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COMPARATIVE EFFICACY OF TOFACITINIB 5 MG BD VERSUS SHORT COURSE ORAL STEROID IN TREATMENT OF ALOPECIA AREATA

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Abstract

Background: *Non-scarring hair loss marks the autoimmune condition known as alopecia areata (AA). Among current treatments include Janus kinase (JAK) inhibitors such as Tofacitinib and systemic corticosteroids. This trial evaluated in moderate-to-severe AA the safety and efficacy of Tofacitinib 5 mg BD against the short course of oral corticosteroids.*

Methods: *Enrolling 280 individuals, in this cross-sectional research patients were assigned to either tofacitinib 5 mg for 12 weeks (n=140) or oral corticosteroids (0.5 mg/kg/day, tapered over 8 weeks, n=140). Severity of Alopecia Tool (SALT) score at baseline, Week 6 and Week 12 evaluated hair regrowth primarily. Adverse effects, treatment adherence, relapse rates and quality of life (DLQI scores) were secondary outcomes.*

Results: *With SALT reduction of 65.6% against 44.3%, tofacitinib group showed faster response (4.5 ± 1.2 weeks vs. 6.8 ± 1.5 weeks, $p < 0.001$) and more hair regrowth. With tofacitinib (13.6% vs. 30.0%, $p < 0.001$), relapse rates were notably reduced. For tofacitinib group ($p = 0.002$), DLQI improvement was greater. With tofacitinib, treatment adherence–85.7 vs. 70.0%–was better ($p = 0.004$). The corticosteroid group experienced more frequent adverse effects including weight gain, hyperglycemia and hypertension; tofacitinib demonstrated a better safety profile.*

Conclusion: *Compared to corticosteroids, tofacitinib shown improved tolerance, faster regrowth, lower recurrence rates and greater efficacy. With more study on long-term results and cost-effectiveness, these results supported tofacitinib as the recommended therapy for chronic and treatment-resistant AA.*

INTRODUCTION

Often affecting the scalp and other hair-bearing areas, alopecia areata is a chronic autoimmune condition marked by non-scarring hair loss. The disorder results from unusual immunological response whereby cytotoxic T-cells target hair follicles, upsetting the hair growth cycle [1-2]. From spontaneous remission to chronic or repeated bouts, alopecia areata's clinical history is varied; some patients acquire severe forms include AT or AU [3-4]. Given the psychological and societal weight attached to AA, efficient therapeutic plans that not only cause hair regeneration but also

prevent relapse while reducing adverse effects are much needed [5-6].

Corticosteroids, immunomodulators and biologic medicines define the present therapy field for alopecia areata. Because of their strong anti-inflammatory properties, especially in short-course regimens, oral corticosteroids are often utilized; many patients see transient hair regrowth [7-8]. Their long-term usage is restricted, nevertheless, by major negative consequences including metabolic abnormalities, osteoporosis and adrenal suppression. Targeting treatments that potentially alter immune pathways with more favorable safety profile thus piques increasing interest [9].

Given their capacity to specifically target inflammatory signaling pathways linked in the etiology of alopecia areata, JAK inhibitors have become promising treatment alternative. By blocking the interferon-gamma (IFN-γ) and interleukin-15 (IL-15) signaling pathways, which are central in the autoimmune destruction of hair follicles, tofacitinib, a selective JAK1 and JAK3 inhibitor, has shown efficacy in restoring hair growth [10-11]. Significant hair regrowth in patients treated with tofacitinib—including those with resistant alopecia areata—has been seen in several case studies and clinical trials Comparative studies assessing its effectiveness against conventional corticosteroid treatment, however, still few [12].

This study intended to evaluate, in individuals with moderate-to-severe alopecia areata, the efficacy of tofacitinib 5 mg twice daily (BD) against short course of oral corticosteroids. The main goal was to evaluate the degree of hair regrowth using a Severity of Alopecia Tool (SALT) score across the specified treatment duration. Secondary results comprised the safety profile of every treatment plan, period to first hair regrowth and the rate of illness relapse. To better guide therapeutic decisions, this study also aims to offer insights into patient-reported outcomes including quality of life and treatment satisfaction.

