Rheumatoid arthritis and hearing impairment: a review
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Objectives: Rheumatoid arthritis (RA) is an autoimmune disease with a prevalence of 0.5–1% in the general population, affecting multiple organs. The auditory system can also be affected by a variety of pathologies (1, 2). We aimed to evaluate possible pathologies, associated factors, and management of hearing impairment in RA.

Method: We performed a thorough literature review using PubMed, Embase, Cochrane, and ComDisDome.

Results: Sensorineural hearing loss is the most common type of hearing impairment in RA (25–72%). Elderly patients and those with long disease duration, active disease, positive rheumatoid factor, and increased laboratory values of acute phase reactants as well as rheumatoid nodules are at increased risk of hearing impairment (Table 1). Environmental factors including smoking, alcohol, and noise exposure can deteriorate the condition. Synovial destruction of the incudostapedial and incudomalleolar joints by an inflammatory process, rheumatoid nodules, auditory neuropathy, destruction of the cochlear hair cells, and drugs (e.g. salicylates, non-steroidal anti-inflammatory drugs, antimalarial and some kinds of disease-modifying anti-rheumatic drugs) are possible pathologies. The results of pure tone audiometry showed a higher prevalence of hearing loss for all frequencies (low, middle, high, and very high) in RA patients. Empirical treatment is based on steroids plus cessation of ototoxic drugs.

Conclusions: Hearing impairment is a multifactorial disease and is more prevalent among RA patients than healthy subjects. This is the first review of hearing impairment in RA.

References

Factors affecting the need for orthopaedic surgery in patients with rheumatoid arthritis. Results from 1010 patients diagnosed with RA from 1972 to 2009
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Objectives: The use of orthopaedic surgery is an important outcome measure in rheumatoid arthritis (RA), and we wanted to investigate how patient characteristics, time of diagnosis, and treatment affect the need for surgical procedures.

Method: We reviewed the medical history of 1544 patients diagnosed with RA at Haukeland University Hospital in Bergen, Norway from 1972 to 2009, of whom 1010 (mean age 57 years, 69% women) were included in the present study. Relevant orthopaedic procedures were obtained from the Norwegian Arthroplasty Register and the hospital’s administrative patient records. A total of 675 procedures (synovectomies 20%, arthrodeses 21%, prostheses 43%, and forefoot procedures 11%) were performed in 310 patients. Survival analyses were performed to evaluate the impact of various factors, including age, sex, radiographic changes, and year of diagnosis, on the risk of undergoing surgery. As knee synovectomies and procedures in the shoulder, elbow, hand, and foot were suspected to be RA specific, separate analyses for these
Treatment in rheumatoid arthritis

**PP17**

Do specific ACPAs or other autoantibodies in a novel assay predict response to methotrexate monotherapy in patients with early and DMARD-naive RA?

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**Objectives:** We aimed to determine whether specific ACPAs, or other autoantibodies (AAbs) on the ImmunoCAP ISAC chip, predict response to methotrexate in early RA.

**Method:** A total of 20 AAbs were measured by a custom-made microarray assay (Phadia AB, Uppsala, Sweden) in baseline samples from patients participating in two randomized controlled trials (RCTs; Swefot, n = 340; Improved, n = 450) and two inception cohorts (EIRA-SRQ, n = 1481; Glasgow, n = 384). The likelihood of a EULAR good/moderate response at 3–6 months follow-up of methotrexate was calculated by logistic regression adjusted for potential confounders (aOR, 95% CI; % positive for AAbs).

