Received: 29 September 2020 | Revised: 8 January 2021 | Accepted: 21 January 2021

DOI: 10.1111/jocd.13966

ORIGINAL CONTRIBUTION

JCD Journal of Cosmetic Dermatology WILEY

Experience with oral tofacitinib in severe alopecia areata with different clinical responses

Didem Dincer Rota MD[1] | Mehmet Ali Can Emeksiz MD, PhD[1] | Fatma Gulru Erdogan MD[1] | Dilsun Yildirim MD[2]

[1]Department of Dermatology, Faculty of Medicine, Ufuk University, Ankara, Turkey

[2]Department of Dermatology, Silifke State Hospital, Mersin, Turkey

Correspondence
Mehmet Ali Can Emeksiz, Department of Dermatology, Department of Dermatology, Ufuk University Faculty of Medicine, Mevlana Avenue No:86-88 06520 Balgat Cankaya Ankara Turkey.
Email: canemeksiz@yahoo.com

Abstract

Background: Alopecia areata (AA) and generalized form, universalis (AU) are common causes of noncicatricial alopecia, targeting anagen hair follicles. A dominant interferon-gamma transcriptional signaling and cytotoxic T lymphocytes were accused as the main drivers of disease pathogenesis. Tofacitinib is a Janus kinase inhibitor that has been proven to interfere with the positive feedback loop between the follicular cell and the cytotoxic T lymphocytes in AA. There is an increasing number of studies reporting success with tofacitinib in AA.

Aims: We aimed to assess oral tofacitinib's safety and efficacy in 13 recalcitrant AA and AU patients.

Methods: This is a retrospective pilot study performed between 2017 and 2020. The demographic features and the treatment responses were evaluated with Severity of Alopecia Tool score changes.

Results: Thirteen recalcitrant alopecia areata patients (3 AA, 10 AU), aged between 17 and 49, were included in the study. The treatment duration was 3-15 months. All three AA patients responded well; however, the therapy was unsuccessful in five of ten AU patients. Relapse was observed in one of the AA and three of the AU responders. Acneiform lesions and elevation of transaminases were the major side effects.

Conclusion: Tofacitinib seems to be more promising and thriving in the treatment of AA than AU. Starting the therapy earlier can bring more successful results. Unfortunately, even in the cases that fully respond to treatment, relapse can be observed after discontinuation of the treatment. It is essential to inform patients about this situation in reducing the frustrations that may occur later.

KEYWORDS

alopecia areata, alopecia universalis, JAK-STAT inhibitors, tofacitinib

1 | INTRODUCTION

Alopecia areata (AA) is a common cause of noncicatricial alopecia with unpredictable prognosis, leading to crucial cosmetic problems for the patients.[1] Alopecia universalis (AU), the most dreaded subtype of AA, is often challenging to treat.[2] The importance of cytotoxic T lymphocytes in AA pathogenesis has been emphasized in recent publications.[3] Tofacitinib is a Janus kinase/Signal transducers and activators of transcription (JAK/STAT) inhibitor proven effective for treating rheumatoid arthritis and ulcerative colitis and has been successfully tested in many dermatological diseases, including AA.[4] It inhibits both JAK 1 and (mainly) JAK 3.[5,6] Although there is an

increasing number of publications[7,8] in the literature finding tofacitinib successful for both hair and beard, the results of the efficacy for AA treatment are variable.[9] Our study aims at investigating the efficacy and safety of long-term tofacitinib therapy in AA and AU.

2 | MATERIALS AND METHODS

2.1 | Study design and selection of the patients

Our study is a retrospective pilot study covering the years 2017-2020. In our study, 13 patients with clinical diagnoses of AA and AU, aged between 17 and 49 years, were included. In eight of them (61.5%), clinical diagnosis was also supported with biopsy reports. All patients had previously used at least one topical immunotherapy or systemic therapy without any satisfactory clinical response before starting systemic tofacitinib (Table 1). Except for one patient who could not continue the treatment after 3 months, as the insurance system did not allow, all of the patients received 10 mg/d of tofacitinib as monotherapy for 12 months on average (3-15 months).