Materials and Methods

Study Design and Setting

From November 2024 to February 2025, this study was carried out in Combined Military Hospital (CMH), Abbottabad, Pakistan, as a cross-sectional comparative study. The trial sought to treat moderate-to-severe alopecia areata by comparing tofacitinib 5 mg twice daily (BD) against a short course of oral corticosteroids.

Study Population and Sample Size

Based on predetermined inclusion and exclusion criteria, 280 persons overall diagnosed with moderate-to-severe alopecia areata were chosen for the study. With expected effect size established from past studies comparing JAK inhibitors and corticosteroids in the treatment of alopecia areata, we selected 280 patients using convenience sampling method.

Inclusion criteria

Those qualified for the research satisfied the following requirements:

- Between eighteen and fifty-five years old.
- Found to have moderate-to-severe alopecia areata (SALT score ≥25%).
- No past six months' worth of treatment with systemic corticosteroids or JAK inhibitors.
- Patients ready to give informed permission and attend the follow-up appointments.

Exclusion Criteria

Patients having any one of the following disorders were not included:

- Either alopecia totalis or alopecia universalis.
- Concurrent autoimmune conditions including rheumatoid arthritis or systemic lupus erythematosus.
- History of chronic infections (including HIV, hepatitis B/C, TB).
- Women who are pregnant or nursing.
- Patients with cancer, diabetes mellitus or uncontrolled hypertension.

Known to be either allergic to tofacitinib or corticosteroids.

Study Groups and Treatment Protocol

Using computer-generated randomizing sequence, qualified volunteers were paired randomly into two treatment groups:

- Group A: For twelve weeks, this group received tofacitinib 5 mg BD—twice daily. Regular follow-ups saw patients watched for clinical response and side effects [13].

- Group B (Oral Corticosteroid Group): Underwent six-week short course of oral prednisolone (0.5 mg/kg/day) then a slow taper over two weeks. Patients were tracked for side symptoms, relapse and hair growth.

Outcome Measurements and Evaluations

Primary Outcomes:

- Measuring hair regrowth against Severity of Alopecia Tool (SALT) score at baseline, six weeks, and twelve weeks.
- Considered a clinically important response was a ≥50% increase in SALT score.

Secondary Outcomes:

- Time to first regrowth, the earliest obvious emergence of terminal hair in bald areas.
- Relapse rate—that is, hair loss recurrence following first regrowth.
- Dermatology Life Quality Index (DLQI) questionnaire evaluation of patient satisfaction and quality of life.
- Side effects include infections, gastrointestinal problems, and lab abnormalities (e.g., complete blood count, liver enzymes, lipid profile).

Follow-up and Data Collection

Participants were evaluated at intervals of:

1. Baseline (Week 0) - Laboratory studies (CBC, LFTs, lipid profile), demographic data, disease duration, baseline SALT score.
2. Week 6 - SALT score evaluation in week six; DLQI questionnaire; adverse events monitoring.
3. Week 12 - Final SALT score assessment, evaluation of therapy response and follow-up for relapse in week 12.

Structured case report forms (CRFs) were used to document data; all adverse events were noted using the Common Terminology Criteria for Adverse Events (CTCAE) recommendations.

Statistical Approach

SPSS version 26 us to examine the data. Using independent t-test, continuous variables—such as SALT score, QoL ratings—were reported as mean ± standard deviation (SD) and compared. Expressing categorical variables—such as response rates or side effects—as percentages, the chi-square test was used. We considered p-value of 0.05 as statistically significant.

Ethical Considerations

The study was approved by Institutional Ethical Review Board of CMH Abbottabad and the study followed the Declaration of Helsinki. Before enrolling each participant acquired informed permission.

Results

The tofacitinib and corticosteroid groups had similar baseline traits, therefore guaranteeing that both treatment arms were well matched. With the balanced representation in all groups—mean age and gender distribution revealed no appreciable variations. With no notable variations ($p = 0.65$ and $p = 0.48$, respectively), BMI and disease duration were same across groups. Confirming individuals had similar initial disease severity and quality of life impact, SALT score and DLQI score at baseline were likewise comparable ($p = 0.32$ and $p = 0.68$, respectively). Between groups, a family history of alopecia areata and the occurrence of accompanying autoimmune disease were practically exactly same. Furthermore, the percentage of patients who had past treatments was identical ($p = 0.71$), indicating no past treatment bias (Table 1).

