Efficacy and safety of tofacitinib therapy in Asian patients with severe alopecia areata

Ying-Xiu Dai1,2, Chen-Pu Yeh1,2, Chih-Chiang Chen1,2,3*
1Department of Dermatology, Taipei Veterans General Hospital, Taipei, Taiwan
2Department of Dermatology, School of Medicine, National Yang-Ming University, Taipei, Taiwan
3Institute of Clinical Medicine, National Yang-Ming University, Taipei, Taiwan

Abstract

Background: Patients with severe alopecia areata (AA), including alopecia totalis (AT) and alopecia universalis (AU), are usually resistant to treatment. While tofacitinib is emerging as a promising therapy for severe AA, little is known about its efficacy and safety in Asians.

Objectives: To evaluate the efficacy and safety of tofacitinib for treating severe AA.

Methods: We included patients with ≥50% scalp hair loss, disease for ≥6 months, and tofacitinib therapy for ≥4 months. The efficacy, evaluated based on the percent change in severity of alopecia tool (SALT) score, and response time, defined as time from baseline to reach >50% improvement in SALT score, were assessed.

Results: We included 35 patients (21 with AA, 3 with AT, and 11 with AU). There were 18 men and 17 women (median age: 39 [range: 12–68] years). After 4–24 months of treatment, 74.3% showed clinical response, with 51.4% of the patients achieving ≥50% hair regrowth. Patients with AA showed higher percent change in SALT score than patients with AT/AU (median [interquartile range]: 60.7% [0–98.2] vs. 41.1% [8.9–98.7], P = 0.047). Initial SALT score was negatively associated with the latest percent change in SALT score (P = 0.025). Duration of disease and current episode were positively associated with response time to tofacitinib (P = 0.018 and 0.026, respectively). Patients tolerated tofacitinib well without serious adverse events.

Conclusion: Tofacitinib effectively promoted hair regrowth in Asian patients with severe AA. Randomized controlled trials with larger sample size are needed to confirm the long-term efficacy and safety of tofacitinib for treating severe AA.

Keywords: Alopecia areata, Janus kinase inhibitor, tofacitinib, treatment

INTRODUCTION

Alopecia areata (AA) is a common autoimmune disorder with an estimated lifetime risk of 1.7%.1-3 AA frequently occurs in association with other autoimmune diseases, such as vitiligo, lupus erythematosus, psoriasis, atopic dermatitis (AD), thyroid disease, allergic rhinitis, pernicious anemia, diabetes mellitus, and rheumatoid arthritis.4-10 It might progress to alopecia totalis (AT) and even alopecia universalis (AU), which significantly affect patients’ psychological well-being and quality of life. Although multiple therapeutic modalities have...
been explored, treatment of severe AA, especially AT and AU, remains challenging.[11] Currently, there is no FDA-approved therapy for AA.[12] While the exact pathogenesis of AA is still unknown, current understanding of AA suggests that cytotoxic CD8+ NKG2D+ T-cells play an important role in the pathogenesis of AA. Cytotoxic CD8+ NKG2D+ T-cells upregulate interleukin-15 in the hair follicles followed by production of interferon-γ, which targets the hair follicle for an immune attack. Janus kinase (JAK) inhibitors eliminate the interferon signature and prevent disease development. Reversal of AA by JAK inhibitors has been successfully shown in the murine model.[13] Several studies and case reports have shown the promising results of JAK inhibitors for the treatment of AA, including ruxolitinib, tofacitinib, and baricitinib.[14] Tofacitinib citrate is a potent and selective inhibitor of the enzyme JAK1 and JAK3. It has been approved for the treatment of moderate-to-severe rheumatoid arthritis in the United States as well as other countries and is evaluated for the treatment of other autoimmune inflammatory diseases, such as psoriasis, vitiligo, inflammatory bowel disease, and spondyloarthritis.[15] Recent reports show that tofacitinib has emerged as a promising therapy for severe AA, AT, and AU.[16-19] Although tofacitinib is the most well-studied JAK inhibitor for the treatment of AA in the western population, there have been few studies regarding its use in Asians with severe AA. This study aimed to examine the efficacy and safety of tofacitinib and adjuvant therapy for the treatment of severe AA, AT, and AU in Taiwanese patients.

