

Treatment outcomes of tofacitinib in rheumatoid arthritis patients previously on methotrexate in a hospital in Kisumu, Western Kenya

Abstract

Introduction: JAK inhibitors are a newer class of drugs for treating Rheumatoid arthritis and they are currently in use in many continents. Their use in sub-Saharan Africa has rarely been documented in referred journals.

Objective: To assess and document the clinical outcomes of Rheumatoid arthritis patients treated with tofacitinib after methotrexate in a hospital in Kisumu, Western Kenya.

Methodology: This was a retrospective chart review of patients who had previous been treated with methotrexate based combinations with poor outcomes or intolerance, and had completed at least 6 months of tofacitinib treatment. CDAI scores were done at baseline and at 6 months post-treatment. Treatment side effects were evaluated from the data and analysed.

Results: There was significant improvement of CDAI scores after 6 months treatment with tofacitinib with minimal serious infections.

Conclusion: Tofacitinib is efficacious in African populations.

Keywords: methotrexate, tofacitinib, treatment failure, improvement, infections

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Introduction

JAK inhibitors are a newer class of drugs used to treat rheumatoid arthritis. Much of sub-Saharan Africa, access to biologic and targeted synthetic DMARDs (including JAK inhibitors) remains constrained, with low penetration largely driven by availability and affordability barriers.¹ Robust real-world JAK-inhibitor data from the region are scarce, highlighting the importance of collating data from local cohorts. In this study, we sought to evaluate 6-month clinical response rates to tofacitinib in a real-world cohort.

Methodology

We reviewed data of patients who had either had poor treatment outcomes or intolerance to methotrexate (Table 1) and who had been started on tofacitinib and had completed at least 6 months of treatment. The Clinical Disease Activity Scores (CDAI) were recorded at baseline and at 6 months of tofacitinib treatment. Records showed 17 patients were available but 3 had missing data, while 2 had not completed the mandatory 6 months treatment. Hence the study included 12 patients with complete CDAI values at baseline and six months (Figure 1).

Table 1 Patient characteristics at baseline and at 6 months

Case	Age	Sex	Rheumatoid Factor status	Reason for Starting tofacitinib	Dapsone Or Septrin use	Baseline CDAI	6-Month CDAI	Anti-CCP Titres	JAK Inhibitor side effects
1	43	F	negative	intolerance	septrin	42	12	207	sepsis
2	72	F	positive	Failure	septrin	45	18	12	none
3	80	F	positive	Failure	dapsone	41	17	400	none
4	46	F	positive	Failure	septrin	44	20	340	none
5	30	F	positive	Failure	septrin	38	17	200	none
6	50	F	positive	Failure	septrin	35	13	380	none
7	50	F	positive	Failure	septrin	36	9	65	none
8	72	F	negative	Failure	septrin	36	18	18	none
9	67	F	positive	Failure	septrin	38	13	107	none
10	65	F	negative	Failure	dapsone	40	18	200	none
11	64	F	negative	Failure	septrin	40	17	64	none
12	58	F	positive	Failure	septrin	29	10	102	none

Table 2 Baseline characteristics and 6-month clinical outcomes (CDAI and age) in patients treated with tofacitinib

Variable	Mean	SD	Min	Max
Baseline CDAI	38.7	4.4	29	45
6-month CDAI	15.2	3.6	9	20
CDAI % Change	-61	7.4	-75	-50
Age (years)	58.1	14.5	30	80



Figure 1 Flow chart of recruitment of subjects for the study.

Results

The cohort had a mean age of 58.1 years (put interquartile range). The cohort was 100% female (12/12) and the major reason for starting tofacitinib was treatment failure on methotrexate, 91.7% (11/12). Only one patient (8.3%) was found to have been intolerant to methotrexate. Rheumatoid factor positivity was observed in two-thirds of patients (66.7%), while anti-CCP antibody, which was positive in the entire cohort, was significantly raised (mean 174.6 IU/ml, SD 137.4), consistent with seropositive rheumatoid arthritis and reflective of greater disease severity in this cohort.

Mean CDAI decreased from 38.7 (SD 4.38) at baseline to 15.2 (SD 3.59) at 6 months. This represented a mean reduction of 61.0% (SD 7.4), with individual improvements ranging from -75.0% to -50.0%. The mean within-patient reduction was -23.5 points (95% CI -25.7 to -21.3), which was statistically significant (paired t-test, $t=-23.6$, $df=11$, $p<0.001$). This demonstrates substantial improvement in disease activity over the treatment period, with all patients achieving at least a 50% reduction in CDAI.

Paired t test

Variable	Obs	Mean	Std. err.	Std. dev.	[95% conf. interval]	
monthC~I	12	15.16667	1.035969	3.588703	12.88651	17.44682
BASELI~I	12	38.66667	1.263313	4.376244	35.88613	41.4472
diff	12	-23.5	.9962049	3.450955	-25.69263	-21.30737
mean(diff) = mean(monthCDAI - BASELINECDAI)						t = -23.5895
H0: mean(diff) = 0						Degrees of freedom = 11
Ha: mean(diff) < 0						Pr(T < t) = 0.0000
Ha: mean(diff) != 0						Pr(T > t) = 0.0000
Ha: mean(diff) > 0						Pr(T > t) = 1.0000

Discussion

This is one of the few studies on tofacitinib use after methotrexate failure to come out of Africa. Our cohort was predominantly elderly females in their late fifties. The tolerance to tofacitinib was good as shown with only one person (8.1%) getting a serious infection.

To contextualise infection risk, current guidance for autoimmune inflammatory rheumatic diseases recommends *Pneumocystis jirovecii* pneumonia (PJP) prophylaxis in selected high-risk patients, and trimethoprim-sulfamethoxazole (TMP-SMX/Seprin) is effective for PJP prevention across non-HIV immunocompromised populations; small rheumatology cohorts also support low-dose regimens.^{2,3} JAK inhibitors carry increased infection risk in some settings, reinforcing a risk-stratified prophylaxis approach.⁴

In our series, most patients received prophylaxis (Seprin 83.3%, Dapsone 16.7%), and we observed only one severe infection (pneumonia requiring hospitalisation) that occurred three weeks after Seprin was stopped, with no mortality, findings that, despite the small sample, support the real-world effectiveness and tolerability of tofacitinib under pragmatic prophylaxis. Case reports further document PJP after tofacitinib initiation, highlighting the relevance of vigilance and targeted prophylaxis policies.⁵ There were no infection related mortalities within the first 6 months of treatment. These results suggest that tofacitinib led to marked clinical improvement within six months in this cohort.⁶⁻⁸ Despite the limited sample size, our data contribute important real-world evidence on the use of JAK inhibitors in a resource-limited setting.

Study limitations

Two limitations were evident in our study: firstly, the study sample was small. Secondly, only one investigator was available for patient care and therefore the discriminant factor between CDAI scores couldn't be calculated.

Declaration

Patient care: Charles Omondi

Author Contribution: Charles Omondi- Study design, data collection, writing of draft manuscript, review of draft manuscript, review of final manuscript.

Sajeev Parmar: Study design, generation of draft manuscript, review of final manuscript.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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