of tapering MTX. Evaluation of clinical disease activity, severe adverse events, and the continuation rate during MTX tapering were also evaluated. According to tapering response, predictors for successful tapering MTX and progression of bone destruction were determined. Statistical analysis was performed by t-test, Mann-Whitney U test or one-way analysis of variance using EZR ver 1.37[4].

Results: Patients' demographic data were as follows, mean age:60.8 yrs, female: 88 pts:25pts, disease duration: 66.1 months, mean MTX dose: 8.51mg/week, mean DAS28-CRP: 1.55, ACPA:179.3U/ml, ACPA positive(%) : 72.6, RF: 48.1 IU/ml, RF(%) :48.1, mean mTSS:17.6, ΔmTSS=1.8% (10 cases) Accord- ing to previous report, 113 RA patients were divided into three groups: Flared (F) group (22 patients), Less tapering (L) group, (37 patients) Good tapering (G) group (54 patients). As shown in Table 1A, significant differences were observed among the three groups in MTX dose at 6M(F vs G, P<0.01, L vs G, P>0.01), MTX dose at 12M(F vs L, P<0.01, F vs G, P<0.01, L vs G <0.01), DAS28-CRP at 12M (F vs G, P<0.05), ΔmTSS=0.5, baseline CDAI value (L vs G group, P<0.05), CDALI, SDAI at 12M(F vs G, P<0.05, F vs L, P=0.05), D-vas at 12M(F vs L, G, P<0.01). Table 1B shows significant differences between F group and unflared (L+G group) in baseline mTSS (P<0.05), ΔmTSS(0.5), CDAI, SDAI, delta ICAS at 12M(P<0.01), MMP9 at 12M(P<0.05). Severe adverse events; cancer(stomach, lung) 2, MTX associated lymphoma/brain 1, pneumoniae 1. These results indicated baseline mTSS may be a predictor for flare and flare response may be a predictor for joint destruction. Baseline CDAI may be a predictor for good response.

Conclusion: Baseline mTSS may be a predictor for flare successive joint damage in MTX tapering. Keeping low CDAI not DAS28 is important for good tapering.

REFERENCES:

Table 1A.

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<th>Group</th>
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<th>Not fared group(N=41)</th>
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<td>age (yr)</td>
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<td>duration(month)</td>
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<tr>
<td>LDA(month)</td>
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<td>31.3±40.7</td>
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<td>MTX dose baseline</td>
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<td>8.9±2.4</td>
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<td>ESR</td>
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<td>N.S</td>
</tr>
<tr>
<td>CRP (mg/dl)</td>
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<tr>
<td>RF</td>
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Table 1B.

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<td>CRP (mg/dl)</td>
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Acknowledgements: NIL.

Disclosure of Interests: None Declared.

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AB0470

EFFECT OF TOFACITINIB IN MODULATING PLATELET FUNCTION IN PATIENTS WITH RHEUMATOID ARTHRITIS

Keywords: Rheumatoid arthritis, Safety, Targeted synthetic drugs

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Background: Recent data suggested an association between Tofacitinib treatment and increased cardiovascular events in patients with Rheumatoid arthritis. Janus Kinase inhibitors (JAKI), specifically JAK3, have been demonstrated to be one of the regulators of platelet function. Treatment platelets with thrombin induces tyrosine phosphorylation of the JAK3 target substrates STAT1 and STAT3, and JAK3 deficiency in mice is protective and improves event-free survival in thromboplastin-induced thromboembolism.

Objectives: This study aimed to study the ability of the JAK1/JAK3 inhibitor, Tofacitinib, to influence platelet activity in patients with Rheumatoid Arthritis.

Methods: We enrolled patients with a diagnosis of RA according to the ACR/EULAR 2010 ACR/EULAR criteria. Peripheral blood was obtained from RA patients at the baseline and after 1, 3 and 6 months of Tofacitinib therapy. Platelet aggregation assay was performed by optical aggregometry stimulated with the thromboxane A2 receptor in RA patients and controls. The aggregation test was performed before starting the therapy with Tofacitinib and after one month, three months and six months.