**Materials and Methods**

**Data collection**

We reviewed the records of patients with AA, AT, or AU who were treated with tofacitinib in Taipei Veterans General Hospital between January 2016 and January 2018. Patients were evaluated by the dermatologists at Taipei Veterans General Hospital. This study includes patients having AA with ≥50% scalp hair loss, AT and AU, stable or worsening disease for ≥6 months, and treatment with tofacitinib for ≥4 months. All patients were treated with 5 mg of tofacitinib twice daily. We also identified the patients who received adjuvant therapy, including methylprednisolone pulse therapy (MPT) or fractional CO2 laser (wavelength = 10,600 nm). The MPT was started for patients with AA, AT, or AU based on the presence or absence of hair regrowth after 3 months. MPT was performed with 500 mg of methylprednisolone once monthly for three sessions. Adjuvant laser therapy was initiated with tofacitinib therapy in the beginning and indicated only for patients with AT or AU in our study. Patients were treated with a fractional CO2 laser (LineXel, Gwangmyeong, Korea) every 2 weeks for a total of six sessions. Laser beam with pulse energy of 7.2 mJ/spot and density of 200–400 spot/cm2 was delivered to square areas (irradiated area = 2 cm2) at four different sections of the scalp.

Demographic and clinical data, including sex, age, duration of disease, duration of current episode, autoimmune comorbidities, family history of AA, and family history of autoimmune diseases, were collected. Photographs were taken before initiation of treatment and at subsequent clinic visits. Investigators used these photographic data to assess disease severity and response to therapy. Disease severity was assessed by the severity of alopecia tool (SALT) score. A SALT score of 0 corresponds to no hair loss; a SALT score of 100 corresponds to complete absence of scalp hair. The institutional review board of Taipei Veterans General Hospital approved this retrospective study and waived the patient informed consent requirement (VGHIRB No.: 2018-01-018AC).

**Outcomes**

The primary outcome of this study was the latest percent change in SALT score, calculated by dividing the absolute change in SALT score from the time of treatment initiation to the last evaluation by the initial SALT score. Percent change in SALT score of 100% indicates complete hair regrowth, whereas 0% indicates no regrowth. There was agreement regarding SALT scores among investigators. Treatment response was categorized according to the latest percent change in SALT score: <5% (nonresponse), 5%–50%, >50%–75%, and >75%. The secondary outcome was the response time, defined as the time taken for baseline SALT score to attain >50% improvement. Adverse events were assessed by physical examinations, review of systems, and laboratory tests after 4 weeks of treatment initiation and then at least every 3 months.

**Statistical analysis**

In this study, statistical analysis was performed using the software SPSS version 22.0 (IBM, Armonk, NY, USA). Descriptive statistics were applied to summarize the data. The Wilcoxon rank-sum test was used to compare different groups. The Spearman rank correlation coefficient was calculated for age, age at onset of the first episode, duration of disease, duration of current episode, initial SALT score, duration of tofacitinib treatment, latest percent change in SALT score, and response time. A two-tailed P < 0.05 was considered statistically significant.