The patient selection and the dosage and duration of the off-label drugs are strictly controlled by the ministry of health in our country. The use of tofacitinib up to 10 mg in adults and 7.5 mg in the pediatric age group is allowed for each patient, only in severe, recalcitrant

TABLE 1 Demographic and other treatment-related features of the patients using oral tofacitinib

Patient	Sex	Age (years)	Type	First SALT score	Last SALT score	Disease duration (years)	First effect (months)	First effect place	Family history	Autoimmunity	Biopsy
1	M	28	AA	75	8	3	1	Vertex	No	No	Yes
2	F	45	AA	65	5	10	1	Vertex	No	Hashimato T.	Yes
3	F	30	AU	100	38	4	1	Temporo-Parietal	No	Hashimato T.	Yes
4	F	29	AU	100	100	26	0	None	No	Hashimato T.	Yes
5	F	17	AU	100	90	14	3	Eye-brow	No	No	Yes
6	F	41	AU	88	20	10	3	Vertex	No	No	No
7	F	32	AU	100	40	18	1	Occipital	Yes	No	No
8	F	49	AU	100	25	12	1,5	Eye-brow	No	Vitiligo	No
9	M	30	AU	100	100	4	0	None	No	No	No
10	M	30	AA	45	0	7	1	Occipital	No	No	Yes
11	M	31	AU	100	100	12	0	None	No	No	Yes
12	F	26	AU	100	100	4	0	None	No	No	Yes
13	M	30	AU	100	100	3	0	None	No	Hashimato T.	No

Note: M, Male; F, Female; AA, Alopecia areata; AU, Alopecia universalis; and T., Thyroiditis.

cases. The laboratory tests and physical examinations were repeated every 3 months to assess the efficacy and permission to continue the treatment.

2.2 | Data collection and measurements

Treatment results were evaluated with photo-documentation and Severity of Alopecia Tool (SALT) scores. SALT scores were calculated as the sum of the percentages of four divided scalp regions (left parietal as 18%, right parietal as 18%, vertex as 40%, and occipital as 24%). A SALT score of "zero" indicates no hair loss, and "100" means that all hair is lost. Of the 13 cases with an initial SALT score of 45 and higher, three (23.1%) had generalized AA, and 10 (76.9%) had AU.

Treatment success was evaluated by the percentage of the difference in the last and the initial SALT scores to the initial SALT score. The improvement in SALT score is categorized by 1%-10% (weak), 10%-75% (moderate), and 76%-100% (good) response.

Before starting the treatment, complete blood count, sedimentation, C-reactive protein, serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT), blood

Side effects	Duration treatment (months)	Follow-up (months)	Relapse	Relapse time	SALT score change	Note	Previous treatments
Yes, transient transaminase elevation	15	18	No	-	% 76-100	-	IL steroids, Diphencyprone
Yes, acneiform lesions, oily skin	15	21	Yes	After 12 mo	% 76-100	-	Cignolin (Dithranol / Anthralin)
Yes, acneiform lesions, oily skin	12	13	Yes	After 2 mo	% 76-100	-	Systemic steroid, Cyclosporin, Cignolin (Dithranol / Anthralin), Topical minoxidil
Yes, acneiform lesions, oily skin	9	13	-	-	%0	-	Phototherapy, Systemic steroid, IL steroid
Yes, acneiform lesions, oily skin	9	13	No	-	%1-10	-	Systemic steroid, Cyclosporin, Zinc, Anthralin& Salisilic acid cream
Yes, acneiform lesions, oily skin	14	13	Yes	During therapy	% 76-100	-	Cyclosporin, Topical and IL steroid, PRP, Dermapen
Yes, acneiform lesions, oily skin	12	13	Yes	After 2 mo	% 76-100	-	Phototherapy, Systemic steroid, Cyclosporin, IL steroid
Yes, acneiform lesions, oily skin	12	13	Yes	After 2 mo	% 76-100	-	Topical steroid, Diphencyprone
Yes, transient transaminase elevation, acneiform lesions, oily skin	15	14	-	-	%0	After adding systemic minoxidil, Last SALT, Score:35	Topical and IL steroid, Cyclosporin
None	8	13	No	-	% 76-100	-	Topical and IL steroid, SADBE (squaric acid dibutylester)
Yes, resistant transaminase elevation	3	13	-	-	%0	Treatment ceased at 3rd mo	Systemic steroid, Cyclosporin, Topical magistrals
None	6	13	-	-	%0	-	Topical and IL steroid, Cyclosporin
None	4	13	-	-	%0	-	Cyclosporin, Topical steroid, Topical magistrals

