an average of 6 ± 7.7 years, and had received an average of 1.26 ± 1.45 previous biologic DMARD. 51.5% were receiving Ustekinumab in a combined therapy with DMARD (most of them Methotrexate) and 48.5% were in monotherapy. The survival rates at 6 and 12 months were 85% and 74.6% respectively. Comparing the subgroups, the naïve patients with biologic DMARDs presented higher survival rates at 6 (91.6% vs 80.7%) and at 12 months (85.1% vs 66.7%), a statistically significant difference with the group that received previous biologic DMARD ($p=0.036$). The patients in combined therapy with DMARDs presented higher rates of survival than the patients in monotherapy with Ustekinumab (90% vs 80%; 78% vs 71.1% at 6 and 12 months respectively), although the differences were not statistically significant. The main reason for discontinuation was the decrease of efficacy (14 patients; 21.2%), mostly in patients who had received previous treatment with biologic DMARD. 5 patients (7.6%) did not continue due to AE (2 due to relapsing herpes zoster; 1 patient deceased, with a personal history of neoplasms and a previous treatment with anti-TNF; and 2 had reactions at the injection site).

Conclusions: Ustekinumab is a safe drug, presenting high rates of drug retention, especially in patients who have not received any previous biologic therapy.

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AB0934 OBESITY IN PATIENTS WITH PSORIATIC ARTHRITIS IN OUR AREA

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Background: Obesity is a comorbid condition in patients with psoriasis which tends to be less common in patients with psoriatic arthritis (PsA). In the general population, obesity is associated with increased inflammatory load and vascular risk, and with hypovitaminosis D.

Objectives: To explore the relationship between obesity and disease activity, vascular damage, serum concentrations of vitamin D (25OHD) and bone mineral density (BMD) in patients with PsA.

Methods: Descriptive cross-sectional study. Patients with PsA patients and peripheral joint involvement were consecutively included. Demographic (age, sex), clinical [duration of the disease, BMI (body mass index), DAS28] and analytical (25OHD, CRP, ESR) variables were collected. Patients with a BMI ≥ 30 kg/m²; were considered obese, and we considered vitamin D deficiency when 25OHD < 20 ng/ml and vitamin D insufficiency when 25OHD $20-30$ ng/ml. Within a period of 3 months, atheroma plaque and intima media thickness (IMT) measurement was performed by ultrasonography of the carotid arterial tree using an Esaote MyLab70XVG with a 7–12 MHz linear transducer and an automated program measuring IMT through radiofrequency (Quality intima media Thickness in real time, QIMT). Pulse wave velocity (PWV) was obtained by analysis of brachial pulse waves with an automated and validated system (Mobil O Graph). IMT > 900 μ and PWV ≥ 10 m/s were considered pathological. Measurement of BMD was performed using an HOLOGIC densitometer. Statistical analysis was performed with the SPSS 17.0 program.

Results: We included 108 patients, 60% women, with a mean age of 55.6 (SD 12.6) years and a mean duration of PsA of 19.5 (SD 28) years. The mean BMI was $27(\text{DE } 5.4)$ kg/m²; 35% of the patients being obese. The mean CRP, ESR and DAS28 were $6.6(\text{DE } 4.5)$ mg/L, $10.7(\text{DE } 11)$ mm/h and $2.32(\text{DE } 0.8)$, respectively. 28% of patients had 25OHD deficiency and 32% had 25OHD insufficiency. The mean values of the IMT and PWV were $715(\text{SD } 149)\mu$ and PWV $8(\text{SD } 1.8)$ m/s, respectively. 12% of patients had osteoporosis, and up to 39% had osteopenia. Obesity was associated with a higher PsA activity measured by CRP (8.5 vs 5.7 mg/L, $p=0.001$) and ESR (38.6 vs 8.7 mm/h, $p=0.04$), although DAS28 was not different between groups. We also observed differences between obese and non-obese regarding the mean values of IMT (770 vs 709μ , $p=0.046$) and PWV (8.9 vs 8 m/s, $p=0.021$), the presence of atheroma plaque nearly reaching statistical significance (39% vs 30% , $p=0.07$). No association was observed between obesity and 25OHD levels, BMD and DAS28.

