Results: 354 women and 123 men with RA (n=477), 81 women and 69 men with PsA (n=150), 121 women and 191 men with SpA (n=312) were included. No significant differences in biometrics was seen between female and male patients at baseline in all diseases.

In RA patients overall DAS28 decreased from baseline (V1) to V2 and V3 (DAS28: V1: male: 4.38 [3.66, 5.11], female: 4.30 [3.68, 5.03], p(m/f) = 0.905; V2: male: 2.66 [1.73, 3.63], female: 3.10 [2.17, 3.98], p(m/f) = 0.015; V3: male: 2.25 [1.39, 3.36], female: 3.01 [1.87, 3.87], p(m/f) = 0.002). For TNF inhibitors (n=311), there was a significant difference between genders at V2 (Fig.1a).

In SpA patients overall BASDAI decreased from baseline (V1) to V2 and V3 (BASDAI: V1: male: 4.70 [2.68, 6.18], female: 4.80 [3.30, 6.20], p(m/f) = 0.465; V2: male: 2.02 [1.20, 3.50], female: 2.80 [1.80, 4.80], p(m/f) = 0.516). For TNF inhibitors (n=79), there was a significant difference between genders at V3 (Fig.1a). For Apremilast (n=39), there was a significant difference between genders at V2 (Fig.1c).

In PsA patients overall SASPA decreased from baseline (V1) to V2 and V3 (SASPA: V1: male: 4.00 [2.80, 5.20], female: 4.40 [2.80, 5.80], p(m/f) = 0.399; V2: male: 2.20 [1.20, 3.50], female: 3.40 [2.00, 5.00], p(m/f) = 0.071; V3: male: 1.80 [0.80, 2.70], female: 3.01 [2.35, 4.80], p(m/f) = 0.001). For TNF inhibitors (n=299), a significant difference in response to individual TNFi (etanercept, infliximab, other TNFi) measured by HAQ were investigated in all diseases together. The difference between male and females was significant at baseline for all 3 TNFi; possible differences of response to individual TNFi (etanercept, infliximab, other TNFi) measured by HAQ. Gender differences were also seen in response to Apremilast.

Conclusion: Female patients showed a statistically lower response to TNFi in all three disease entities (RA, SpA and PsA) to a variable degree in our homogeneous central European population. Interestingly, the difference was not uniform across individual TNFi when measured by HAQ. Gender differences were also seen in response to Apremilast.

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ASSOCIATION OF FIRST, SECOND, AND THIRD-LINE BDMARDS AND TSMDARD WITH DRUG SURVIVAL AMONG RHEUMATOID ARTHRITIS PATIENTS: A COHORT STUDY

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Background: Since tofacitinib is a tsDMARD with a different mechanism of action compared to bDMARDS, along with the fact that recent releases of bDMARDS or tsDMARDS have led to numerous possible switching patterns, there is a need for comparative studies of bDMARDS and tofacitinib with subsequent drug survival among RA patients.

Objectives: To determine the association of first, second, and third-line biologic disease- modifying antirheumatic drugs (bDMARDS) and tofacitinib with drug survival among rheumatoid arthritis (RA) patients.

Methods: The study population was composed of 8,018 seropositive RA patients who were prescribed bDMARDS or tofacitinib between January 2014 and January 2019 from the Korean Health Insurance Review and Assessment Service database. First, second, and third-line choice of tumor necrosis factor inhibitors (TNFi) including etanercept, infliximab, adalimumab, and golimumab, as well as non-TNFi including tocilizumab, rituximab, tofacitinib, and abatacept were assessed. Multivariate cox proportional hazards regression was used to determine the adjusted hazard ratios (aHRs) and 95% confidence intervals (CIs) for drug failure according to bDMARD or tofacitinib choice starting from the initial prescription date.

Results: Compared to first etanercept users, patients with first tocilizumab (aHR 0.56, 95% CI 0.46-0.68), tofacitinib (aHR 0.27, 95% CI 0.18-0.42), or abatacept (aHR 0.83, 95% CI 0.69-0.99) had lower risk of drug failure. Second choice of tocilizumab (aHR 0.38, 95% CI 0.29-0.55), tofacitinib (aHR 0.23, 95% CI 0.15-0.37), or abatacept (aHR 0.54, 95% CI 0.35-0.84) was associated with lower drug failure risk compared to second etanercept users. Finally, third choice of tocilizumab (aHR 0.32, 95% CI 0.16-0.62) or tofacitinib (aHR 0.35, 95% CI 0.19-0.63) was associated with lower drug failure risk compared to third TNFi users.

Conclusion: First and second-line tocilizumab, tofacitinib, or abatacept may lead to improved drug survival. Third choices of tocilizumab or tofacitinib may be beneficial in reducing drug failure risk among RA patients.

REFERENCES:

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Table 1. Risk of drug failure on rheumatoid arthritis patients according to first, second, and third choice of bDMARD or tofacitinib.

<table>
<thead>
<tr>
<th>Drug</th>
<th>First-line choice (n=8,018)</th>
<th>Second-line choice (n=1,645)</th>
<th>Third-line choice (n=853)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Events</td>
<td>Person-years</td>
<td>Incidence</td>
</tr>
<tr>
<td>Etanercept</td>
<td>290</td>
<td>2,396</td>
<td>121</td>
</tr>
<tr>
<td>Infliximab</td>
<td>236</td>
<td>1,417</td>
<td>167</td>
</tr>
<tr>
<td>Adalimumab</td>
<td>449</td>
<td>2,718</td>
<td>165</td>
</tr>
<tr>
<td>Golimumab</td>
<td>258</td>
<td>1,955</td>
<td>132</td>
</tr>
<tr>
<td>Tocilizumab</td>
<td>171</td>
<td>2,623</td>
<td>65</td>
</tr>
<tr>
<td>Tofacitinib</td>
<td>3</td>
<td>29</td>
<td>48</td>
</tr>
<tr>
<td>Abatacept</td>
<td>216</td>
<td>2,207</td>
<td>98</td>
</tr>
</tbody>
</table>

Adjusted hazard ratios calculated by multivariate Cox proportional hazards regression after adjustments for age, sex, hospital type, number of csDMARDs, first bDMARDs or tofacitinib for second and third-line choice analysis, Charlson comorbidity index, and enrollment year.

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REFERENCES:

Figure 1. Cumulative incidence of ILD in patients diagnosed with RA between 1999 and 2014, adjusted for the competing risk of death. Abbreviations. ILD: interstitial lung disease; RA: rheumatoid arthritis.

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