**Results:** Significant associations were observed for five and one out of 20 AAbs in Improved and Swefot, respectively. Fib36-52-cit (citrullinated fibrinogen) was significantly associated with good/moderate response in both RCTs (Improved: aOR 2.27, 95% CI 1.19–4.31, 32% positive; Swefot: aOR 1.87, 95% CI 1.06–3.29, 44% positive). Fib591-cit (also a citrullinated fibrinogen) was associated with good/moderate response in Improved (aOR 3.70, 95% CI 1.27–10.76, 15% positive) and a non-significant trend was observed in Swefot (aOR 1.65, 95% CI 0.89–3.06, 30% positive). Three AAbs predicted response in Improved only [Vim60-75-cit (citrullinated vimentin, aOR 1.85, 95% CI 1.06–3.21, 46% positive), pept-Z2 (aOR 1.81, 95% CI 1.02–3.23, 41% positive), and pept-5 (aOR 2.25, 95% CI 1.27–4.01, 44% positive)] and a trend was observed for CEP-1 (aOR 1.70, 95% CI 0.96–3.00, 40% positive). Of interest, none of the above associations were replicated in the inception cohorts, nor did we observe any trend for a dose–response association with response for the total number of positive AAbs.

**Conclusions:** Whereas two AAbs against citrullinated fibrinogen predicted response to methotrexate in both early RA RCTs, neither these nor other AAbs predicted response in the real-life population-based cohort setting.

**PP18**

Lack of early change in DAS28-4(ESR) predicts the likelihood of achieving LDA at month 6: tofacitinib monotherapy vs. MTX in MTX-naive patients with RA

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**Background:** Tofacitinib is an oral Janus kinase inhibitor for the treatment of RA. Guidelines recommend targeting remission or low disease activity (LDA; DAS28 ≤ 3.2) and adjusting therapy after 3–6 months.

**Method:** This post-hoc analysis of phase 3 data (ORAL Start; NCT01039688) investigated the relationship between timing/magnitude of early changes in DAS28-4(ESR) and LDA likelihood at month 6 in 948 MTX-naïve RA patients randomized 2:2:1 to tofacitinib 5 or 10 mg twice daily (BID) or MTX. Conditional probability of LDA at month 6 was calculated, given failure to achieve DAS28-4(ESR) improvement from baseline (range ≥ 0.3 to 1.8) at month 1 or 3. Two-year data with non-responder imputation were used.

**Results:** At month 3, 20.9% of tofacitinib 5 mg BID patients did not improve DAS28-4(ESR) ≥ 1.2 from baseline vs. 38.0% for MTX patients. Failure to achieve DAS28-4(ESR) ≥ 1.2 at month 3 was associated with low probability of LDA at month 6 (≤ 6.8%) for tofacitinib 5 mg BID and MTX (Table 1). Failure to achieve DAS28-4(ESR) improvement from baseline at month 3 (range ≥ 0.30–1.8) for tofacitinib 5 mg BID and MTX was associated with ≤ 10% probability of LDA at month 6. For tofacitinib 5 mg BID, failure to achieve lower (range ≥ 0.3 to 0.9) vs. higher (≥ 1.2 to 1.8) thresholds of DAS28-4(ESR) improvement at month 1 was associated with lower probability of LDA at month 6.

**Conclusions:** MTX-naïve RA patients receiving tofacitinib 5 mg BID, tofacitinib 10 mg BID, or MTX who failed to improve DAS28-4(ESR) ≥ 1.2 by month 3 have a low probability of achieving LDA at month 6.
Tofacitinib, an oral JAK inhibitor, in the treatment of RA: safety and clinical and radiographic efficacy in open-label, long-term extension studies over 7 years

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Background: Tofacitinib is an oral Janus kinase inhibitor for the treatment of RA. Here, we describe tofacitinib safety, tolerability, and clinical response over 84 months, and radiographic data over 12 months, in long-term extension (LTE) studies.

Method: Data were pooled from two open-label studies [NCT00413699 (ongoing; database unlocked at March 2015); NCT00661661] of RA patients who completed randomized phase 1/2/3 studies (tofacitinib 5 or 10 mg twice daily ± DMARDs). Primary endpoints were adverse events and confirmed laboratory safety (decreased haemoglobin, neutrophil and lymphocyte counts, increases > 50% from baseline in creatinine). Secondary endpoints were DAS28-4(ESR), HAQ-DI, and the modified Total Sharp Score (mTSS). Safety and efficacy data were included over 96 and 84 months (n ≤ 30 post-month 84), respectively.