**Results**

**Characteristics of patients**

Overall, 35 patients were identified in this study [Table 1]. There were 18 men (51.4%) and 17 women (48.6%), with a median age of 39 years (range: 12–68 years). Different AA variants were represented, including 21 patients with AA (60%), three patients with AT (8.6%), and 11 patients with AU (31.4%). The median duration of disease before initiating therapy was 2 years (range: 1–34 years). Overall, seven patients had autoimmune comorbidities, including AD, allergic rhinitis, and asthma. Besides, three patients reported family history of AA, and seven patients reported family history of other autoimmune diseases. Before tofacitinib therapy, all patients had failed previous treatment, including topical corticosteroids, intralesional triamcinolone, and topical immunotherapy.
Table 1: Baseline characteristics of the 35 patients

| Characteristics                              | 12-18 | 19-50 | >50 | 1-5 | 6-10 | 11-20 | >20 | 30.5 (4-63) |
|----------------------------------------------|-------|-------|-----|-----|------|-------|-----|-------------|
| Age (years), n (%)                           | 7 (20) | 24 (68.6) | 4 (11.4) | 26 (74.3) | 2 (5.7) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| Sex, n (%)                                   | Female | 17 (48.6) | 18 (51.4) | 30.5 (4-63) | 2 (5.7) | 6 (17.1) | 7 (20) | 9 (25.7) |
| Male                                         | 1 (2.9) | 5 (14.2) | 5 (14.2) | 6 (17.1) | 2 (5.7) | 6 (17.1) | 4 (11.4) | 11 (31.4) |
| Age of onset (years), median (range)         | 30.5 (4-63) | 6 (17.1) | 7 (20) | 9 (25.7) | 2 (5.7) | 6 (17.1) | 4 (11.4) | 11 (31.4) |
| Duration of disease (years), n (%)           |       |       |       |       |       |       |       |             |
| 1-5                                          | 26 (74.3) | 2 (5.7) | 6 (17.1) | 6 (17.1) | 1 (2.9) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| 6-10                                         | 2 (5.7) | 6 (17.1) | 6 (17.1) | 6 (17.1) | 1 (2.9) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| 11-20                                        | 5 (14.2) | 2 (5.7) | 6 (17.1) | 6 (17.1) | 1 (2.9) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| >20                                          | 2 (5.7) | 6 (17.1) | 6 (17.1) | 6 (17.1) | 1 (2.9) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| >5 years                                     | 6 (17.1) | 2 (5.7) | 6 (17.1) | 6 (17.1) | 1 (2.9) | 5 (14.2) | 2 (5.7) | 27 (77.1) |
| Duration of current episodes, n (%)          |       |       |       |       |       |       |       |             |
| ≤3 months                                    | 9 (25.7) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| 4-12 months                                  | 13 (37.1) | 11 (31.4) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| 13-24 months                                 | 1 (2.9) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| >2-5 years                                   | 6 (17.1) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| >5 years                                     | 6 (17.1) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| AA variant, n (%)                            | AA    | 21 (60) | 3 (8.6) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| AT                                           | 3 (8.6) | 11 (31.4) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| AU                                           | 11 (31.4) | 7 (20) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| Autoimmune comorbidities, n (%)              | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| Family history of AA, n (%)                  | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |
| Family history of autoimmune diseases, n (%) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 3 (8.6) | 7 (20) | 7 (20) | 21 (60) |

AA: Alopecia areata, AT: Alopecia totalis, AU: Alopecia universalis

Efficacy and safety

As shown in Table 2, 23 patients received tofacitinib monotherapy, whereas 12 patients were treated with tofacitinib plus adjuvant therapy. The median duration of tofacitinib therapy was 7 months (mean: 8.8, range: 4–24 months). Overall, the median percent change in SALT score was 52.9% (mean: 54.1%, range: 0%–100%). A total of 26 patients (74.3%) experienced ≥5% change in SALT score including 51.4% of patients achieving ≥50% hair regrowth over 4–24 months of treatment. Eight patients achieved 100% change in the SALT score over a median duration of 10 months of treatment; Figure 1 shows the photographic data of a patient who achieved complete hair regrowth.

