negatively impacted sexual function in both male and female patients. Active treatment with FIL or ADA resulted in early and sustained improvements from baseline in sexual function.

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Table 1. Mean (standard deviation) baseline characteristics

	FINCH 1		FINCH 2		FINCH 3	
	Male n=258	Female n=990	Male n=79	Female n=270	Male n=233	Female n=691
Duration of RA, yr	6.8 (7.38)	8.0 (7.63)	11.3 (8.53)	12.7 (9.35)	2.1 (5.35)	2.2 (4.85)
SJC28	11 (5.0)	11 (5.1)	13 (6.2)	12 (6.1)	11 (5.4)	11 (5.7)
TJC28	14 (6.5)	15 (6.4)	15 (7.5)	16 (7.0)	14 (6.6)	15 (6.7)
HAQ-DI	1.35 (0.614)	1.64 (0.601)	1.41 (0.689)	1.73 (0.634)	1.37 (0.651)	1.62 (0.617)
DAS28 (CRP)	5.6 (0.95)	5.8 (0.90)	5.8 (1.08)	5.9 (0.92)	5.7 (1.00)	5.7 (0.99)
SF-36 PCS	34.3 (7.72)	33.0 (7.34)	31.7 (8.48)	30.9 (7.75)	34.4 (7.72)	33.4 (7.47)
SF-36 MCS	45.9 (10.15)	43.6 (10.65)	43.7 (11.20)	44.5 (11.71)	46.2 (11.75)	43.0 (10.89)
FACIT-fatigue	30.0 (10.00)	26.8 (10.49)	26.3 (11.15)	24.0 (11.64)	30.7 (10.93)	26.6 (10.89)
Patient global VAS (mm)	64 (19.8)	67 (19.0)	66 (20.0)	70 (19.5)	65 (22.4)	66 (20.3)
Pain VAS (mm)	61 (20.6)	66 (19.5)	62 (22.3)	68 (20.6)	64 (22.3)	66 (20.9)
VAS sexual function score	44 (30.2)	49 (32.3)	48 (34.6)	49 (36.8)	42 (34.4)	48 (35.1)

Variables in bold significantly correlated with VAS sexual function score ($P < 0.05$) DAS28 (CRP), Disease Activity Score-28 using C-reactive protein; FACIT, Functional Assessment of Chronic Illness Therapy; HAQ-DI, Health Assessment Questionnaire Disability Index; MCS, mental component summary; PCS, physical component summary; RA, rheumatoid arthritis; SF-36, 36-Item Short Form Survey; S/TJC28, swollen/tender joint count based on 28 joints; VAS, visual analogue scale

Figure. Change from baseline in VAS sexual function over time for female and male subgroups in FINCH 1

Patients receiving placebo at Week 24 were re-randomised 1:1 to receive filgotinib 200 mg or 100 mg (data not shown)
* $P \leq 0.05$, ** $P \leq 0.01$ vs placebo
ADA, adalimumab; CI, confidence interval; FIL, filgotinib; LS, least-squares; MTX, methotrexate; PBO, placebo; VAS, visual analogue scale

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ACUTE KIDNEY INJURY (AKI) IN PATIENTS WITH RHEUMATOID ARTHRITIS (RA)

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Background: Methotrexate has been an anchor drug for patients with rheumatoid arthritis (RA). However, it is strictly prohibited to prescribe MTX to patients with severely decreased renal function because it can induce a fatal adverse event such as pancytopenia in these patients. On the other hand, since the average age of RA patients is gradually increasing, and many of them already have mildly to moderately impaired renal function, their renal function can easily decrease to below the critical level of the estimated glomerular filtration rate. Therefore, new development of acute kidney injury (AKI) during MTX administration might induce a fatal adverse event, making the identification of patients susceptible to AKI very important.

Objectives: To clarify the frequency of AKI and the factors involved in it in RA patients.

Methods: Two hundred and fifty-two RA patients (211 females, 41 males, mean age 62.3 ± 12.5 years, disease duration 11.0 ± 9.5 years) diagnosed more than 3 years earlier and followed for more than 5 years, and also, others diagnosed ≥ 3 years earlier but followed for ≤ 5 years were enrolled. We measured BUN, Cr, RF and aCCP in patient serum, urinary proteins, urinary blood, and urinary casts and evaluated CDAI, SDAI, disease activity score (DAS) 28-CRP and DAS28-ESR. Steinbrocker functional classification and radiological grading were evaluated. History of diabetes mellitus, hypertension and hyperlipidemia was determined from the medical records. Medications for RA, including non-steroid anti-inflammatory drugs (NSAIDs), prednisolone, csDMARD (MTX, Tacrolimus, etc.), bDMARDs and tsDMARDs were evaluated. Estimated glomerular filtration rate (eGFR) was calculated by the new Japanese coefficient-modified Modification of Diet in Renal disease (MDRD) study equation. The criteria of AKI were that serum Cr increased by 0.3 mg/dl or increased by 1.5-fold between consecutive visits according to the KIDIGO criteria¹⁾ and the report of Leither et al²⁾.

Results: Twenty (7.9%) patients developed AKI, 22 times. The causes of AKI were 10 infections, 6 dehydrations, 2 enteritis, 1 urticaria, 2 hypercalcemia due to VitD administration, and 1 ureteral stone. We divided our patients into group A (with AKI) and group B (without AKI). Group A was older (69.9 ± 10.1 vs 61.7 ± 12.6 years), had greater physician VAS (29.5 ± 27.7 vs 15.7 ± 18.3 mm), higher serum creatinine (0.79 ± 0.19 vs 0.60 ± 0.16 mg/dl), higher BUN (18.4 ± 5.7 vs 15.1 ± 4.4 mg/dl), lower eGFR (65.5 ± 23.3 vs 86.4 ± 22.4 ml/min), more frequent prednisolone administration (75.0% vs 41.9%), more frequent hyperlipidemia (50.0% vs 19.2%) and more frequent hypertension (60.0% vs 30.6%) than Group B by univariate analysis significantly ($p < 0.01$). We then performed multifactorial analysis using logistic regression analysis. Greater physician VAS (OR 1.02, 1.00-1.04), lower eGFR (OR 1.04, 1.01-1.08) and prednisolone administration (OR 3.29, 1.02-10.63) were found as independent relevant factors for group A.

Conclusion: Our study indicated that AKI developed in RA patients and suggested that renal function decline and prednisolone administration may be implicated. RA patients with impaired renal function and prednisolone administration need to be treated with special attention to the onset of AKI.

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