Results: In total, 4867 patients were treated [mean (max) duration: 1107 (2895) days]. Most patients (90.9%) had baseline data from index studies. Total tofacitinib exposure was 14 926 patient-years; 79.2% of patients maintained the initial dose. No new safety signals were detected (Table 1). At baseline, month 1, and month 84, respectively, the mean DAS28-4(ESR) was 6.29, 3.74, and 3.20 and the mean HAQ-DI score was 1.42, 0.81, and 0.78. mTSS data were available for 1099 patients. The mean mTSS was 24.0 at baseline (last index value), 25.1 at month 6, and 24.3 at month 12; the mean change from baseline in mTSS was 0.3 at month 6 and 0.2 at month 12. Conclusions: Consistent safety and sustained efficacy over 84 months was seen in RA patients receiving tofacitinib in LTE studies. Changes in mTSS were minimal at month 12 in LTE studies.

Efficacy and safety of baricitinib in patients with rheumatoid arthritis and inappropriate response to conventional disease-modifying anti-rheumatic drugs: 24-week phase 3 RA-BUILD study summary results

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Background: We present efficacy, safety, and patient-reported outcome (PRO) analyses from patients with active rheumatoid arthritis (RA) and inappropriate response (IR) to conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs) in the randomized 24-week phase 3 RA-BUILD study of baricitinib, an oral JAK1/2 inhibitor.
Method: Patients with active RA and IR to csDMARDs (n = 684) received placebo or baricitinib (2 or 4 mg QD) for 24 weeks. The primary endpoint was the ACR20 response at week 12 for baricitinib 4 mg vs. placebo. Safety and other efficacy analyses are also reported.

Results: Significant improvements in ACR 20/50/70, DAS28-ESR, SDAI remission, and HAQ-DI, and faster decreases in morning joint stiffness, worst joint pain, and tiredness were seen with baricitinib vs. placebo at weeks 12 and 24. At week 24, the modified Total Sharp Score (mTSS) was reduced with baricitinib 4 mg vs. placebo. Baricitinib 4 mg produced a significant rapid decrease (within 1 week) in DAS28-ESR and CDAI vs. placebo. TEAE and SAE rates, including serious infections, were similar among groups. Increases in total lymphocyte count (TLC) including T, B, and NK cells at week 4 for baricitinib were within the normal ranges. T cells and NK cells decreased and B cells increased at weeks 12 and 24 vs. placebo.

Conclusions: Baricitinib 4 mg resulted in significant improvement in structural progression and PROs at weeks 12 and 24. Safety and infection rates were acceptable regardless of TLC changes.

PP21

Efficacy and safety of baricitinib in patients with active rheumatoid arthritis and inadequate response to tumour necrosis factor inhibitors: the 24-week phase 3 RA-BEACON study

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Table 1. Safety summary over 96 months.
Background: Baricitinib (BARI), an oral JAK1/JAK2i, was investigated in the phase 3 RA-BEACON study.

Method: A total of 527 patients with active rheumatoid arthritis (RA) despite previously using ≥ 1 tumour necrosis factor inhibitor (TNFi) were randomized to placebo (PBO) or BARI (2 or 4 mg, QD). The primary endpoint was week-12 ACR20 (BARI 4 mg vs. PBO). Subgroup efficacy by prior biologic use, safety, and changes in total lymphocyte count (TLC) and NK cells are reported.

