A study on efficacy of 308 nm excimer light therapy in alopecia areata

Badania nad skutecznością promieniowania ekscymerowego o długości fali 308 nm w leczeniu łysienia plackowatego

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ABSTRACT

Introduction. Alopecia areata (AA) is an autoimmune condition characterized by T-cell-mediated attack on hair follicles.

Objective. Evaluation of efficacy of 308 nm excimer light in the treatment of alopecia areata.

Material and methods. Patches of AA were treated twice weekly, but not on 2 consecutive days, with UVB excimer 308 nm light. In all patients, half of the AA patch was irradiated and the other half was spared, covered with a black rubber shield and taken as a control. Therapy was started with an initial dose of 300 mJ/cm^2, with an incremental dose of 100 mJ/cm^2 at every sitting until fine erythema appeared.

Results. At the end of the treatment, one patient (3.33%) at the test site showed > 50% hair regrowth vs. none at the control site. After a 2-month post-treatment period, successful hair regrowth (grades 3 and 4) was seen in 12 patients at test sites and in 2 patients at control sites. The differences were statistically significant ($p < 0.05$). After 4 months successful hair regrowth (grades 3 and 4) was seen in 16 patients at test sites and in 5 patients at control sites. The differences were statistically significant ($p < 0.05$). There was no statistically significant difference in the response to treatment in relation to the duration of the disease ($p > 0.05$).

Conclusions. Excimer light has the potential to stimulate hair regrowth in patches of alopecia areata.

STRESZCZENIE

Wprowadzenie. Łysienie plackowate (*alopecia areata* – AA) jest chorobą autoimmunologiczną charakteryzującą się atakiem limfocytów T na mieszek włosowy.

Cel pracy. Ocena skuteczności światła ekscymerowego w terapii pojedynczych ognisk AA.

Materiał i metodyka. Ogniska AA naświetlano 2 razy w tygodniu (ale nie w kolejnych dniach) promieniowaniem ekscymerowym (UVB) o długości fali 308 nm. U wszystkich pacjentów napromieniowywano jedną połowę ogniska, natomiast druga, osłonięta czarną gumą, służyła jako kontrola. Naświetlania rozpoczynano od dawki 300 mJ/cm^2, którą zwiększano o 100 mJ/cm^2 podczas kolejnych naświetlań aż do uzyskania wyraźnego rumienia.

Wyniki. Bezpośrednio po zakończeniu terapii tylko u 1 pacjenta (3,33%) obserwowano w polu naświetlanym > 50% odrostu włosów, natomiast nie stwierdzono żadnego odrostu w polu kontrolnym. Po 2 miesiącach od zakończenia terapii uzyskano zadowalający (50–75%) odrost włosów u 12 pacjentów w ogniskach naświetlanych oraz u 2 w okolicy kontrolnej. Różnica była istotna statystycznie ($p < 0,05$). Po 4 miesiącach od zakończenia naświetlań dobry wynik terapeutyczny obserwowano u 16 chorych w okolicy naświetlanej i u 5 w okolicy kontrolnej. Ta różnica była także istotna statystycznie. Nie stwierdzono istotnych statystycznie różnic w odpowiedzi na leczenie w zależności od czasu trwania ognisk AA ($p > 0,05$).

Wnioski. Światło ekscymerowe ma właściwości stymulujące odrost włosów w ogniskach AA.

INTRODUCTION

Alopecia areata (AA) is an autoimmune condition characterized by T-cell mediated attack on hair follicles. It is the most common hair loss after androgenic alopecia. It affects all ages and both sexes equally [1–4]. It can present with demarcated patches of hair loss, multiple patches, or extensive hair loss (alopecia totalis/universalis) [2–7]. The disease causes significant cosmetic and psychological distress in affected persons [5–7].

Because of the unpredictable course of the disease, management of patients with AA is a challenging task. No definite cure is established. Glucocorticoids have been the mainstay of treatment and have been used via topical, systemic or intralesional routes [4–6]. Other treatment options include contact sensitizers such as dinitrochlorobenzene (DNCP), diphencyprone (DPCB), squaric acid dibutylester (SADBE), systemic immunosuppressants such as methotrexate, azathioprine, and disease-modifying anti-rheumatic drugs (DMARDs) such as sulfasalazine [4–6].