Results: 25 RA patients treated with Tofacitinib were recruited, 86% female and 14% male, with a mean age of 56.5 years (SD 9.7 yrs.), mean disease duration of 16.3 years, mean ESR 28.2 mm, mean CRP 0.9 mg/dl, mean SDAI 18.2 and mean prednisone equivalent dose 3.75 mg/day. 78% of the patients were positive for Rheumatoid factor and 57.1% for ACPA. Looking at the classical risk factors, 35.7% had hypertension, 21.4% had hypercholesterolemia, 16.2% had diabetes, and 14.2% were smokers.; only one patient had a previous cardiovascular event. The platelet aggregation was not influenced by Tofacitinib treatment at any time points (T1, T3 and T6) at any Thromboxane dose (5μM and 20 μM), furthermore did not differ from patients and controls basally (64%, SD 15.84% vs 62%, SD 10.5%).

Conclusion: In conclusion, Tocfatinib does not increase platelet aggregation in patients treated for Rheumatoid Arthritis.

REFERENCES:

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Disclosure of Interests: Daniele Mauro Grant/research support from: Research was supported by an unrestricted grant by Pfizer, Daniela Iacono Grant/research support from: Research was supported by an unrestricted grant by Pfizer, Ilenia Pantano Grant/research support from: Some research was supported by an unrestricted grant by Pfizer, Maria Laura Marchesano Grant/research support from: Some research was supported by an unrestricted grant by Pfizer, Flavia Riccio Grant/ research support from: Some research was supported by an unrestricted grant by Pfizer, Anna Pellegrino Grant/research support from: Some research was supported by an unrestricted grant by Pfizer, Flavia Riccio Grant/ research support from: Some research was supported by an unrestricted grant by Pfizer, Francesco Ciccia Grant/research support from: Some research was supported by an unrestricted grant by Pfizer.

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AB0471

FEASIBILITY STUDY OF METHOTREXATE USE IMPROVEMENT IN RHEUMATOID ARTHRITIS (MIRA) USING BIOMARKER FEEDBACK: QUALITATIVE RESULTS FROM PATIENT INTERVIEWS

Keywords: Biomarkers, Rheumatoid arthritis, Qualitative research methods

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Background: MTX non-adherence is associated with reduced response, increased healthcare costs to the NHS and reduced quality of life for the patient. Often the prescriber is unable to determine if a patient is taking their medication as prescribed. There is a need to measure adherence directly to facilitate more precise and objective measurements of adherence to add to the clinicians tools and help to open up honest discussions and supportive interventions with patients. Our group has previously developed a sensitive biochemical assay for the detection of MTX adherence, MIRA is a feasibility trial designed to assess the feasibility of a randomised controlled trial of MTX biochemical adherence biofeedback. This paper reports on the patients’ perspectives on adherence monitoring.

Objectives: MIRA is a prospective multi-centre randomised controlled trial to examine the feasibility of a fully powered randomised controlled trial to examine if
Conclusion: The drug survival rate of 90.3% after efficacy assessment at clinical review suggests a good tolerance, patient acceptability and clinical effectiveness. This Edinburgh-based, real-world, unselected patient cohort includes a high rate of multidrug-resistant RA yet sustains a high drug survival rate at 6 months. The cohort is limited by the lack of accurate DAS-28 recording in the clinical record. This may in part reflect the Lothian model of treating imaging-confirmed synovitis in the absence of a DAS-28 result.

REFERENCES:


Title: Filgotinib for Rheumatoid Arthritis - an observational study to assess clinical effectiveness in NHS Lothian.

Christopher D Box, Ewan L Swann, Helen E Harris, Neil D McKay.

Author affiliations: Rheumatic Diseases Unit, Western General Hospital, Edinburgh.

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Disclosure of Interests: Christopher Box: None declared, Ewan Swann: None declared, Helen Harris: None declared, Neil McKay: Speakers bureau: Presentation support from Galapagos NV.

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AB0472 FILGOTINIB FOR RHEUMATOID ARTHRITIS - AN OBSERVATIONAL STUDY TO ASSESS CLINICAL EFFECTIVENESS IN NHS LOTHIAN

Keywords: Real-world evidence, Targeted synthetic drugs, Rheumatoid arthritis

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Background: Filgotinib (Jysela) is a Janus-kinase (JAK) inhibitor which in 2021 was approved for use in the UK in moderate or severe rheumatoid arthritis (RA) either as monotherapy or in combination with Methotrexate. A reduced dose of 100 mg is recommended for patients aged 75 years or over, or with moderate or severe renal impairment. Clinical efficacy of filgotinib alongside a conventional disease-modifying anti- rheumatic drug (CDMARD) was demonstrated in the FINCH 1 and FINCH 2 trials [1], but evidence of real-world effectiveness is currently limited.

Objectives: To describe a cohort of patients prescribed filgotinib (Jysela) for RA in NHS Lothian and assess clinical response and discontinuation rates in regular clinical practice.