Results: Week-12 ACR20 was higher with BARI 4 mg vs. PBO (55% vs. 27%; p ≤ 0.001). Improvements in ACR20/50/70 and DAS28-CRP occurred with BARI 4 mg (one prior TNFi) at week 12/24; improvements in CDAI, SDAI, and HAQ-DI were observed at week 24. A decrease ≥ 0.6 in DAS28 and ≥ 6 in CDAI at week 4 was observed in 79% and 80% of patients on BARI 4 mg, respectively, associated with low disease activity (LDA)/remission at week 12/24. More TEAEs occurred with BARI 2 and 4 mg vs. PBO, including infections. TLC changes in BARI groups were similar vs. PBO at week 12/24. There were increases in T, B, and NK cells at week 4, and decreases in T and NK cells and an increase in B cells at week 12/24 for BARI groups (all TLC changes within the normal range; the NK-cell decrease was not associated with increased infection).

Conclusions: BARI showed clinical improvements from week 4 to week 24 with an acceptable safety profile. The week-4 clinical response might predict later LDA/remission.

PP22

Baricitinib, methotrexate, or baricitinib plus methotrexate in patients with early rheumatoid arthritis who had received limited/no treatment with disease-modifying anti-rheumatic drugs: Phase 3 trial results

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Background: We report results from a phase 3 study of baricitinib.

Method: Patients with active RA (TJC+SJC ≥ 6, hsCRP ≥ 3.6 mg/L) and no previous disease-modifying anti-rheumatic drugs (DMARDs) other than ≤ 3 doses of methotrexate (MTX) were randomized to MTX, baricitinib 4 mg QD, or baricitinib 4 mg QD +MTX for ≤ 52 weeks. MTX (±baricitinib) was uptitrated from 10 to 20 mg QW over 8 weeks. The primary objective was to evaluate the non-inferiority of baricitinib monotherapy to MTX on ACR20 at week 24.

Results: Of 584 randomized patients, 87%, 91%, and 89% completed week 24 in the MTX, baricitinib, and baricitinib+MTX groups, respectively. ACR20 response at week 24 was higher with baricitinib monotherapy vs. MTX (77% vs. 62%; p ≤ 0.01). Baricitinib produced greater improvements in secondary disease activity measures than MTX as early as week 1. Baricitinib+MTX did not appear to increase the benefit observed with baricitinib monotherapy. Clinical remission was seen in significantly higher proportions of patients receiving baricitinib or baricitinib +MTX vs. MTX alone. TEAE and SAE rates were similar across groups. Up to 24 weeks, two (1.0%), six (3.8%), and 14 (6.5%) patients discontinued treatment because of an AE in the MTX, baricitinib, and baricitinib+MTX groups, respectively.

Conclusions: All groups experienced disease activity improvements; baricitinib monotherapy produced significantly larger, more rapid improvements and higher rates of remission compared to MTX monotherapy, with a satisfactory safety profile.

PP23

Efficacy and safety of sirukumab in patients with active rheumatoid arthritis despite disease-modifying anti-rheumatic drug treatment: results of a randomized, double-blind, placebo-controlled study

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Background: This phase 3 global study was designed to evaluate the efficacy and safety of sirukumab, a selective, high-affinity human monoclonal antibody to IL-6, in patients with rheumatoid arthritis (RA) refractory to conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs).

Method: A total of 1670 patients with active RA and inadequate response to csDMARDs were randomized (1:1:1) to sirukumab subcutaneous (sc) 50 mg q4w, sirukumab sc 100 mg q2w, or placebo sc q2w. Co-primary endpoints were the week-16 American College of Rheumatology (ACR)20 response and week-52 modified van der Heijde/Sharp (vdH-S) score change from baseline.

Results: Patients showed significant improvement with sirukumab treatment (both doses) vs. placebo based on both co-primary endpoints (p < 0.001) and all major secondary endpoints: the week-24 Health Assessment Questionnaire Disability Index (HAQ-DI) change from baseline, ACR50, and Disease Activity
Score based on C-reactive protein (DAS-CRP) remission and week-52 major clinical response \( (p \leq 0.001; \) Table 1). In addition, there were significant improvements at week 52 in Short Form-36 physical and mental component summary scores \( (p < 0.001) \) and radiographic progression score change \( \leq 0 \) \( (p<0.001) \).