Among the patients achieving >50% hair regrowth, the median response time was 4 months (range: 2–12 months). There was a negative correlation between initial SALT score and hair regrowth (P = 0.025) [Table 3]. Disease duration and current episode duration were positively associated with response time (P = 0.018 and 0.026, respectively). Patients with AA showed higher percent change in SALT score than patients with AT/AU (median [interquartile range]: 60.7% [0%–98.2%] vs. 41.1% [8.9%–98.7%], P = 0.047). No significant difference was found between men and women in hair regrowth (median [interquartile range]: 45.9% [0%–97.3%] vs. 60.7% [0%–99.1%], P = 0.918).

In AT/AU patients, tofacitinib monotherapy and adjuvant laser therapy showed no significant differences in hair regrowth (median [interquartile range]: 44% [11.2%–98.2%] vs. 38.2% [2%–100%], P = 0.499). However, shorter response time was observed in laser therapy group (median [interquartile range]: 3.0 [2.3–3.0] vs. 6.0 [4.0–6.0] months, P = 0.109). Patients treated with tofacitinib monotherapy achieved greater hair regrowth compared to patients treated with tofacitinib plus MPT, although the difference was not statistically significant (median [interquartile range]: 60.7% [11.2%–100%] vs. 21.2% [0%–93.2%], P = 0.075).

Until the end of the study, 27 patients (77.1%) continued tofacitinib therapy. Among the eight patients who discontinued tofacitinib treatment, four patients achieved adequate hair regrowth (>75% hair regrowth) and the other four patients discontinued treatment due to financial burden. In addition, eight patients were on tapered doses of tofacitinib after achieving clinical response. Disease relapse (patchy or total scalp hair loss) occurred in nine patients (25.7%) during treatment. Among them, eight patients were on tapered doses of tofacitinib and one patient, who was on the same dose, had...
been experiencing hair regrowth before relapse. The patient with spontaneous relapse exhibited hair regrowth after the dose was adjusted to 5 mg of tofacitinib three times daily. Among the eight patients experiencing hair shedding subsequent to tapering or discontinuing tofacitinib, hair regrowth was observed in four patients who returned to standard dose of tofacitinib, while the treatment of the other four patients remained unchanged after detailed discussion. Further analysis showed no significant difference in relapse risk between monotherapy group and adjuvant therapy group.

Adverse events
Over a median duration of 8.8 months of tofacitinib treatment, there was no serious adverse event observed in patients. Adverse events were mild, including diarrhea (four patients), upper respiratory infections (three patients), fatigue (three patients), stomachache (two patients), nausea/vomiting (one patient), headache (one patient), and mildly elevated liver transaminase levels (one patient with 52 U/L of alanine aminotransferase [normal range: 0–40 U/L]), which returned to normal when rechecked even though the therapy continued. In patients receiving adjuvant MPT or laser therapy, no serious or persistent side effects were observed. Side effects of laser therapy included pain during the laser treatment, and transient posttreatment crusting, scaling, erythema, and edema. Other possible side effects, such as secondary bacterial or viral infection, posttherapy blister formation, hypopigmentation, and scarring, were not observed.

Discussion
Our study demonstrated that tofacitinib is effective to promote hair regrowth in patients with severe AA, AT, and AU. Among 35 patients, 74.3% achieved a clinical response (≥5% change in SALT score) with 51.4% of patients achieving >50% hair regrowth over 4–24 months of treatment. Overall, the median percent change in SALT score was 52.9%. The efficacy of tofacitinib therapy observed in our study was comparable to that in previous reports, although our cohort possessed lower percent change in SALT score (mean: 47.7%) in patients without AD. In patients without AD, the patients with AD seemed to achieve more hair regrowth than those without AD. A previous study showed that six out of seven patients exhibited variable hair shedding after discontinuation of tofacitinib, with two and four patients showing initial signs of shedding within 3–4 and 8 weeks after the end of treatment, respectively. This study also found that after discontinuing tofacitinib for 24 weeks, only three patients maintained lower SALT scores compared to the baseline, while the other four patients became even worsening. In our study, eight out of 16 patients experienced disease relapse within 1–3 months after dose tapering or discontinuing treatment. These observations suggest that maintenance of the therapy is necessary for sustaining remission status.

