POS0121

RESPONSE OF EOSINOPHILIC GRANULOMATOSIS WITH POLYANGIITIS TO MEPOLIZUMAB ACCORDING TO DISEASE MANIFESTATIONS. A SINGLE CENTRE EXPERIENCE

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Background: Eosinophilic granulomatosis with polyangiitis (EGPA) is a relapsing disease with frequent glucocorticoid (GC) dependence. Mepolizumab (MEPO) has been demonstrated to reduce flares and spare GC. However, EGPA is a heterogeneous disease and the effects of MEPO on specific disease manifestations has not been completely delimited.

Objectives: To analyze the impact of MEPO on manifestations derived from small-vessel vasculitis, ENT symptoms, asthma, eosinophilic tissue infiltration and ANCA status in a single-centre cohort of EGPA patients.

Methods: Medical chart of EGPA patients treated with MEPO were reviewed to describe demographics, clinical characteristics, steroid dose at the initiation of MEPO and during follow-up, flares, disease activity, damage accrual and laboratory results.

Results: Among 52 EGPA patients regularly controlled in our department, 11 patients were treated with MEPO. MEPO was prescribed when a) patients required prednisone (PDN) at ≥ 7.5 mg/d to maintain stability, or b) when maintained with < 7.5 mg/d, presented at least 4 exacerbations/year requiring an increase in PDN dose. 6 were males and 5 females, with a mean age of 54 years at MEPO initiation. Baseline characteristics of the patients and course under treatment are presented in Table 1. ENT involvement, followed by asthma and eosinophil-related tissue-infiltration (ETI) were the most common symptoms when prescribing MEPO. Regarding treatment, patients received MEPO at 100-300mg SC monthly. The definition of flare was the same used in the MIRRA trial¹. The mean time of treatment with MEPO was 34 months. All patients achieved a BVAS score of 0 points at 12 months or earlier. In general, patients reduced the number of flares, which tended to be milder, and all related to asthma or ENT manifestations. All improved their asthma control, but 3 of them persisted with recurrent ENT symptoms in spite of treatment with MEPO. None of them had vasculitic manifestations (cutaneous, neurological, gastrointestinal, renal) manifestations during treatment. All patients were able to taper their PDN dose to ≤ 5 mg/day or less, except 3 patients. Of the 3 patients who required ≥ 5 mg/d, 1 had severe asthma, but diminished the previous PDN dose (22.5 mg/d pre-MEPO, 10 mg/d currently) and the yearly rate of flares (8.2 pre-MEPO, 0.64 under-MEPO). The other one notably improved his asthma, but had ENT symptoms that responded unsatisfactorily to MEPO and required a maintenance PDN dose of 7.5 mg/d. The last one, improved her asthma control and was able to begin PDN tapering, but persisted with ENT symptoms. Regarding damage accrual, 6 patients remained stable during treatment, and 5 worsened. Two of three ANCA positive patients remained positive in spite of treatment.

Table 1.

	Baseline characteristics at diagnosis	At mepolizumab initiation	After mepolizumab (last follow-up)
Age, median (range) years	49 (23-67)	54 (35-69)	-
Male/Female, n (%)	6/5 (54.5%/45.5%)	-	-
BVAS, median (range)	11 (2-20)	2 (0-6)	0
FFS, value (n, %)	1 (1, 9.1%)	-	-
VDI, mean (range)	-	1.7 (0-5)	2.3 (0-5)
Asthma, n (%)	11 (100%)	3 (27.3%)	0 (0%)
ENT, n (%)	10 (90%)	4 (36.4%)	3 (27.3%)
SVV, n (%)	4 (36.4%)	0 (0%)	0 (0%)
ETI signs/symptoms, n (%)	7 (63.6%)	2 (18.2%)	0 (0%)
Constitutional symptoms, n (%)	4 (36.4%)	0 (0%)	0 (0%)
Eosinophils, mean (range) cells $\times 10^9$	5500 (600-8850)	240 (0-600)	55 (0-200)
ANCA positivity (IIF)	7 (77.8%)	3	2
Anti-MPO titers, mean (range)	286 (93-740)	88 (3-739)	7 (3-37)
Yearly rate of flares	-	1.75	0.51
Immunosuppressants, n	-	3	1
Prednisone dose, mg/d (range)	8 (7.5-25)	11.4 (5-22.5)	5.125 (0-10)

Conclusion: MEPO was effective for the treatment of patients with EGPA, with a reduction in the number and severity of flares and a decrease in PDN doses. A worse response of ENT involvement was observed. No vasculitic flares were observed in spite of GC reduction. Mepolizumab did not prevent damage accrual during the treatment period.