Up to week 52, treatment-emergent adverse events (TEAEs) and serious AE incidences, respectively, were numerically higher with sirukumab 50 mg q4w \( (79.6\% \text{ and } 11.0\%) \) and 100 mg q2w \( (80.2\% \text{ and } 9.8\%) \) vs. placebo \( (65.5\% \text{ and } 6.8\%) \). The most common AEs \( (\geq 8\%) \) with sirukumab were elevated liver enzymes, upper respiratory tract infections, injection site erythema, and nasopharyngitis.

**Conclusions:** In DMARD-inadequate responders, both sirukumab doses significantly reduced RA signs/symptoms, inhibited radiographic progression, and improved health-related quality of life. The safety profile of sirukumab was consistent with the known safety profile of anti-IL-6 treatment.

### Table 1. Key endpoints by prior biologic use.

<table>
<thead>
<tr>
<th></th>
<th>Placebo (n = 556)</th>
<th>Sirukumab 50 mg q4w (n = 557)</th>
<th>Sirukumab 100 mg q2w (n = 557)</th>
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<tbody>
<tr>
<td><strong>Primary endpoints</strong></td>
<td></td>
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<tr>
<td>ACR20 at week 16, n (%)</td>
<td>147 (26.4)</td>
<td>305 (54.8)*</td>
<td>298 (53.5)*</td>
</tr>
<tr>
<td>vdH-S mean (sd) change from baseline at week 52</td>
<td>3.69 (9.25)</td>
<td>0.50 (2.96)*</td>
<td>0.46 (3.26)*</td>
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<tr>
<td><strong>Major secondary endpoints</strong></td>
<td></td>
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<tr>
<td>HAQ-DI mean (sd) change from baseline at week 24</td>
<td>-0.22 (0.53)</td>
<td>-0.43 (0.58)*</td>
<td>-0.46 (0.57)*</td>
</tr>
<tr>
<td>ACR50 at week 24, n (%)</td>
<td>69 (12.4)</td>
<td>168 (30.2)*</td>
<td>185 (33.2)*</td>
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<tr>
<td>DAS28 (CRP) remission at week 24, n (%)</td>
<td>31 (5.6)</td>
<td>145 (26.0)*</td>
<td>142 (25.5)*</td>
</tr>
<tr>
<td>Major clinical response at week 52, n (%)</td>
<td>10 (1.8)</td>
<td>30 (5.4)*</td>
<td>50 (9.0)*</td>
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\*\( p \leq 0.001 \) vs. placebo.

**PP24**

**Response and radiographic progression in biologic-naïve and biologic-experienced patients with rheumatoid arthritis treated with sirukumab**

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**Background:** This subgroup analysis of a phase 3 study of sirukumab (a selective, high-affinity human monoclonal antibody to IL-6) compared efficacy and radiographic progression of sirukumab between subgroups of patients with active rheumatoid arthritis (RA) despite conventional disease-modifying anti-rheumatic drug (DMARD) treatment who had previously received biologic therapy (biologic-experienced) and synthetic DMARDs only (biologic-naïve).
**Method:** Patients were randomized (1:1:1) to sirukumab subcutaneous (sc) 50 mg q4w, sirukumab sc 100 mg q2w, or placebo sc q2w. Co-primary efficacy endpoints were: week 16 ACR20 response and week 52 change from baseline in modified vdH-S radiographic score. This post-hoc analysis compared efficacy for biologic-naïve and -experienced patients within the biologic-experience category for sirukumab vs. placebo and across the biologic-experience category for the sirukumab dose group.

