DO BIOLOGICAL DMARDS PREVENT LONG TERM ARTICULAR DAMAGE BY RHEUMATOID ARTHRITIS IN ROUTINE CLINICAL CARE?

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Background

Treatment of rheumatoid arthritis is primarily aimed to achieve remission and thereby reduce the risk of permanent joint damage. Preventing disabling joint damage is an important long term goal for patients. Effectiveness of bDMARDs on disease activity has been proven in randomized controlled studies; however studies on long term joint damage are scarce and show conflicting results (1,2).

Objectives

- Compare joint damage more than 5 years after RA diagnosis in patients who have never used bDMARDs and in patients treated with bDMARDs. Joint damage is measured with the Rheumatoid Arthritis Articular Damage (RAAD) score, a clinical method comprising all joints that may be affected by RA .
- Evaluate functional disability measured by Health Assessment Questionnaire (HAQ) score in patients who have never used bDMARDs and in patients treated with bDMARDs.

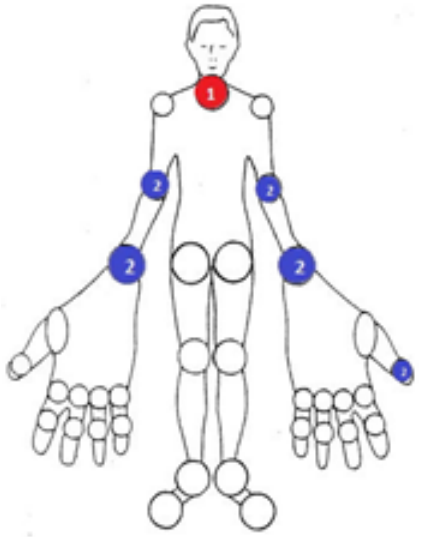
Methods

Retrospective cohort study in RA patients in a Dutch teaching hospital diagnosed between 2006 and 2011. Inclusioncriteria were receiving a DMARD for at least 12 months and availability of a RAAD-score after a disease duration of 5 years or more.

Exclusion criterion was receiving a DMARD for an other indication than RA. The exposure of interest was the type of treatment (sDMARD or bDMARD).

The RAAD-score: irreversible joint damage

Scores of 0 (no damage), 1 (mild) or 2 (ankylosis, luxation or joint surgery/prosthesis) are assigned to 35 joints (maximum score: 70).



	bDMARD n = 101	No bDMARD n = 204	P
Age, mean (SD)	49 (13)	57 (14)	<0.001
Female, N. (%)	63 (62.4)	129 (63.2)	0.884
Erythrocyte Sedimentation Rate (ESR), median (IQR)	22.0 (14.0 - 37.5)	27.5 (13.2 - 43.7)	0.174
C-Reactive Protein (CRP), median (IQR)	10.0 (3.0 - 21.0)	15.0 (4.0 - 36.0)	0.102
ACR 2010 ≥ 6, N. (%)	79 (78.2)	158 (78.2)	1.000
Rheumatoid Factor (RF) ≥ 19, N (%)	81 (81.0)	145 (71.1)	0.058
Antibodies to Cyclic Citrullinated Peptides (anti-CCP) ≥ 7, N (%)	54 (76.1)	90 (70.9)	0.432
Presence of rheumatoid nodulosis, N (%)	3 (3.2)	12 (6.2)	0.288
Presence of bone erosions, N (%)	17 (16.8)	46 (22.9)	0.222
DAS28 (BSE), median (IQR)	4.9 (4.3 - 5.6)	4.7 (3.8 - 5.5)	0.166

Results

Among 305 patients included, 101 used a bDMARD (Table). Baseline characteristics were not significantly different in terms of prognostic factors for joint damage progression, with the exception of patients in the bDMARD group being younger. During follow up, the highest DAS28 was higher in the bDMARD group 5.0 vs 4.3, p<0.001. After 8.5 years, more than half of the patients in bDMARD (55.4%) and sDMARD (57.4%) groups had no joint damage (RAAD-score 0). No significant differences in long term joint damage were observed, even after adjusting for potential confounders (b=-0.26, 95% CI -1.9-1.4, p = 0.76).

Follow-up characteristics and Outcomes	bDMARD n = 101	No bDMARD n = 204	P
RAAD, median (IQR)	0.0 (0.0 - 2.0)	0.0 (0.0 - 2.0)	0.787
HAQ, median (IQR)	0.3 (0.0 - 0.9)	0.1 (0.0 - 0.4)	0.002
Highest measured DAS (BSE), median (IQR)	5.0 (4.2 - 5.8)	4.3 (2.9 - 5.1)	<0.001
Disease duration, yrs, mean (SD)	8.5 (1.9)	8.5 (2.2)	0.703
Treatment period with DMARD, yrs, mean. (SD)	8.5 (2.1)	8.2 (2.6)	0.415
Prednis(ol)on, % N	57 (56.0)	63 (30.0)	<0.001

Conclusion

In this cohort joint damage is not significantly different between patients treated with sDMARD(s) alone and those also using a bDMARD. Given the higher disease activity in the bDMARD group it is possible that this treatment prevented long-term joint damage by RA. Since there was limited joint damage in these groups this analysis should be repeated after longer disease duration and preferably in randomised cohorts.

References

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