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Central pain and other extra-articular associations to KOA

POS0122

THE ASSOCIATIONS BETWEEN DISEASE MODIFYING ANTI-RHEUMATIC DRUGS AND INCIDENT, AND PROGRESSION OF, RADIOGRAPHIC HAND OSTEOARTHRITIS IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Hand osteoarthritis (OA) is characterized by bone erosions, joint space remodeling, and new bone formation mainly in distal interphalangeal (DIP) joints and thereby differs from hand manifestations in rheumatoid arthritis (RA). There are conflicting data about the benefit of treatment with conventional synthetic (cs) and biologic (b) disease modifying anti-rheumatic treatment (DMARD) on DIP OA.

Objectives: To assess the associations between DMARDs and incident, and progression of, radiographic DIP OA in RA patients.

Methods: We performed two observational cohort studies in the longitudinal Swiss Clinical Quality Management in Rheumatic Diseases registry (SCQM) between 1997 and 2014. RA patients who had ≥ 2 eligible hand radiographs were included at their first eligible radiograph (baseline) and were followed until the outcome or their last eligible radiograph. Radiographs were eligible if all 8 DIP joints could be scored. Modified Kellgren-Lawrence scores (KLS) were obtained by evaluating DIP joints for severity of osteophytes, joint space narrowing, subchondral sclerosis, and erosions. Incident/existing DIP OA was defined as KLS ≥ 2 in ≥ 1 DIP joint. Progression of existing DIP OA was defined as an increase of ≥ 1 in KLS in ≥ 1 DIP joint. We divided the study population into two cohorts based on whether DIP OA was present or absent at cohort entry (cohorts 1 and 2, respectively). Exposure status was defined time-dependently into mutually exclusive exposure groups: csDMARD monotherapy, bDMARD monotherapy, bDMARD/csDMARD combination therapy, past bDMARD/csDMARD therapy, or never DMARD use. Cox time-varying proportional hazard regression analyses were used to estimate hazard ratios (HRs) with 95% confidence intervals (CI) of DIP OA progression (cohort 1) or DIP OA incidence (cohort 2) associated with DMARD exposure categories (csDMARD monotherapy was the reference group because it was the largest group). Exposure and covariate information were extracted at every radiograph and other visit date. Missing covariate information was imputed using multiple imputation by chained equations. In sensitivity analyses, we repeated all analyses using generalised estimation equations (GEE).

Results: Among 2234 RA patients with 5928 eligible radiographs, 1340 patients had radiographic DIP OA at cohort entry (cohort 1) and 894 were DIP OA naïve (cohort 2). In cohort 1, radiographic progression of existing DIP OA was characterized by new osteophyte formation (666, 52.4%), followed by joint space narrowing (379, 27.5%), subchondral sclerosis (238, 17.8%), and erosion (62, 4.3%). bDMARD monotherapy was associated with an increased risk of radiographic DIP OA progression compared to csDMARD monotherapy (adjusted HR 1.34, 95% CI 1.07–1.69). The risk of DIP OA progression was not significant in csDMARD/bDMARD combination therapy users (adjusted HR 1.12, 95% CI 0.96–1.31), absent in past DMARD users (adjusted HR 0.96, 95% CI 0.66–1.41), and significantly lower among never DMARD users (adjusted HR 0.54, 95% CI 0.33–0.90), compared to csDMARD monotherapy users. In cohort 2, the risk of incident OA did not differ materially between treatment groups. Results from GEE analyses corroborated all findings.

Conclusion: Our results from this real-world RA cohort suggest that monotherapy with bDMARDs is not associated with incident DIP OA but may increase the risk of radiographic progression of existing DIP OA when compared to csDMARDs.

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POS0123

NEUROPATHIC PAIN SYMPTOMS IN INFLAMMATORY HAND OSTEOARTHRITIS(OA) LOWERS HEALTH RELATED PHYSICAL QUALITY OF LIFE AND MAY REQUIRE ANOTHER APPROACH THAN ANTI-INFLAMMATORY TREATMENT

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Background: Pain is a common, difficult to manage symptom in hand osteoarthritis (OA). Multiple pain mechanisms may play a role in hand OA.