Recent studies have shown favorable response to tofacitinib among pediatric patients. In our study, there were seven adolescent patients aged 12–18 years receiving tofacitinib therapy. These seven patients achieved a median change in SALT score of 98.2% (range: 82%–100%) over a median duration of 10 months (range: 8–11 months) of tofacitinib treatment. In agreement with previous reports, our results suggested that tofacitinib may be a therapeutic option for pediatric patients with severe AA who have failed conventional therapy. However, because of the potential serious adverse effects of JAK inhibitors, including infection and malignancy, a thorough discussion of risks and benefits is necessary before the initiation of treatment.

While tofacitinib has shown promising response in patients with AA, its effect is not durable. In an open-label study of 66 patients, all 20 patients available for follow-up experienced hair loss after therapy was discontinued (median: 8.5 weeks after ceasing treatment). Another open-label study showed that six out of seven patients exhibited variable hair shedding after discontinuation of tofacitinib, with two and four patients showing initial signs of shedding within 3–4 and 8 weeks after the end of treatment, respectively. This study also found that after discontinuing tofacitinib for 24 weeks, only three patients maintained lower SALT scores compared to the baseline, while the other four patients became even worsening. In our study, eight out of 16 patients experienced disease relapse within 1–3 months after dose tapering or discontinuing treatment. These observations suggest that maintenance of the therapy is necessary for sustaining remission status.

Among the 35 patients, two patients, an 18-year-old man and a 30-year-old man, had AD. After 7 and 4 months of tofacitinib treatment, those two patients achieved 96.4% and 100% hair regrowth, respectively. As indicated by the median percent change in SALT score of 41.5% (mean: 47.7%) in patients without AD, the patients with AD seemed to achieve more hair regrowth than those without AD. A previous study showed that patients with either AA have significantly increased risk for AD compared with control individuals (odds ratio 2.57, 95% confidence interval 2.25–2.94). The study showed that six out of seven patients exhibited variable hair shedding after discontinuation of tofacitinib, with two and four patients showing initial signs of shedding within 3–4 and 8 weeks after the end of treatment, respectively. This study also found that after discontinuing tofacitinib for 24 weeks, only three patients maintained lower SALT scores compared to the baseline, while the other four patients became even worsening. In our study, eight out of 16 patients experienced disease relapse within 1–3 months after dose tapering or discontinuing treatment. These observations suggest that maintenance of the therapy is necessary for sustaining remission status.
the small number of AD patients in our study, further studies with larger sample size are required to fully answer this question. In our study, six patients treated with tofacitinib plus MPT achieved a median of 21.2% of hair regrowth. Liu et al. had shown that adjuvant pulsed prednisone therapy (300 mg once monthly for three doses) was associated with sustained hair regrowth in patients who failed to achieve significant hair regrowth with tofacitinib monotherapy. However, the retrospective study design prevented differentiation between the delayed effects of tofacitinib and the efficacy of adjuvant MPT, therefore making it difficult to evaluate additional benefit of the combination therapy.

Over the past several years, there have been some preliminary studies supporting the efficacy of fractional CO2 laser in the treatment of AA. By creating thermal injury zones, fractional CO2 laser was shown to trigger hair regrowth via activation of the Wnt/b-catenin pathway in murine models. Cho et al. had demonstrated that fractional CO2 laser can promote hair growth and is effective in ophiasis, autosomal recessive woolly hair/hypotrichosis, secondary cicatricial alopecia, pubic hypotrichosis, frontal fibrosing alopecia, as well as perifolliculitis capitis abscedens et suffodiens. Another clinical trial also showed that combination treatments with fractional CO2 laser, topical application of triamcinolone, and acoustic pressure wave ultrasound completely restored hair growth in patients with AA. However, the patients in this trial had much less disease severity than our patients. Our study is the first to examine the efficacy and safety of tofacitinib plus fractional CO2 laser for the treatment of severe AA. Compared to patients receiving tofacitinib monotherapy, patients receiving adjuvant laser therapy seemed to have shorter response time for hair regrowth. Further research is required to confirm these results and unveil the molecular mechanism of the combination therapy.