There have been some studies reporting success of phototherapy such as PUVA and narrowband UVB (NBUVB) in patients with AA. However, one of the major disadvantages of the conventional phototherapeutic modalities is that along with the alopecia patches, large areas of normal skin are also exposed to irradiation [7, 8]. To overcome this problem, targeted phototherapy such as excimer laser/light therapy has been used for treating AA [7–9]. The device is thought to be capable of inducing apoptosis of affected T-cells [7]. It has the advantages of a lower cumulative UV dose involved, shorter time frame required for treatment, and the option of targeting individual lesions without affecting surrounding healthy skin [7–9]. However, there are only a few studies that have evaluated the efficacy and safety of UVB excimer laser/light in AA.

OBJECTIVE

The aim of our prospective non-randomized study was to determine the efficacy of 308 nm excimer light for the treatment of AA.

MATERIAL AND METHODS

The study was carried out in the Dermatology Outpatient Department of Rabindranath Tagore Medical College, Udaipur, Rajasthan Medical College and Associated Hospitals between June 2013 and October 2014, after approval of the study protocol by the Ethics Committee of the institution. A total of 40 patients with a single active AA patch (activity defined as positive pull test at the margin of the lesion) [10] were enrolled in the study. The diagnosis of AA was made clinically. Inclusion criteria included patients who were willing and capable of cooperating to the extent and degree required by the protocol, patients presenting with a single patch of AA over the scalp of at least 3 months duration, having a diameter between 2 cm and 10 cm, and patients who had not received any treatment for 8 weeks prior to enrolment in the study. The following patients were excluded from the study: patients who did not give consent, patients with multiple alopecia patches, alopecia totalis or alopecia universalis, patients with a history of photosensitive disorder, pregnant or breast-feeding women, and patients with a history of previous skin cancer.

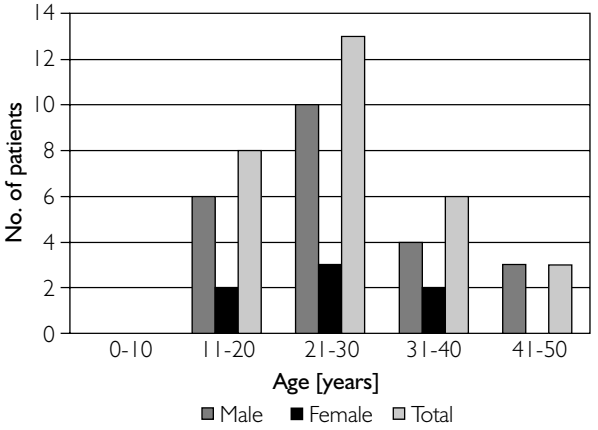
All patients were subjected to detailed history and examination recorded in the appropriate document. The following laboratory investigations were performed: complete blood count with peripheral blood smear, liver function test, renal function test, fasting blood sugar and thyroid stimulating hormone (TSH).

The excimer UVB source was a 308 nm excimer lamp. It emits a monochromatic non-coherent light

Table 1. Distribution of patients according to age and sex

Tabela 1. Wiek i płeć pacjentów

Age [years]	Male, *n* (%)	Female, *n* (%)	No. of patients (*N* = 30), *n* (%)
0–10	0	0	0
11–20	6 (75)	2 (25)	8 (26.67)
21–30	10 (76.92)	3 (23.08)	13 (43.33)
31–40	4 (66.67)	2 (33.33)	6 (20)
41–50	3 (100)	0	3 (10)



The age group 21–30 contained the highest number of patients (13 (43.33%)). Male to female ratio was 3.29 : 1.

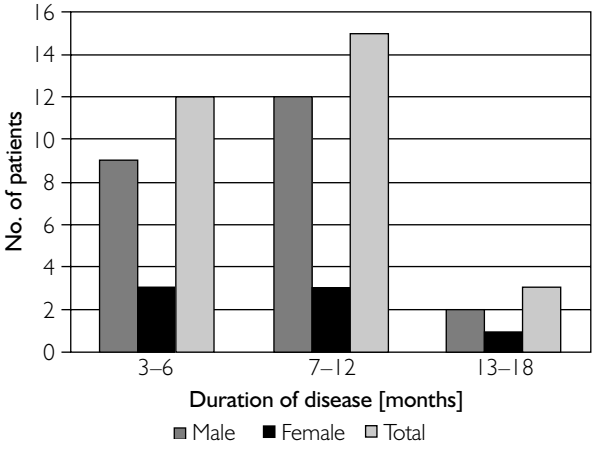
Figure 1. Distribution of patients according to age and sex

Rycina 1. Wiek i płeć pacjentów

Table 2. Distribution of patients according to duration of the disease

Tabela 2. Czas trwania choroby

Duration [months]	Male, *n* (%)	Female, *n* (%)	No. of patients (*N* = 30), *n* (%)
3–6	9 (75)	3 (25)	12 (40)
7–12	12 (80)	3 (20)	15 (50)
13–18	2 (6.67)	1 (3.33)	3 (10)



The mean duration was 8.2 months.