Methods: The electronic patient record was reviewed for patients prescribed filgotinib in NHS Lothian between December 2021 and August 2022. Baseline data was recorded including time since diagnosis of RA, previous and concurrent treatment, duration of treatment with filgotinib and discontinuation reason if applicable. Comorbidities of intestinal lung disease (ILD), prior venous thromboembolism (VTE), major adverse cardiovascular events (MACE) or malignancy were recorded. steroid prescriptions at the time of commencing filgotinib were recorded. DAS-28 scores within 6 months prior to starting filgotinib were recorded where they had been performed, along with most recent DAS-28 scores.

Results: 145 patients were prescribed filgotinib for RA. 106 females and 39 males. Median age was 64 years. Median treatment duration was 213 days. 35 (24.1%) patients were treated with the reduced 100mg dose. 14 (9.7%) discontinued treatment, 61 (83.6%) patients were in remission or low disease activity, and 12 (16.4%) patients had high disease activity. In the third months after MTX treatment, 71 (49.0%), with mean VAS 62. Post-treatment VAS was recorded in 32 (22.1%), with mean VAS 43. 12 (8.3%) patients had DAS-28 scores recorded before and after. 7 were classed as a good response by EULAR criteria, 2 were a moderate response, and 3 were non-responders.

Results: The mean age of the patients was 54.82±11.65 years. Before MTX treatment, 27 (37%) patients had moderate DAS-28 scores and 46 (63.0%) patients had high disease activity. In the third months after MTX treatment, 44 (60.3%) patients were in remission or low disease activity, and 29 (39.7%) patients were moderate or high disease activity. In the sixth months after MTX treatment, 61 (83.6%) patients were in remission or low disease activity, and 12 (16.4%) patients were moderate or high disease activity (Table 1). The median DAS-28 score of the patients before MTX treatment was 5.51 (min-max: 3.49-7.77), the median score of the third months after treatment was 2.86 (min-max: 0.77-5.63) and the sixth months after treatment was 2.33 (min-max: 0.77-5.24). A significant improvement was observed in the DAS-28 score evaluated during the

AB0473 THE EFFICACY OF METHOTREXATE MONOTHERAPY STRATEGY CHosen as first-line DMARD therapy in PATients with NEWLY DIAGNOSED RHEUMATOID ARTHRITIS: RETROSPECTIVE EVALUATION OF SINGLE TERTIARY CENTER EXPERIENCE

Keywords: Remission, Disease-modifying drugs (DMARDs), Rheumatoid arthritis

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Background: Rheumatoid Arthritis (RA) is a chronic systemic disease in which immunologically mediated inflammation of synovia-lined joints can disrupt joint structure and function. Methotrexate is the first recommended conventional disease-modifying antirheumatic drugs (DMARD) when diagnosed in patients with RA. The goal of treatment is low disease activity or remission [1].

Objectives: It was aimed to retrospectively evaluate the response of patients who received methotrexate (MTX) monotherapy as a first-line DMARD strategy in newly diagnosed RA patients.

Methods: Between January 2018-September 2022, 642 patients diagnosed with RA in the Department of Internal Medicine Rheumatology of Sakarya University were retrospectively analyzed. All patients fulfilled the criteria for the 2010 Rheumatoid arthritis classification. Patients with newly diagnosed RA and obtaining MTX monotherapy as the first conventional DMARD strategy were included in the study. Patients diagnosed in another center and initially started conventional DMARD combination therapy were excluded from the study. Patients who discontinued treatment due to side effects were excluded and 73 were included in the study. Age, gender, rheumatoid factor and anticitrulized peptide antibody levels of the patients were recorded. Disease activity was evaluated at 0-3-6 months with the Disease Activity Score Calculator for Rheumatoid Arthritis (DAS-28). Data were analyzed using the computer program SPSS 21. Wilcoxon Signed Rank test and Friedman test were used from non-parametric tests. p<0.05 was considered statistically significant.

Results: The mean age of the patients was 54.82±11.65 years. Before MTX treatment, 27 (37.0%) patients had moderate DAS-28 scores and 46 (63.0%) patients had high disease activity. In the third months after MTX treatment, 44 (60.3%) patients were in remission or low disease activity, and 29 (39.7%) patients were moderate or high disease activity. In the sixth months after MTX treatment, 61 (83.6%) patients were in remission or low disease activity, and 12 (16.4%) patients were moderate or high disease activity (Table 1).