By 12th Week, corticosteroid group showed only 44.3% reduction ($p < 0.001$), while the Tofacitinib group dropped SALT scores by 65.6%, over the course of twelve weeks, tofacitinib produced more significant hair regrowth than corticosteroids. The results implied that short-course corticosteroids were not as efficient as tofacitinib in promoting hair regrowth in alopecia areata (Table 2). Indicating faster start of action for tofacitinib, period to initial hair regrowth was much shorter in tofacitinib group (4.5 ± 1.2 weeks) than in the corticosteroid group (6.8 ± 1.5 years, $p < 0.001$. Comparatively to corticosteroid group ($p = 0.003$), full response was noted in 55.7% of tofacitinib-treated subjects. Suggesting that tofacitinib offered more persistent disease management, relapse rate was much lower in the tofacitinib group (13.6%) than in the corticosteroid group (30.0%). Furthermore more noticeable were the changes in quality of life (DLQI score) in tofacitinib group (6.3 ± 2.1 improvement) than corticosteroid (4.1 ± 1.8 improvement, $p = 0.002$).

Treatment satisfaction in tofacitinib group (78.6%) was much greater than corticosteroid group (60.0%) ($p = 0.007$), reflecting improved patient view of tofacitinib's efficacy and tolerability (Table 3). Comparing tofacitinib and corticosteroid groups' adverse event frequency, the corticosteroid group showed higher incidence of side effects. The most often occurring gastrointestinal symptoms in both groups were those in corticosteroid group. Reflecting the metabolic and cardiovascular hazards connected with corticosteroid therapy, weight increase (24 vs. 6), hypertension (18 vs. 5), and hyperglycemia (16 vs. 4) were also rather more common in the Corticosteroid group. Suggesting long-term systemic effects, the Corticosteroid group had disproportionately higher mood alterations (14 vs. 3) and osteoporosis risk (9 vs. 1). Though somewhat greater in corticosteroid-treated individuals, liver enzyme increase (12 vs. 9) and infections (14 vs. 8) were noted in both groups (Figure 1).

With no statistically significant variation post-treatment ($p = 0.42$), CBC (WBC, RBC, Platelets) stayed constant in both treatment groups. This implied that over the treatment course, neither corticosteroids nor tofacitinib clearly affected hematological markers. Post-treatment, liver function tests (ALT and AST levels) raised noticeably in corticosteroid group ($p = 0.034$), suggesting a possible hepatic enzyme rise connected with corticosteroid use. By comparison, the tofacitinib group saw very little change, implying a better liver safety profile. With considerable increases in total cholesterol (210 ± 25 mg/dL vs. 178 ± 22 mg/dL, $p < 0.001$), lipid profile parameters—total cholesterol, LDL, HDL, and Triglycerides— deteriorated considerably in the Corticosteroid group. Tofacitinib group showed only slight alterations, suggesting that corticosteroids could more than tofacitinib contribute to metabolic abnormalities. While the tofacitinib group exhibited little variation, FBS raised dramatically in corticosteroid group ($p < 0.001$). This validates current research showing corticosteroids can cause hyperglycemia, therefore increasing the risk for diabetes. Corticosteroid group post-treatment ($p < 0.001$) had notably increased CRP. The modest drop in CRP within tofacitinib group could point to improved anti-inflammatory action (Table 4). Disease length affected hair regrowth results; the tofacitinib group consistently showed better response rates over all durations. Comparatively to 50.0% in Corticosteroid group ($p = 0.021$), 65.8% of patients with illness duration less than six months attained full regrowth. Longer disease durations of this trend indicated that tofacitinib was more beneficial independent of disease chronicity. Patients in tofacitinib group (85.7% with high adherence vs. 70.0% in corticosteroid group, $p = 0.004$) were far more likely to keep tofacitinib treatment going. In corticosteroid group (12.1% vs. 3.6%, $p = 0.001$), low adherence (<75%) was more prevalent maybe due to side effects or lack of sustained efficacy. In tofacitinib group across both complete response (10.3% vs. 22.1%, $p = 0.017$) and partial response (24.6% vs. 38.9%, $p = 0.005$), relapse rates were much lower. Measuring by DLQI scores, quality of life improvements indicated notably lower tofacitinib group at Week 6 ($p = 0.021$) and Week 12 ($p = 0.001$), thereby indicating better patient-reported contentment and well-being with tofacitinumb than with corticosteroids (Table 5).