Figure 2. Distribution of patients according to duration of the disease

Rycina 2. Czas trwania choroby

with wavelength 308 nm, spot size 40 × 40 mm (16 cm^2), pulse duration 1–120 s, output power 800 mW ±10% for 2.48 in^2 (16 cm^2). The light source is a Xenon chloride lamp.

Patches of AA were treated twice weekly, but not on any two consecutive days, with UVB excimer 308 nm light. In all patients, half of the AA patch was irradiated and the other half covered with black rubber shield was spared and taken as a control. Therapy was started with an initial dose of 300 mJ/sq · cm, with an incremental dose of 100 mJ/sq · cm at every sitting until fine erythema appeared. At this point, the dose of irradiation was fixed. Spot diameter was adjusted according to the size of the patches. The end point was either a total of 16 treatments given over 8 weeks, or until complete hair regrowth was achieved, whichever was earlier. Patients were followed up until 4 months after treatment. Clinical photographs were taken with Nikon cool pix S6300 camera with standard settings at the beginning and at the end of treatment, and at 2 months and 4 months after the end of therapy (post-treatment). Photographic evaluation formed the basis of therapeutic assessment. Scoring was done according to the regrowth of both vellus and/or terminal hairs as follows: < 25% hair regrowth (poor response – grade 1), 25–49% (fair response – grade 2), 50–74% hair regrowth (good response – grade 3) and 75–100% hair regrowth (excellent response – grade 4). Grades 1 and 2 were considered as "ineffective treatment/failure", and grades 3 and 4 were considered as "successful/effective treatment".

Statistical analysis

Chi-square (χ^2) test was calculated based on the results obtained from the study. For statistical calculations, SPSS version 20.0 was used. The probability (*p* value) was calculated to determine the significance level; $p < 0.05$ was considered as significant.

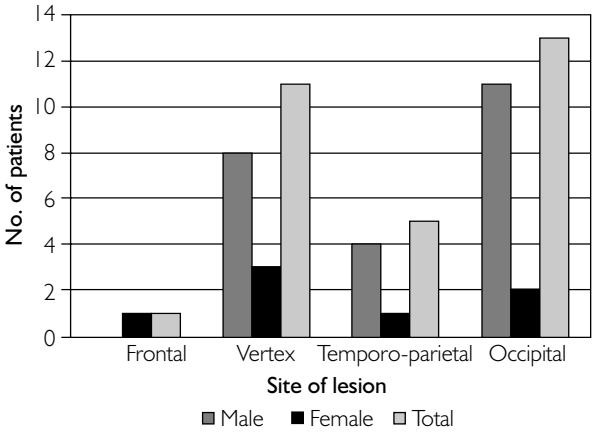
RESULTS

A total of 40 patients were enrolled in the study, out of which only 30 persons completed the study (M = 23, F = 7, M : F = 3.29 : 1). Thirteen patients (43.33%) belonged to the age group of 21–30 years. The mean age was 21.8 years (Table 1, Figure 1). Half of the patients (15 patients, 50%) had alopecia for 7–12 months (Table 2, Figure 2). Occipital area was the commonest localization (13 patients, 43.33%) (Table 3, Figure 3). Intralesional corticosteroid injections were the most common medication received by patients before enrolment in the study (18 patients, 60%) (Table 4, Figure 4). The erythema dose in the majority of patients ranged from 1200 to 1600 mJ/cm^2. Cumu-

lative dose ranged from 14.7 J/cm² to 16.8 J/cm² (Table 5). At the end of the treatment, one patient (3.33%) showed > 50% hair regrowth at the test site vs. none at the control site (Table 6, Figure 5). At 2 months after treatment, successful hair regrowth (grades 3 and 4) was seen in 12 patients at test sites and in 2 patients at control sites. The difference was statistically significant ($p < 0.05$). At 4 months after treat-

Table 3. Distribution of patients according to site of lesion over scalp
Tabela 3. *Lokalizacja zmian na głowie*

Site	Male, *n* (%)	Female, *n* (%)	No. of patients (*N* = 30), *n* (%)
Frontal	0	1	1 (3.33)
Vertex	8 (72.73)	3 (27.27)	11 (36.67)
Temporo-parietal	4 (80)	1 (20)	5 (16.66)
Occipital	11 (84.62)	2 (15.38)	13 (43.34)

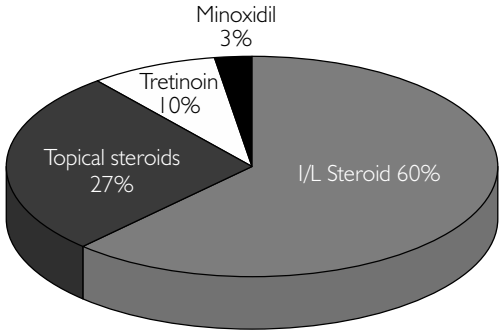


The largest group of patients (13, 43.33%) had lesions on the occipital area.