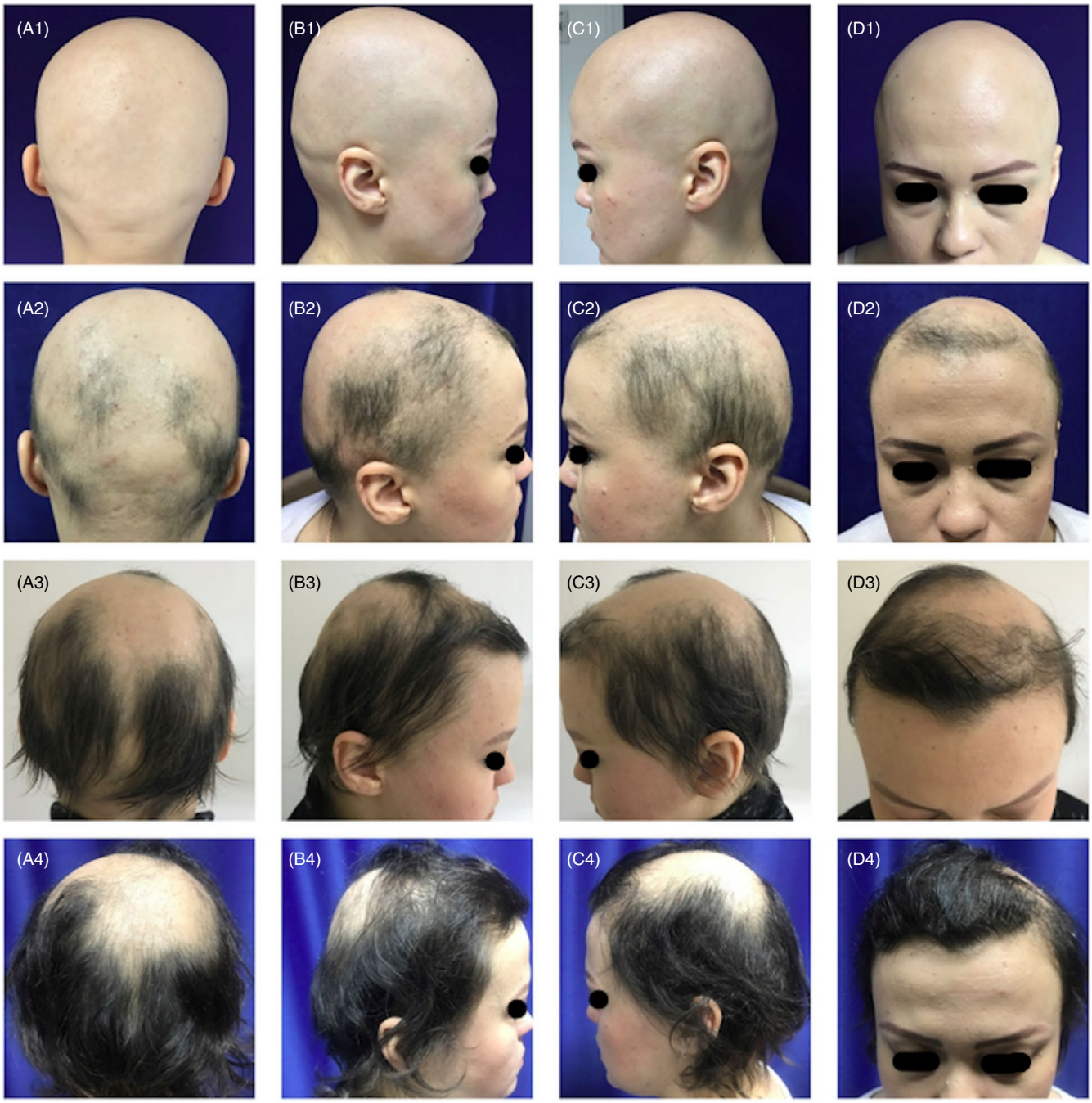

FIGURE 1 Change in the SALT scores of the "patient 3" at 0-3-6 and 12th months of follow-up in the pictures 1-2-3-4, respectively (A. Occipital view, B. Right parietal view, C. Left parietal view, and D. Frontal view). The informed consent form was established

lipids, autoimmune markers, cancer markers, Chest X-rays, purified protein derivative (PPD) tests have been performed in these patients. The results were reviewed by a pulmonologist and a specialist in internal medicine. Just after approvals were obtained, the off-label use reports were issued, and tofacitinib treatments were initiated.

