DAPSA REM/LDA and those with moderate/high disease activity was performed using generalized estimating equation (GEE), adjusted by Bonferroni test. Correlation between HAQ-DI and DAPSA was analyzed by Spearman correlation method. A multivariate hierarchical regression model was applied in order to evaluate predictors of achieving a MCID in HAQ-DI scores.

Results: Seventy-three patients were included in the analysis, of which 58.9% were female, with a median (25/75th) of 8 (3-15) years since PsA diagnosis and a mean follow up time of 6.2±1.2 years. In total, 37% of patients (N=27) presented a MCID in HAQ-DI during the follow-up. Function measured by HAQ-DI was determined by PsA disease activity measured by DAPSA (interaction test: $p < 0.0001$) (Figure 1). A moderate and statistically significant correlation between Δ DAPSA and Δ HAQ-DI was observed ($r_s = 0.60$; $p < 0.001$) (Figure 2), demonstrating that a decrease in PsA disease activity was associated to improvement in function. Only patients in DAPSA REM demonstrated a constant declining in HAQ-DI scores during the 6 years of follow-up (Figure 1). White ethnicity and older age at baseline were predictors for not achieving MCID in HAQ-DI (RR 0.33 95% CI 0.16-0.67, $p=0.002$ and RR 0.96 95% CI 0.93-0.98, $p<0.0001$, respectively), while higher scores of HAQ-DI at baseline were predictors of achieving a MCID (RR 1.71 95%CI 1.12-2.60, $p=0.013$).