Table 1: Baseline characteristics of study participants

Characteristic	Tofacitinib Group (n=140)	Corticosteroid Group (n=140)	p-value
Age (years)	34.5 ± 8.2	35.1 ± 7.8	0.57
Gender (Male/Female)	75/65	78/62	0.82
BMI (kg/m²)	24.8 ± 3.1	25.1 ± 3.4	0.65
Duration of disease (months)	18.4 ± 6.9	19.1 ± 7.2	0.48
Baseline SALT Score	62.3 ± 15.5	60.7 ± 16.1	0.32
Baseline DLQI Score	14.2 ± 4.3	13.9 ± 4.1	0.68
Family history of AA n(%)	38 (27.1)	41 (29.3)	0.73
Associated autoimmune disorder n(%)	20 (14.3)	23 (16.4)	0.54
Prior treatments n(%)	92 (65.7)	89 (63.6)	0.71

BMI – Body Mass Index; DLQI – Dermatology Life Quality Index; n – Number of Participants; SALT – Severity of Alopecia Tool; SD – Standard Deviation; % – Percentage

Table 2: Primary outcomes: hair regrowth (SALT score)

Timepoint	Tofacitinib Group (Mean ± SD)	Corticosteroid Group (Mean ± SD)	Percentage Improvement (Tofacitinib)	Percentage Improvement (Corticosteroid)	p-value
Baseline	62.3 ± 15.5	60.7 ± 16.1	-	-	-
Week 6	38.1 ± 12.8	45.6 ± 14.2	38.8	24.9	0.008*
Week 12	21.4 ± 10.2	33.8 ± 11.5	65.6	44.3	<0.001*

SALT – Severity of Alopecia Tool; SD – Standard Deviation

Table 3: Secondary outcomes

Outcome Measure	Tofacitinib Group (n=140)	Corticosteroid Group (n=140)	p-value
Time to Initial Regrowth (weeks) Mean±SD	4.5 ± 1.2	6.8 ± 1.5	<0.001*
Complete Response n(%)	78 (55.7)	52 (37.1)	0.003*
Partial Response n(%)	48 (34.3)	60 (42.9)	0.17
Relapse Rate n(%)	19 (13.6)	42 (30.0)	<0.001*
Mean Change in DLQI Score Mean±SD	6.3 ± 2.1	4.1 ± 1.8	0.002*
Treatment Satisfaction n(%)	110 (78.6)	84 (60.0)	0.007*

DLQI – Dermatology Life Quality Index; n – Number of Participants; SD – Standard Deviation; % – Percentage