There are some limitations in this study. First, the retrospective study design and lack of randomization are subject to confounding and selection bias, thereby affecting the estimation of treatment effects. Second, the unblinded outcome assessment may lead to observer bias. Third, the small sample size in this study precludes subgroup analyses. Finally, the retrospective study design prevented differentiation between the delayed effects of tofacitinib and the efficacy of adjuvant therapy.

**Conclusion**

In summary, tofacitinib therapy was effective and well tolerated in Taiwanese patients with severe AA. Further studies with larger sample size are required to address some important questions, including appropriate length of treatment, duration of response, factors affecting the response, relapse rates, and long-term side effects of JAK inhibitors for the treatment of AA.

**Financial support and sponsorship**

This study was supported by the Ministry of Science and Technology, R.O.C., under grant (MOST 107-2314-B-075-032-MY3-1), and Taipei Veterans General Hospital under grant (V106D25-002-MY3-1, V106D25-002-MY3-2, VN107-10, V107C-124).

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Dai YX, Chen TJ, Chang YT. Ambulatory practice of dermatologists in Taiwan: A nationwide survey. J Chin Med Assoc 2018;81:729-34.
2. Dai YX, Chen TJ, Chang YT. Skin care services and disease prevalence in Taiwan: A nationwide study. Dermatol Sin 2018;36:124-30.
3. Safavi KH, Muller SA, Suman VJ, Mosshell AN, Melton LJ 3rd. Incidence of alopecia areata in Olmsted County, Minnesota, 1975 through 1989. Mayo Clin Proc 1995;70:628-33.
4. Biran R, Zlotogorski A, Ramot Y. The genetics of alopecia areata: New approaches, new findings, new treatments. J Dermatol Sci 2015;78:11-20.
5. Chi SY, Chen YJ, Tseng WC, Lin MW, Chen TJ, Hwang CY, et al. Comorbidity profiles among patients with alopecia areata: The importance of onset age: a nationwide population-based study. J Am Acad Dermatol 2011;65:949-56.
6. Huang KP, Mullangi S, Guo Y, Qureshi AA. Autoimmune, atopic, and mental health comorbid conditions associated with alopecia areata in the United States. JAMA Dermatol 2013;149:789-94.
7. McDonagh AJ, Tazi-Ahnini R. Epidemiology and genetics of alopecia areata. Clin Exp Dermatol 2002;27:405-9.
8. Dai YX, Wang SC, Chou YJ, Chang YT, Chen TJ, Li CP, et al. Smoking, but not alcohol, is associated with risk of psoriasis in a Taiwanese population-based cohort study. J Am Acad Dermatol 2019;80:727-34.
9. Chen CC, Chang YT, Liu HY, Chen YJ. Cancer risk in patients with alopecia areata: A nationwide population-based matched cohort study. Cancer Med 2018;7:2153-9.
10. Li CY, Dai YX, Chen YJ, Chu SY, Chen YJ. Cancer risks in vitiligo patients: A nationwide population-based study in Taiwan. Int J Environ Res Public Health 2018;15:1847.
11. Kassira S, Korta DZ, Chapman LW, Damm F. Review of treatment for alopecia totalis and alopecia universalis. Int J Dermatol 2017;56:801-10.
12. Dainichi T, Kabashima K. Alopecia areata: What’s new in epidemiology, pathogenesis, diagnosis, and therapeutic options? J Dermatol Sci 2017;86;3-12.
13. Xing L, Dai Z, Jabbbar A, Cerise JE, Higgins CA, Gong W, et al. Alopecia areata is driven by cytotoxic T lymphocytes and is reversed by JAK inhibition. Nat Med 2014;20:1043-9.