Undesirable results in the laboratory examinations, repeated every 3 months during the treatment process, were accepted as a reason for cessation.

2.3 | Statistical analysis and approval of the ethics committee

Our study was carried out with "the approval of the ethics committee" numbered 18/304. Statistical analysis of the study was carried out using Statistical Package for the Social Sciences version 24.0 (IBM SPSS Statistics for Mac, Version 24.0; IBM Corp.) program.

Descriptive statistics were used to define demographic features. Categorical variables were presented as percentages and frequencies.

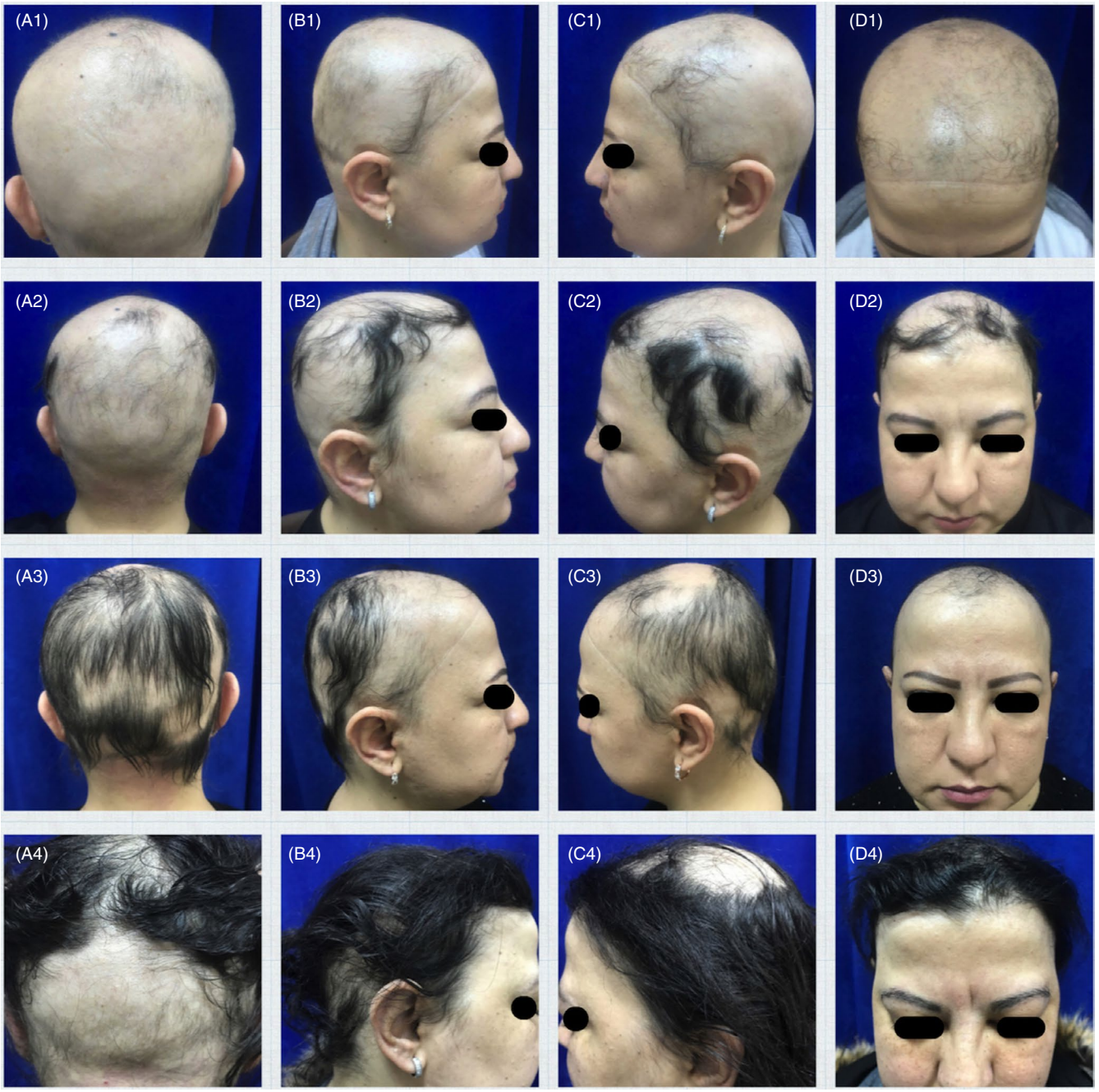

FIGURE 2 Change in the SALT scores of the "patient 7" at 0-4-8 and 12th months of follow-up in the pictures 1-2-3-4, respectively (A. Occipital view, B. Right parietal view, C. Left parietal view, and D. Frontal view). The informed consent form was established

To evaluate numerical variables for the normal distribution, Shapiro-Wilkinson test was used. Data were expressed as median (minimum-maximum) when appropriate. The statistical significance level was determined as $P < .05$.

All the procedures performed were carried out under the ethical principles of the revised Helsinki declaration. Written consent was obtained from the patients to use their photographs.

3 | RESULTS

A total of 13 recalcitrant AA and AU patients, five (38.5%) male, eight (61.5%) female, whose ages range from 17 to 49 years (median age: 30.0), resistant to various topical and systemic treatments were included. The shortest treatment duration was 3 months (only in one patient), and the longest was 15 months (median: 12.0). The changes

in the SALT scores at baseline and control examinations were investigated. The demographic variables and some patient characteristics are summarized in Table 1.

Improvement in SALT scores was established in eight patients (61.5%), while no response was observed in five (38.5%) out of 13 patients. Treatment success was assessed by the change in SALT scores. Accordingly, the change was 1%-10% (weak) in one patient (7.7%); 10%-75% (moderate) in none of the patients; and 76%-100% (good) response in seven patients (53.8%).

All three AA patients improved by 76%-100%. Only one of them showed recurrence after the treatment.