Objectives: To investigate presence of neuropathic pain symptoms in patients with inflammatory hand OA, characteristics of those patients, their impact on health related quality of life (HR-QoL), and the influence of anti-inflammatory treatment on neuropathic pain symptoms.

Methods: Data from a randomised, double-blind, placebo-controlled trial of prednisolone including 92 patients with hand OA fulfilling ACR criteria were used. At baseline patients had signs of synovial inflammation, a VAS finger pain of ≥ 30 mm and who flared ≥ 20 mm upon NSAID washout. The primary endpoint was VAS finger pain (0-100) at week 6.

Neuropathic pain symptoms were measured at baseline and week 6 using the validated painDETECT questionnaire, consisting of questions on pain quality, pain intensity over time and radiating pain. Scores range -1 to 38 and patients are classified as having unlikely (<13), indeterminate (13-18) and likely (>18) neuropathic pain. HR-QoL was measured with physical component scale (PCS) of Short-Form 36 (SF36; 0-100), comorbidities with the Self-administered Comorbidities Questionnaire (SCQ; 0-45), radiographic severity with Kellgren-Lawrence (KL) sum score (0-120), and treatment response with OMERACT-OARSI responder criteria.

Association of patient characteristics with neuropathic pain symptoms was analysed with univariate and multivariate ordinal logistic regression, with painDETECT as dependent variable. Association of neuropathic pain symptoms with HR-QoL was analysed with multivariate linear regression, adjusted for age, sex, BMI, VAS finger pain, SCQ score and KL sum score, with PCS as dependent variable. Response of neuropathic pain symptoms and VAS pain to prednisolone was analysed with generalised estimating equations. Association of neuropathic pain symptoms at baseline with response to treatment was analysed using χ^2 -tests and GEE.

Results: 91 patients had complete painDETECT data at baseline (mean painDETECT score 12.8 [SD 5.9]). Scores were <13 in 53%, 13-18 in 31% and >18 in 16%. Higher painDETECT score categories were associated with less radiographic damage, more comorbidities, female sex and higher VAS finger pain in multivariate analysis. (table 1)

Patients with painDETECT scores >18 had a lower HR-QoL (PCS -6.5 [95%CI -10.4 to -2.6]) than those with painDETECT scores <13.

PainDETECT scores remained unchanged throughout the trial in both prednisolone-treated and placebo-treated patients, and there was no between-group

difference at week 6. VAS pain improved more in the prednisolone group than in the placebo group (mean between-group difference -16.5 [95%CI -26.1 to -6.9]) (figure 1). No association between the presence of neuropathic pain symptoms at baseline and OMERACT-OARSI response to treatment was found.

Conclusion: Patients with inflammatory hand OA and additional neuropathic pain symptoms are more often female and have more comorbidities, and report a lower QoL, than those without. Neuropathic pain symptoms seem unresponsive to anti-inflammatory therapy. Clinicians should be aware of neuropathic pain symptoms in their patients as they might benefit from additional, specific treatment.

Table 1. Ordinal logistic regression with painDETECT categories as dependent variable

Variables	Mean (SD) N=91 (100%)	Odds ratio (95% CI)
Age	64 (9)	0.96 (0.90 to 1.02)
Female sex; N (%)	72 (79%)	3.84 (1.19 to 12.39)*
BMI; median (SD)	27 (24 to 29)	0.97 (0.89 to 1.06)
SCQ score; median (SD)	2 (1 to 5)	1.04 (1.04 to 1.36)*
VAS finger pain	53.8 (2.1)	1.02 (1.00 to 1.04)*
KL sum score	37 (16)	0.96 (0.93 to 1.00)*

*p<0.05. BMI = body mass index. SCQ = Self-administered comorbidities questionnaire. VAS = visual analog scale. KL= Kellgren-Lawrence.

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Figure 1

Figure 1. Mean score at week 0 and week 6, per treatment arm, for VAS finger pain and PainDETECT. Placebo shown in dashed line, prednisolone 10 mg daily in solid line. Whiskers indicate 95% CI.

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POS0124

QUALITY OF LIFE FOR PATIENTS WITH KNEE OSTEOARTHRITIS WITH OR WITHOUT PAIN SENSITIZATION

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Background: Patients with central sensitization (CS) may have poorer quality of life (QOL).