Conclusion: At 12 months, drug retention rate was 63.6% in patients receiving TCZ sc in real life, with no difference between Mono and Combo groups. EULAR good/moderate response was 69.0%. QoL improved in all EQ-5D domains. No new safety signal occurred.

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FRI0117

THE COMPARATIVE RISK OF OSTEOPOROTIC FRACTURES AMONG PATIENTS WITH RHEUMATOID ARTHRITIS RECEIVING TNF INHIBITORS VERSUS OTHER BIOLOGICS: A NATION-WIDE COHORT STUDY IN KOREA

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Background: Rheumatoid arthritis (RA) is associated with an increased risk of osteoporosis and osteoporotic fracture, but little is known about comparative risk of osteoporotic fractures between biologic agents.

Objectives: To investigate the comparative risk of osteoporotic fractures among RA patients who initiated TNF inhibitors, abatacept, and tocilizumab.

Methods: We identified patients aged ≥ 40 years with at least two ICD-10 diagnosis codes for RA who initiated TNF inhibitor, abatacept, or tocilizumab from the 2005-2015 Korean National Health Insurance Service datasets. The primary outcome was a composite osteoporotic fracture endpoint of spine, hip, forearm or humerus fractures requiring hospitalization. Secondary outcomes were spinal and non-spinal fractures. Follow-up period started from the day after the first dispensing date of the study drug to the earliest date among the following events: discontinuation of the study drugs, outcome occurrence, disenrollment, end of study dataset, or death. Propensity score (PS) matching with a variable ratio of 10:1 was conducted for TNF inhibitor versus abatacept initiators and for TNF inhibitor versus tocilizumab initiators to adjust for baseline confounding. We estimated hazard ratio (HR) and 95% confidence interval (CI) of osteoporotic fracture risks comparing TNF inhibitors to abatacept or tocilizumab by Cox proportional hazard models stratified by a matching ratio.

Results: We included 2,339 TNF inhibitor initiators and 594 abatacept initiators, and 2,486 TNF inhibitor initiators and 647 tocilizumab initiators in each PS-matched cohort. The incidence rates per 100 person-years for the primary outcome were 1.57 and 1.65 for TNF inhibitor and abatacept initiators, and 1.69 and 2.05 for TNF inhibitor and tocilizumab initiators, respectively. The HR (95% CI) for the primary outcome was 1.10 (1.54-2.23) comparing TNF inhibitors versus abatacept and it was 1.00 (0.53-1.92) comparing TNF inhibitor versus tocilizumab initiators. We also did not find any significant difference for secondary outcomes (Table1).

Table 1. Risk of osteoporotic fractures in TNF inhibitor initiators vs. abatacept initiators and tocilizumab initiators

	TNF inhibitor (n=2,339)			Abatacept (n=594)			HR (95% CI)
	Events	PYs	IR per 100 PY	Events	PYs	IR per 100 PY	
Primary outcome							
Osteoporotic fracture	47	2,998	1.57	12	727	1.65	1.10 (1.54-2.23)
Secondary outcomes							
Spinal fracture	25	3,018	0.83	8	731	1.10	0.95 (0.41-2.20)
Non-spinal fracture	22	3,022	0.73	4	737	0.54	1.52 (0.41-5.65)
	TNF inhibitor (n=2,468)			Tocilizumab (n=647)			HR (95% CI)
	Events	PYs	IR per 100 PY	Events	PYs	IR per 100 PY	
Primary outcome							

Osteoporotic fracture	54	3,196	1.69	16	781	2.05	1.00 (0.53-1.92)
Secondary outcomes							
Spinal fracture	29	3,221	0.90	10	788	1.27	0.98 (0.43-2.24)
Non-spinal fracture	25	3,225	0.78	6	793	0.76	1.03 (0.36-2.90)

Conclusion: We did not find a significant difference in the risk of osteoporotic fractures between TNF inhibitor and abatacept initiators, or between TNF inhibitor and tocilizumab initiators in this Korean population-based cohort study.

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FRI0118

INFECTION OUTCOMES IN PATIENTS WITH RA TREATED WITH ABATACEPT AND OTHER DMARDS: RESULTS FROM A 10-YEAR INTERNATIONAL POST-APPROVAL STUDY

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Background: The abatacept global post-marketing epidemiology programme consists of observational studies based on biologic disease registries and healthcare claims databases to assess infection and malignancy risks associated with abatacept treatment, as used in routine clinical practice.

Objectives: To evaluate the risk of infection in patients with RA treated with abatacept versus conventional synthetic (cs)DMARDs and other biologic (b) or targeted synthetic (ts)DMARDs.

Methods: Data were analysed from five cohorts: two biologic registries (the Anti-Rheumatic Therapy in Sweden register and the Rheumatoid Arthritis Observation of Biologic Therapy German registry), a disease registry (FORWARD, The National Databank for Rheumatic Diseases in the USA) and two healthcare claims databases (the population-based British Columbia Canadian RA Cohort and the US Optum Research Database). Crude incidence rates (per 1000 patient-years of exposure) with 95% CIs were calculated for hospitalised infections and opportunistic infections. Adjusted risk ratios (RRs) with 95% CIs were estimated using multivariate models adjusting for demographics, co-morbidities and other potential confounders within each database and were subsequently pooled using a random effects model for meta-analyses.¹

Results: From all cohorts, patients treated with abatacept (6400), csDMARDs (137K) and other b/tsDMARDs (54K) were followed up for a mean of 2.3–3.7, 2.2–6.2 and 2.3–4.7 years, respectively. Patients were mainly female (71–86%), with a mean age ranging from 49–63 years, and 3–18% had a history of prior severe infections across treatment groups/cohort. A greater number of abatacept-treated patients had a history of ≥ 2 prior biologics (44–85% vs csDMARDs, 11% [FORWARD] and other b/tsDMARDs, 0–19%). In abatacept-treated patients, incidence rates for hospitalised infections ranged from 16–56 and opportunistic infections from 0.4–7.8 (Table). Adjusted RRs (95% CIs) for abatacept vs csDMARDs (range: 0.3 [0.2, 0.7] to 2.2 [1.3, 3.7]; pooled estimate: 1.2 [0.6, 2.2]) and abatacept vs other b/tsDMARDs (range: 0.5 [0.3, 0.8] to 1.3 [0.8, 2.1]; pooled estimate: 0.9 [0.6, 1.3]) showed no increased risk in hospitalised infections.

Conclusion: In this large, international post-marketing epidemiology programme, pre-specified infection risks were more frequent with abatacept than csDMARDs and b/tsDMARDs in some, but not all, cohorts. Increased risks for individual outcomes/cohort may be attributed to prior biologic exposure, study design and practice pattern differences. These data are consistent with the current safety profile of abatacept.

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