Only five out of 10 AU patients responded to treatment (four were 76%-100%; one was 0%-10%). In the responders in the AU group, the first (median: 100; (88-100)) and the last (median: 38; (20-100)) SALT scores were recorded. Four of the responsive 5 AU patients recurred the disease within at least 2 months.

After the treatment, the patients were followed up for at least 13 months (13-21 months). New hair loss occurred in five of eight patients who responded to therapy. One of those relapses occurred during treatment, two of them occurred after the second month, and one happened 1 year later. Two of the representative patient's clinical follow-up pictures are shown in Figures 1 and 2.

Tofacitinib treatment was started as monotherapy. However, only "patient 9," who was accepted as "unresponsive" in this study, added oral minoxidil independently without any doctor prescription.[10]

The average disease duration was 10 years (3-26 years). The average onset of efficacy was one month (1-3 months). No other side effects were reported other than transaminase elevation and acneiform eruption with oily skin findings (Table 1).

During the treatment, the elevation of transaminases was recorded in two (15.3%) patients as a transient side effect. However, the resistant elevation of transaminases caused discontinuation of the therapy in another (7.7%) patient. Undesirable results in the laboratory examinations repeated every 3 months during the treatment were accepted as a reason for cessation. Other laboratory results of all patients were in normal ranges.

Acneiform lesions were reported as the most common side effects in 9 (69.2%) patients. Only one patient was free of any side effects. None of the patients showed any signs of systemic infection during the treatment.

Hashimoto thyroiditis was detected in four of the patients. The treatment was ineffective in two of them, and in the other two, recurrence was seen after 2 and 12 months. In one patient with vitiligo, no recurrence was observed.

4 | DISCUSSION

Tofacitinib seems promising in the treatment of AA and AU in recent studies.[1,2] We tried to evaluate oral tofacitinib treatment's efficacy and safety in 13 recalcitrant AA and AU patients.