14. Samadi A, Ahmad Nasrollahi S, Hashemi A, Nassiri Kashani M, Firooz A. Janus kinase (JAK) inhibitors for the treatment of skin and hair disorders: A review of literature. J Dermatol Treat 2017;28:476-83.
15. Shreiber-Kassidim R, Ramot Y, Zlotogorski A. Janus kinase inhibitors in dermatology: A systematic review. J Am Acad Dermatol 2017;76:745-53.
16. Ibrahim O, Bayart CB, Hogan S, Piliang M, Bergfeld WF. Treatment of alopecia areata with tofacitinib. JAMA Dermatol 2017;153:600-2.
17. Jabbbar A, Sansaricq F, Cerise J, Chen JC, Bitterman A, Ucerio G, et al. An open-label pilot study to evaluate the efficacy of tofacitinib in moderate to severe patch-type alopecia areata, totalis, and universalis. J Invest Dermatol 2018;138:1539-45.
18. Kennedy Crispin M, Ko JM, Craiglow BG, Li S, Shankar G, Urban JR, et al. Safety and efficacy of the JAK inhibitor tofacitinib citrate in patients with alopecia areata. JCI Insight 2016;1:e89776.
19. Liu LY, Craiglow BG, Dai F, King BA. Tofacitinib for the treatment of severe alopecia areata and variants: A study of 90 patients. J Am Acad Dermatol 2017;76:22-8.
20. Craiglow BG, King BA. Tofacitinib for the treatment of alopecia areata and variants in adolescents. J Am Acad Dermatol 2019;80:568-70.
21. Craiglow BG, Liu LY, King BA. Tofacitinib for the treatment of alopecia areata and variants in adolescents. J Am Acad Dermatol 2017;76:29-32.
22. Dai YX, Chen CC. Tofacitinib therapy for children with severe alopecia...
areata. J Am Acad Dermatol 2019;80:1164-6.
23. Mohan GC, Silverberg II. Association of vitiligo and alopecia areata
with atopic dermatitis: A systematic review and meta-analysis. JAMA
Dermatol 2015;151:522-8.
24. De Waard-van der Spek FB, Oranje AP, De Raeymaecker DM,
Peereboom-Wynia JD. Juvenile versus maturity-onset alopecia
areata – A comparative retrospective clinical study. Clin Exp Dermatol
1989;14:429-33.
25. Lee CH. Immune regulation in pathophysiology and targeted therapy for
itch in atopic dermatitis. Dermatol Sin 2016;34:1-5.
26. Cho YT, Chu CY. Advances in systemic treatment for adults with
moderate-to-severe atopic dermatitis. Dermatol Sin 2019;37:3-11.
27. Malik K, Gutman-Yassky E. Cytokine targeted therapeutics for
alopecia areata: Lessons from Atopic dermatitis and other inflammatory
skin diseases. J Invest Dermatol Symp Proc 2018;19:S62-S64.
28. Mlacker S, Aldahan AS, Simmons BI, Shah V, McNamara CA,
Samarkandy S, et al. A review on laser and light-based therapies for
alopecia areata. J Cosmet Laser Ther 2017;19:93-9.
29. Welsh O. Phototherapy for alopecia areata. Clin Dermatol 2016;34:628-32.
30. Bae JM, Jung HM, Goo B, Park YM. Hair regrowth through wound
healing process after ablative fractional laser treatment in a murine
model. Lasers Surg Med 2015;47:433-40.
31. Cho S, Choi MJ, Zheng Z, Goo B, Kim DY, Cho SB. Clinical effects
of non-ablative and ablative fractional lasers on various hair disorders:
A case series of 17 patients. J Cosmet Laser Ther 2013;15:74-9.
32. Issa MC, Pires M, Silveira P, Xavier de Brito E, Sasajima C.
Transdermal drug delivery: A new treatment option for areata
alopecia? J Cosmet Laser Ther 2015;17:37-40.