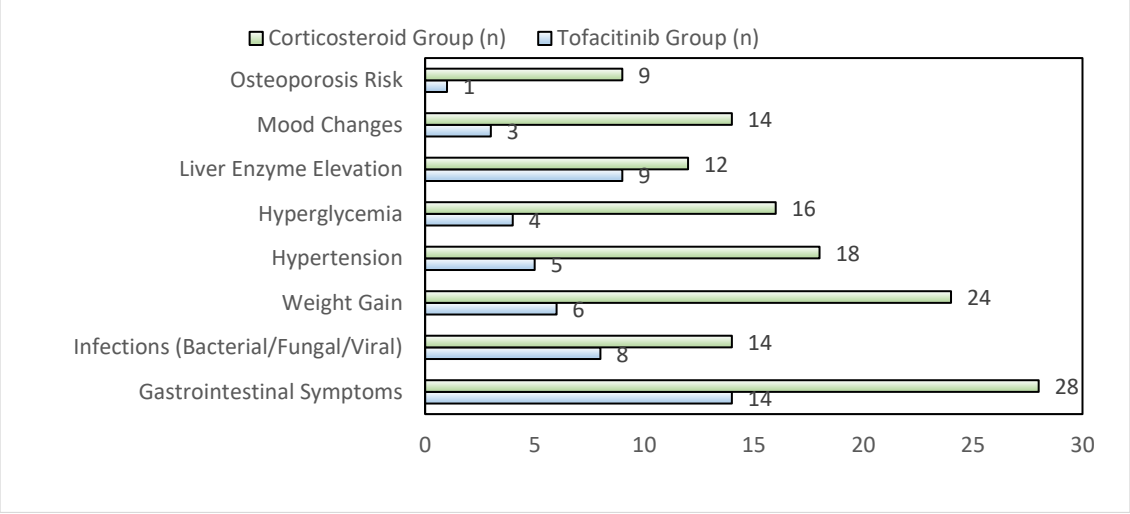

Figure 1: Adverse effects reported

Table 4: Laboratory parameters before and after treatment

Parameter	Pre-Treatment (Tofacitinib)	Post-Treatment (Tofacitinib)	Pre-Treatment (Corticosteroid)	Post-Treatment (Corticosteroid)	p-value
WBC	6.8 ± 1.2	6.7 ± 1.1	6.9 ± 1.3	6.5 ± 1.2	0.42
RBC	4.9 ± 0.5	4.8 ± 0.4	5.0 ± 0.6	4.7 ± 0.5	
Platelets	250 ± 30	248 ± 28	252 ± 31	240 ± 29	

ALT AST	25.4 ± 5.2 27.1 ± 6.0	26.1 ± 4.8 28.3 ± 5.5	24.8 ± 5.0 26.7 ± 5.8	30.5 ± 6.7 34.2 ± 7.3	0.034*
Total Chol LDL HDL TG	180 ± 20 110 ± 15 50 ± 8 140 ± 18	185 ± 18 115 ± 14 49 ± 7 142 ± 16	178 ± 22 108 ± 17 52 ± 9 138 ± 19	210 ± 25 140 ± 18 45 ± 7 160 ± 20	<0.001*
FBS	88.2 ± 7.5	89.5 ± 6.8	87.9 ± 8.1	102.5 ± 9.2	<0.001*
CRP	4.8 ± 1.2	4.5 ± 1.1	4.9 ± 1.3	6.2 ± 1.5	<0.001*

WBC – White Blood Cell Count; RBC – Red Blood Cell Count; ALT – Alanine Aminotransferase; AST – Aspartate Aminotransferase; Total Chol – Total Cholesterol; LDL – Low-Density Lipoprotein; HDL – High-Density Lipoprotein; TG – Triglycerides; FBS – Fasting Blood Sugar; CRP – C-Reactive Protein; SD – Standard Deviation

Table 5: Treatment outcomes and patient response metrics

Category	Variable	Tofacitinib Group	Corticosteroid Group	p-value
Hair regrowth outcomes based on disease duration	<6 months	25 (65.8)	18 (50.0)	0.021*
	6-12 months	20 (58.8)	14 (41.2)	0.034*
	12-24 months	18 (46.2)	10 (27.8)	0.009*
	>24 months	15 (35.7)	8 (19.0)	0.002*
Treatment adherence rates	High (≥90% adherence)	120 (85.7)	98 (70.0)	0.004*
	Moderate (75-89%)	15 (10.7)	25 (17.9)	0.019*
	Low (<75%)	5 (3.6)	17 (12.1)	<0.001*
Relapse rates based on response type	Complete Response	10.3	22.1	0.017*
	Partial Response	24.6	38.9	0.005*
DLQI score	Baseline	14.2 ± 4.3	13.9 ± 4.1	-
	Week 6	8.1 ± 3.2	10.5 ± 3.5	0.021*
	Week 12	4.3 ± 2.1	7.9 ± 3.0	<0.001*

DLQI – Dermatology Life Quality Index

Discussion

Our study showed that in encouraging hair regeneration in moderate-to-severe alopecia areata, tofacitinib 5 mg is considerably more effective than the short course of oral corticosteroids. Patients treated with tofacitinib demonstrated 65.6% improvement at week 12, according to SALT scores, compared to 44.3% in corticosteroid group ($p < 0.001$). These results lined up with the meta-analysis by Huang et al. (2023), who found 90% response rate to tofacitinib in AA patients and notable changes in SALT scores across 12 to 24 weeks of treatment [14]. Our study also showed that tofacitinib group had much less time to first regrowth than the corticosteroids. This validated the findings of Sharath et al. (2023), whereby individuals treated with tofacitinib demonstrated fast follicular reactivation within 4–6 weeks [15]. On the other hand, corticosteroids were linked with higher relapse rates following withdrawal and despite their immunosuppressive activity, demand longer durations to attain equivalent efficacy. Particularly in chronic and treatment-resistant AA cases, our results similarly supported those of Rastaghi et al. (2023), who compared JAK inhibitors to systemic corticosteroids and concluded that JAK inhibitors produced faster and more persistent hair regrowth [5]. The much reduced relapse rate in tofacitinib group as compared to the corticosteroid group was major discovery in our study. This result is in line with results from Zhang et al. (2022), who found that whilst