Tofacitinib has been used for more than 9.5 years in more than 7000 patients for rheumatoid arthritis.[11] The first report about the success of oral tofacitinib in the treatment of AA was in 2014.[12] Since then, JAK inhibitors, ruxolitinib, baricitinib, and tofacitinib have been tried for AA/AU with changing success rates. Both tofacitinib and ruxolitinib were found effective similarly in AA/AU.[13] However, which one's superior to the other in AA treatment and the relative efficacy of JAK1/2/3 inhibition is still unknown.[14] We use tofacitinib as monotherapy without switching to any other JAK inhibitors as they have not been found in our country yet.

The lowest side effect and highest clinical response was obtained with tofacitinib in a case-control study, comparing tofacitinib with topical immunotherapy and oral steroid ± immunosuppressant 6-month treatment results in resistant AU and AT patients.[2]

In the reviews about tofacitinib's clinical safety and efficacy in AA, the overall response rate was 72.4%,[15] and the complete and partial response rates were 26.1% and 54.0%, respectively.[1] Acceptable treatment success is generally defined as an improvement over 50% in the SALT scores,[9] reported between 32% and 66% in the literature.[6] In our study, although 61.5% of the patients using tofacitinib had varying degrees of hair growth, only 53.8% were found to be good responders (76%-100% SALT score change), which is compatible with the literature.[15-18]

In the study of Serdaroglu et al, 5% of patients showed resistance to treatment.[19] In another report, four of 9 patients did not respond tofacitinib.[16] In our study, resistance rate was found as 38.5%. The success of the treatment is appeared to be closely related to the maintenance of the therapy.

Although accepted as "unresponsive" on monotherapy, one of our patients showed impressive improvement after addition of oral minoxidil by the sixth unresponsive month of tofacitinib treatment. With the addition of oral minoxidil independently without any doctor's prescription, the SALT score decreased from 100 to 35 at the end of the 11th month. Oral minoxidil, which has been tried for AA,[3] may have an additive effect on tofacitinib and needs supportive randomized controlled trials. However, all of the hair fell out within just a month after tofacitinib and minoxidil treatment was stopped.[10]

In the study of Guo et al, a quarter of patients relapsed mostly soon after cessation of the treatment.[1] Phan et al found the mean recurrence time as 2.7 months, and the treatment success as four times higher in patients in systemic use than topical.[15]

In our study, the average recurrence time was four months. This period ranges from 4-5 weeks to 10-12 weeks in the literature, supporting the need for long-term treatment.[9] The treatment's success will only continue as long as the treatment is continued, and relapse can be expected within an average of four months after the treatment is stopped. However, the most significant drawbacks are the economic costs that this treatment will bring to the patient and the health-care system and the unknown safety profile in the long-term. This drug is an off-label drug used for AA in our country and is allowed to continue after a highly detailed evaluation by the insurance system. Some patients are forced to discontinue treatment when

they cannot obtain insurance permission, which generally causes compulsory relapses.[20]

All of the patients who participated in our study were resistant AA or AU cases who had previously used at least one topical immunotherapy or systemic therapy. The median disease duration in our study was 10 years (3-26 years). While the treatment's success is similar to the literature,[15-19] five of the eight patients (63.5%) who responded to varying degrees had recurrence no later than 12 months (median four months) after the treatment. We also think that the selection of resistant cases was a reason for the high recurrence rate. Although Phan et al opposed this opinion in their meta-analysis,[15] our study results supported the view that prolonged disease duration, high initial SALT scores, and AT/AU subtypes may be responsible for the high recurrence rate.[5,16]

In a review by Sonthalia et al, they suggested a daily treatment dose of 20 mg instead of 10 to prevent a recurrence.[6] Hogan et al also preferred high doses such as 20 mg in their case series and achieved hair growth in 70% of the patients.[9]

Serdaroglu et al reported that long-term continuation at low doses of 10 mg/day was sufficient to prevent recurrence and to provide a low side-effect profile.[19]

An optimal dose and duration of treatment for tofacitinib have not yet been defined. The most important reason for us to choose the dose of 10 mg/d in our study is to minimize possible side effects, which were reported in the literature, such as upper respiratory tract infections: 56.8%, acneiform eruptions: 13.2%, liver enzyme disorder: 7.7%., headache: 7.7%, weight gain: 5.7%, folliculitis: 4.5%, and conjunctivitis: 3.5%.[1] In patients with rheumatoid arthritis, an increase in the frequency of severe side effects such as herpes and similar viral infections, malignancy, tuberculosis activation, and gastrointestinal perforation has been reported, especially with tofacitinib at higher doses.[15,16] Moreover, there is a strict policy in our country to use tofacitinib up to 10 mg in adults and 7.5 mg in pediatric patients.