Conclusions: obesity is linked to an increased inflammatory load and more vascular damage in our PsA patients. A strategy of tight control of obesity and other vascular risk factors should be implemented when monitoring PsA patients.

Disclosure of Interest: None declared

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AB0935

CANADIAN ADALIMUMAB POST-MARKETING OBSERVATIONAL EPIDEMIOLOGICAL STUDY ASSESSING THE EFFECTIVENESS OF ADALIMUMAB VS NON-BIOLOGIC DMARDS IN PSORIATIC ARTHRITIS (COMPLETE-PSA): 12-MONTH EFFECTIVENESS DATA

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Background: To date, observational studies comparing the effectiveness of adalimumab (ADA) to non-biologic DMARDs (nbDMARD) in psoriatic arthritis (PsA) patients failing prior treatment are scarce. COMPLETE-PSA is a Canadian post-marketing observational study which assessed real-life effectiveness of ADA and non-biological therapies (NSAIDs and DMARDs) in PsA management following initial treatment failure.

Objectives: This analysis aimed to describe the baseline demographic and disease parameters of patients initiating nbDMARD or ADA and compare the 12 month real-life effectiveness of both treatments.

Methods: Patients eligible for COMPLETE PsA are anti-TNF α naïve adults, with active PsA who require change in their treatment regimen, per the judgment of the treating physician. In the current analysis patients enrolled during Jul/2011–Jun/2016 were included. Outcome measures analysed were: DAS28, SF-12, DLQI, presence of extra-articular manifestations (EAMs; enthesitis and dactylitis), psoriasis BSA, achievement of modified MDA (defined as achievement of [i] 4 of 6 (MDA 1) and [ii] 5 of the following 6 criteria (MDA 2): TJC ≤ 1 , SJC ≤ 1 , BSA $\leq 3\%$, pain VAS ≤ 15 mm, PtGA ≤ 20 and HAQ ≤ 0.5), modified remission (defined as SJC=0, TJC=0, absence of enthesitis and dactylitis, BSA $\leq 3\%$ and HAQ ≤ 0.5), DAPSA LDA (≤ 14), and DAPSA remission (REM; ≤ 4). Analyses were conducted by initial group assignment (intent-to-treat approach).

Results: 406 patients were included (nbDMARD n=146, ADA n=260). Baseline demographics were comparable between treatment groups. However, patients initiating ADA were more likely to be unemployed (47.3% ADA vs 34.9% nbDMARD, $p=0.015$), had higher DAS28 (4.8 vs 4.4 , $p=0.002$) and total DLQI score (6.1 vs 4.3 , $p=0.007$), and were more likely to have BSA $\geq 3\%$ (44.6% vs 35.0% , $p=0.063$) and high DAPSA disease activity (50.8% vs 32.3% , $p=0.015$). A higher proportion of nbDMARD patients had dactylitis (36.1% vs 25.3% , $p=0.023$). No differences were observed between groups in enthesitis, overall EAMs, or QoL at baseline.

Upon 12 months of treatment, mean adjusted DAS28 (2.6 vs 3.4 , $p<0.001$) and DLQI (2.2 vs 2.9 , $p=0.530$) scores, but not SF-12, were lower in the ADA group. Furthermore, although statistical significance was not always met, patients treated with ADA had lower DAPSA score ($p=0.025$) (LDA/REM: 64.9% vs 58.6% ; REM: 37.7% vs 17.1%), were more likely to be in modified remission (14.7% vs 9.7% , $p=0.311$), and to have mMDA (mMDA 1: 15.6% vs 12.6% , $p=0.529$; mMDA 2: 17% vs 11.5% , $p=0.253$) and BSA $< 3\%$ (89.3% vs 83.9% , $p=0.207$). Also, EAM prevalence decreased in both groups but was significantly lower in the ADA group (17.4% vs 35.8% , $p<0.001$).

Over time, 9.6% of ADA patients initiated another biologic and 32.2% of patients in the nbDMARD group initiated biologic treatment ($p<0.001$).

Conclusions: PsA patients initiating ADA in Canadian routine clinical care have significantly greater baseline disease severity compared with those initiating nbDMARDs. However, 12 month ADA treatment improved disease control and EAMs. DAPSA-REM evaluation seems more sensitive than mMDA in differentiating both populations.

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