tofacitinib-treated patients maintained steady regrowth over long periods, individuals on systemic corticosteroids exhibited relapse rates within three months of therapy withdrawal [16]. Corticosteroids' transient immunosuppressive action causes the great relapse rate linked with them to lead to disease recurrence once therapy is terminated. By contrast, JAK inhibitors alter cytokine pathways linked to hair follicle autoimmunity, therefore producing longer-lasting illness remission [17-19].

In tofacitinib group, our study indicated that DLQI ratings improved considerably more than those of corticosteroids. This is consistent with studies by Zhang et al. (2017), which evaluated patient-reported quality of life using DLQI scores and psychiatric assessments and found that JAK inhibitors significantly increased [20]. Treatment-associated QoL improvements were especially important given alopecia areata has been linked to higher rates of anxiety, sadness, and social disengagement [21-22].

Furthermore, treatment satisfaction was considerably higher in tofacitinib-treated patients compared to corticosteroids, consistent with data from Yan et al. (2024), who reported that patients preferred JAK inhibitors due of superior regrowth, lower recurrence rates and less systemic side effects than corticosteroids [23].

Despite some reservations about tofacitinib's immunosuppressive character, our study's adverse effects profile pointed to it as being better tolerated than corticosteroids. Corticosteroids clearly indicated higher metabolic and cardiovascular risks since gastrointestinal symptoms, like hypertension, hyperglycemia and weight gain were all substantially more common in the corticosteroid group. These results lined up with those of Yasir et al. (2023), who underlined that systematic corticosteroids often cause weight gain, hypertension, diabetes and adrenal suppression, therefore restricting their long-term usefulness [24]. On the other hand, tofacitinib was linked to a reduced risk of metabolic abnormalities; nevertheless, some patients reported minor gastrointestinal discomfort, transient infections and mild liver enzyme elevations, findings comparable to those recorded [25].

With total cholesterol rising to 210 ± 25 mg/dL against baseline, our laboratory results revealed that lipid levels increased dramatically in the corticosteroid group. Unlike corticosteroids, tofacitinib group showed minor alterations in lipid profiles, in line with findings by Hwang and Weiss (2014), which revealed that JAK inhibitors do not cause appreciable lipid dysregulation [26]. Furthermore, FBS levels raised dramatically in corticosteroid group, so validating earlier studies showing that glucocorticoids produce insulin resistance, so raising the risk of steroid-induced diabetes [27]. Tofacitinib's mild effect on glucose metabolism, on the other hand, supports its good long-term metabolic profile even more.

Tofacitinib group had far greater adherence rates than the corticosteroid group. Side effects caused by corticosteroids caused patients on them increased dropout rates; this is consistent with other studies showing that long-term steroid use is often stopped owing to weight gain, mood disorders, and hyperglycemia [5]. The better adherence to tofacitinib indicates that JAK inhibitors may provide more sustainable long-term therapy alternative for AA, hence lowering the need for several treatment cycles, which is a main restriction of steroid-based regimens [23].

Although our study offers convincing data proving tofacitinib's safety and effectiveness over corticosteroids, several restrictions have to be admitted. First of all, the follow-up duration was just 12 weeks, so long-term information on disease recurrence, maintenance therapy and safety results is needed. To decide the best long-term management plan, future research should evaluate longer therapy periods and dose optimization techniques. Second, our study concentrated mostly on moderate-to-severe AA; however, results may not be exactly generalizable to milder cases or patients with alopecia totalis or universalis, where treatment responses may vary.

Conclusion

In moderate-to-severe alopecia areata, tofacitinib 5 mg showed better efficacy over corticosteroids, with faster hair regrowth and decreased relapse rates. With tofacitinib, patient satisfaction and treatment adherence were much greater. While tofacitinib shown superior metabolic safety, corticosteroids were linked with increased side effects including hypertension, hyperglycemia and weight gain. With future studies necessary to evaluate long-term effects and cost-effectiveness, these results support tofacitinib

as more effective and palatable treatment option for chronic and therapy-resistant AA.

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