**Results:** All outcomes improved with sirukumab vs. placebo regardless of prior biologic use (all p < 0.001 within the biologic-experience categories; Table 1). A total of 34.9% (583/1670) of patients were biologic experienced; sirukumab efficacy was comparable with 1 or ≥ 2 prior biologics. At baseline, compared with biologic-naïve patients, biologic-experienced patients had longer disease duration (mean, 11 vs. 7 years; p < 0.0001) and worse HAQ-DI scores (mean, 1.58 vs. 1.49; p = 0.02). No differences between biologic-naïve and -experienced patients were observed in either sirukumab group for the co-primary endpoints (both p ≥ 0.1), as well as the week 24 ACR50 response, DAS28 (CRP) remission, and week 52 mean DAS28 change from baseline (all p ≥ 0.1).

**Conclusions:** Both sirukumab doses significantly reduced RA signs/symptoms and inhibited radiographic progression vs. placebo within prior biologic-use categories, with comparable efficacy observed between biologic-naïve and biologic-experienced patients for both sirukumab doses.

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**PP25**

**Improvement in measures of prevalent depressed mood and anhedonia (PDMA) and fatigue in a randomized, double-blind, placebo-controlled, phase 2 study of sirukumab in patients with rheumatoid arthritis**

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**Background:** Depressive symptoms and fatigue commonly affect patients with rheumatoid arthritis (RA); IL-6 appears to have a role in depression. We assessed the effects of treatment with human IL-6 antibody sirukumab on measures of prevalent depressed mood and anhedonia (PDMA) and fatigue in RA patients.

**Method:** This post-hoc analysis evaluated SF-36 Mental Health and Vitality domains from a phase 2 study of subcutaneous sirukumab in patients with active RA despite methotrexate. Patients were grouped by baseline PDMA status (self-reported depressed mood/anhedonia on the SF-36 ‘most of the time’ for 4 weeks).

**Results:** At trial entry, about 26% of patients were classified as having PDMA. This group experienced significantly more fatigue and nervousness than those without PDMA. The presence of PDMA was not fully explained by RA chronicity or severity, or baseline serum levels of CRP, SAA, IL-6, sIL-6R, and sgp130. Clinical efficacy of sirukumab with regard to RA disease measures occurred in patients with and without PDMA. Patients with PDMA receiving sirukumab, but not placebo, achieved significant improvements at week 12 in depressed mood/anhedonia (p = 0.0006) and fatigue (p = 0.0157); in these patients, baseline sIL-6R levels correlated significantly with improvement at week 12 in depression/anhedonia (Spearman r = 0.44, p = 0.015). Furthermore, patients with PDMA on sirukumab, but not placebo, demonstrated significant improvements in depressed mood/anhedonia in both ACR50 responders (p = 0.0024) and non-responders (p = 0.0014).

**Conclusions:** These findings link IL-6 signalling pathway dysregulation to depression and fatigue, and suggest that peripheral anti-IL-6 treatment can improve depressive symptoms in RA patients independently of RA clinical response.

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**PP26**

**mPGES-1 deletion increases prostacyclin and evades the elevated systemic ADMA associated with COX-2 inhibitors: relevance to cardiovascular safety of mPGES-1 inhibitors**

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**Background:** Cardiovascular side-effects caused by non-steroidal anti-inflammatory drugs (NSAIDs), which inhibit COX-2, are a global health issue preventing development of new drugs that target prostaglandins in inflammation and cancer therapy. Microsomal prostaglandin E synthase (mPGES)-1 inhibitors encapsulate all of the therapeutic promise of NSAIDs with the potential of reduced side-effects. However, unfounded and untested concerns over their cardiovascular toxicity remain. Here we have profiled mPGES-1 in renal and vascular pathways that reflect what we know of NSAID cardiovascular toxicity; specifically, these are the COX product prostacyclin, which is cardioprotective, and the endogenous eNOS inhibitor ADMA, which is cardiotoxic.