Concerning the side-effect profile, our study's most common side effects were acneiform eruption and oily skin findings in 69.2% with an unknown mechanism, and elevation of transaminases in 23.0% of the patients.

Theoretically, tofacitinib may be effective in severe acne, where JAK 1 and JAK 3 expression are significantly higher in acne lesion than in normal skin.[21] Although not clear, acneiform lesions during AA treatment may be associated with an increase in seborrhea, an increase in the colonization of Cutibacterium acnes, and a change in the hormonal profile during the treatment.

We follow-up the patients with "3-month" intervals, as suggested by Phan et al, who calculated the average treatment response as 2.2 months and the full response as 6.7 months.[15]Our median time onset of efficacy was one month (1-3 months).

All three AA patients improved by 76%-100%. Although two of these patients did not have any recurrence, in the case AA recurred, Hashimoto thyroiditis was detected. Moreover, four of the five responsive patients in the AU group recurred within at least two months; one was accompanied by Hashimoto thyroiditis.

From the perspective of autoimmunity, Hashimoto thyroiditis was detected in four of 13 patients. The treatment was ineffective in two of them, and in the other two, recurrence was observed after 2 and 12 months. However, in one patient with vitiligo, no recurrence has been observed. Consistent with our findings, in the literature, thyroid diseases were associated with severe AA, but vitiligo was not.[22]

Although the exact duration is not clear, the maximum cumulative tofacitinib treatment duration in AU or AA that we found in the literature was 28 months in a case series.[9] Long-term results are missing; however, the same is true for other novel biological agents being tried for many dermatological conditions such as psoriasis, atopic dermatitis, vitiligo, and many others. The continuation of the treatment life-long with a close follow-up may be considered according to the cost-benefit ratio individually.[23]

5 | RESTRICTIONS

Being a single-center, small-sample study without a control group is the most significant limitation of our research.

6 | CONCLUSIONS

Tofacitinib is found more effective in widespread AA than AU. Unfortunately, the efficacy in AU is low, and relapses are common after the end of treatment. For severe cases, as biological agents that are used continuously in the treatment of psoriasis, continuous use of tofacitinib is required. It is an expensive treatment, and the treatment should be planned considering the long-term side-effect profile, cost-benefit ratio, and individual expectations. Nevertheless, further randomized controlled trials are needed to determine tofacitinib efficacy, safety profile, ideal dose, and treatment duration. While planning such studies, it is necessary to pay attention to the situations that may cause bias in evaluating treatment success by selecting only patients resistant to treatment, including only positive results or, conversely, including mild cases where the disease may regress spontaneously.

ACKNOWLEDGMENTS

The authors declare no funding sources supporting the work.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ETHICAL APPROVAL

Reviewed and approved by "ethics committee" numbered 18/304.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ORCID

Didem Dincer Rota https://orcid.org/0000-0002-3772-6943
Mehmet Ali Can Emeksiz https://orcid.org/0000-0002-0933-4619
Fatma Gulru Erdogan https://orcid.org/0000-0002-7102-7329
Dilsun Yildirim https://orcid.org/0000-0002-9144-6790

REFERENCES

1. Guo L, Feng S, Sun B, Jiang X, Liu Y. Benefit and risk profile of Tofacitinib for the treatment of alopecia areata: a systemic review and meta-analysis. *J Eur Acad Dermatol Venereol*. 2020;34(1):192-201.
2. Shin JW, Huh CH, Kim MW, et al. Comparison of the treatment outcome of oral tofacitinib with other conventional therapies in refractory alopecia totalis and universalis: a retrospective study. *Acta Derm Venereol*. 2019;99(1):41-46.
3. Strazzulla LC, Wang EHC, Avila L, et al. Alopecia areata: An appraisal of new treatment approaches and overview of current therapies. *J Am Acad Dermatol*. 2018;78(1):15-24.
4. de Oliveira AB, Alpalhão M, Filipe P, Maia-Silva J. The role of Janus kinase inhibitors in the treatment of alopecia areata: a systematic review. *Dermatol Ther*. 2019;32(5):e13053.
5. Park H, Yu DA, Kwon O. Janus kinase inhibitors: an innovative treatment for alopecia areata. *J Dermatol*. 2019;46(8):724-730.
6. Sonthalia S, Aggarwal P. Oral tofacitinib: contemporary appraisal of its role in dermatology. *Indian Dermatol Online J*. 2019;10(5):503-518.
7. Kerkemeyer KLS, John JM, Sinclair R, Bhoyrul B. Response of alopecia areata of the beard to oral tofacitinib. *J Am Acad Dermatol*. 2020;82(5):1228-1230.
8. Liu LY, King BA. Tofacitinib for the treatment of severe alopecia areata in adults and adolescents. *J Investig Dermatol Symp Proc*. 2018;19(1):S18-S20.
9. Hogan S, Wang S, Ibrahim O, Piliang M, Bergfeld W. Long-term treatment with Tofacitinib in severe alopecia areata: an update. *J Clin Aesthet Dermatol*. 2019;12(6):12-14.
10. Dincer D, Tanacan E, Kose OC. Efficacy of systemic minoxidil and tofacitinib combination in treatment-resistant alopecia universalis. *J Cosmet Dermatol*. 2020;1-3. https://doi.org.10.1111/jocd.13812
11. Cohen SB, Tanaka Y, Mariette X, et al. Long-term safety of tofacitinib up to 9.5 years: a comprehensive integrated analysis of the rheumatoid arthritis clinical development programme. *RMD Open*. 2020;6(3):e001395.
12. Craiglow BG, King BA. Killing two birds with one stone: oral tofacitinib reverses alopecia universalis in a patient with plaque psoriasis. *J Invest Dermatol*. 2014;134(12):2988-2990.
13. Almutairi N, Nour TM, Hussain NH. Janus kinase inhibitors for the treatment of severe alopecia areata: an open-label comparative study. *Dermatology*. 2019;235(2):130-136.
14. Liu LY, King BA. Ruxolitinib for the treatment of severe alopecia areata. *J Am Acad Dermatol*. 2019;80(2):566-568.
15. Phan K, Sebaratnam DF. JAK inhibitors for alopecia areata: a systematic review and meta-analysis. *J Eur Acad Dermatol Venereol*. 2019;33(5):850-856.
16. Akdogan N, Ersoy-Evans S, Doğan S, Atakan N. Experience with oral tofacitinib in two adolescents and seven adults with alopecia areata. *Dermatol Ther*. 2019;32(6):e13118.
17. Morales-Miranda AY, Bueno-Arias GM, Aguirre-Félix ÓG, Tovar-Franco R. Tofacitinib as a treatment of alopecia areata in adolescents. *Bol Med Hosp Infant Mex*. 2019;76(4):182-187.
18. Hernandez-Montoya C, Ruiz-Villaverde R. The Role of Tofacitinib in the Management of Alopecia Totalis. *Sultan Qaboos Univ Med J*. 2019;19(1):e77-e78.
19. Serdaroğlu S, Engin B, Çelik U, et al. Clinical experiences on alopecia areata treatment with tofacitinib: A study of 63 patients. *Dermatol Ther*. 2019;32(3):e12844.
20. Thompson HJ, Vavra T, Jabbari A. Factors associated with insurance coverage of tofacitinib for alopecia areata: a retrospective review from an academic institution. *J Am Acad Dermatol*. 2020;83(5):1509-1510.
21. Awad SM, Tawfik YM, El-Mokhtar MA, El-Gazzar AF, Abdel Motaleb AA. Activation of Janus kinase signaling pathway in acne lesions [published online ahead of print, 2020 Nov 19]. *Dermatol Ther*. 2020;e14563.
22. You HR, Kim SJ. Factors associated with severity of alopecia areata. *Ann Dermatol*. 2017;29(5):565-570.
23. Solimani F, Meier K, Ghoreschi K. Emerging topical and systemic JAK inhibitors in Dermatology. *Front Immunol*. 2019;10:2847.

How to cite this article: Dincer Rota D, Emeksiz MAC, Erdogan FG, Yildirim D. Experience with oral tofacitinib in severe alopecia areata with different clinical responses. *J Cosmet Dermatol*. 2021;00:1–8. https://doi.org/10.1111/jocd.13966