**Method and Results:** Deletion of mPGES-1 reduced vascular PGE2 formation but increased plasma levels of prostacyclin. In the kidney, mPGES-1 and COX-2 were compartmentalized to the renal cortex and renal medulla, respectively. In vivo, COX-2 inhibition altered renal medullary expression of genes associated with the production (Prmt1) and metabolism (Agxt2) of ADMA, resulting in significantly elevated plasma ADMA levels. These changes were mirrored in mice lacking prostacyclin synthase (PGIS),

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but in contrast, deletion of mPGES-1 had no effect on the ADMA pathway. Vascular NO responses, a readout of ADMA activity, were in fact improved by mPGES-1 deletion, consistent with a preserved ADMA pathway coupled with the loss of constrictor PGE2 responses.

Conclusions: PGIS but not mPGES-1 mediates the cardiovascular protective functions of COX-2 on the renal ADMA pathway. These data should provide renewed confidence in the development of selective inhibitors of mPGES-1 as safer alternatives to NSAIDs for inflammation, pain, and cancer.

Registers in rheumatology

PP27

Rheumatoid arthritis (RA) treatment guidelines in Finland

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Background: The treatment target of RA was set by Finnish rheumatologists decades ago (1).

Objectives: To describe the Finnish Current Care Guidelines for RA (FinRAG).

Method: The FinRAG are developed through an open and transparent process according to evidence-based clinical practice guidelines and form the basis for treatment decisions. The drafted guidelines are circulated to relevant interest groups prior to being finalized. The FinRAG were updated in 2015 by the Finnish Medical Society Duodecim in association with the Finnish Society for Rheumatology.

Results: The FinRAG place emphasis on early diagnosis and the prompt start of effective treatment to achieve fast remission and maintain the patient’s functional capacity and working ability. The following points are highlighted:

- Initiation of combination therapy with methotrexate, sulfasalazine, hydroxychloroquine, and prednisolone (5–7.5 mg).
- Preference to sc methotrexate where possible (higher bioavailability than oral).
- The importance of treating swollen joints with intra-articular glucocorticoids.
- Prompt use of biologics for persistently active disease.
- Patient education to ensure compliance with long-term treatment.
- Physiotherapy input for engagement in regular physical exercises.
- Prevention of osteoporosis and management of cardiovascular risks.
- Regular patient monitoring as part of clinical care.
- Multidisciplinary team input for every patient in early disease.
- Annual review by a doctor with a good understanding of rheumatology.

Conclusions: In current times where highly effective treatments and therapeutic strategies are available, care guidelines and recommendations should reflect the best available evidence within the therapeutic window of opportunity while taking into account cost-effectiveness. The FinRAG have been developed according to these principles.

References


PP28

Screening for tuberculosis before TNF-α treatment in routine rheumatic care. Results from the nationwide ICEBIO registry

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Objectives: Following the introduction of TNFi, the number of cases of tuberculosis (TB) has increased. Therefore, screening patients with rheumatic diseases for TB before initiating TNFi is recommended. Iceland has a low prevalence of TB infection and BCG vaccinations are not recommended. Our aim was to review the results from TB screening in routine practice and analyse whether changes in the screening process are needed.

Method: All patient (RA, PsA, AS) records of those individuals who were filed in ICEBIO (1999–2014) due to TNFi treatment were reviewed with respect to: age, sex, a tuberculin skin test (TST), start date of TNFi treatment, DMARD use at the time of the TST, and which TNFis were selected. The data were then crosschecked with the Berkill registry, a nationwide database for TB.

Results: We reviewed data from 756 individuals (58% female, mean age 54 years). The TST was negative in 614 cases (81%) and positive in 41 (5.4%), there were nine false positives (1.2%) and data for 94 cases were missing (12%). A total of 119 patients were registered in the Berkill; of these, 62 had a history of positive TST and 54 had been vaccinated, while 11 patients had been diagnosed with TB (of whom five had a negative TST on screening). Three patients were diagnosed with active TB after TNFi treatment had started. The data were then crosschecked with the Berkill registry, a nationwide database for TB.

Conclusions: These results illustrate the importance of TB screening before starting TNFi treatment. Improvement in registration of TST results is necessary, and whether the screening process should be intensified by repeated TST testing or the use of interferon gamma release assays (IGRAs) needs to be discussed.