Figure 3. Distribution of patients according to site of lesion over scalp
Rycina 3. *Lokalizacja zmian na głowie*

Table 4. Previous treatments taken by the patients
Tabela 4. *Dotychczasowe leczenie*

Treatment	No. of patients (*N* = 30), *n* (%)
I/L Steroids	18 (60)
Topical steroid (class-1 steroids)	8 (26.67)
Topical tretinoin 0.05–0.1%	3 (10)
Topical minoxidil 2-5%	1 (3.33)



A majority of patients (18, 60%) had received intralesional steroid injection previously.

Figure 4. Previous treatments taken by the patients
Rycina 4. *Dotychczasowe leczenie*

Table 5. Erythemogenic and cumulative dose of excimer light
Tabela 5. *Rumieniotwórcze i łączne dawki światła ekscymerowego*

Patient no.	Dose at which fine erythema appeared [mJ/cm²]	Cumulative dose [J/cm²]
1	1200	14.7
2	–	16.8
3	1400	15.8
4	1400	15.8
5	–	16.8
6	1600	16.6
7	–	16.8
8	1200	14.7
9	1200	14.7
10	1400	15.8
11	–	16.8
12	1300	15.3
13	1200	14.7
14	–	16.8
15	1600	16.6
16	–	16.8
17	1600	16.6
18	1200	14.7
19	1600	16.6
20	1200	14.7
21	–	16.8
22	1500	16.2
23	1400	15.8
24	1600	16.6
25	1400	15.8
26	1600	16.6
27	1300	15.3
28	–	16.8
29	1300	15.3
30	–	16.8

Most patients showed erythema at doses varying between 1200 and 1600 mJ/cm². Cumulative dose ranged between 14.7 J/cm² and 16.8 J/cm².

Table 6. Efficacy of excimer light at the end of 16 weeks of treatment
Tabela 6. *Wyniki leczenia po 16 tygodniach*

Improvements (grade)	Test site (*N* = 30), *n* (%)	Control site (*N* = 30), *n* (%)
1 (< 25%)	18 (60)	29 (96.67)
2 (26–49%)	11 (36.67)	1 (3.33)
3 (50–75%)	1 (3.33)	0
4 (> 75%)	0	0

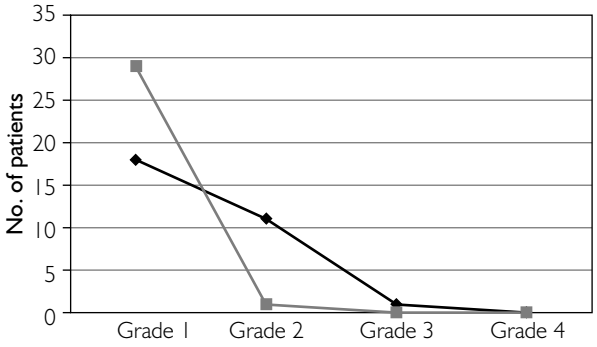


Figure 5. Efficacy of excimer light at the end of treatment

Rycina 5. *Wyniki leczenia po 16 tygodniach*

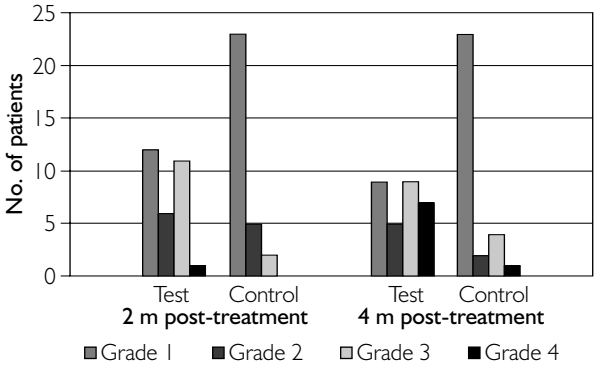


Figure 6. Efficacy of excimer light therapy during follow-up period

Rycina 6. *Wyniki terapii w okresie oberwacji po leczeniu*

Table 7. Efficacy of excimer light therapy during follow-up period

Tabela 7. *Wyniki terapii w okresie obserwacji po leczeniu*

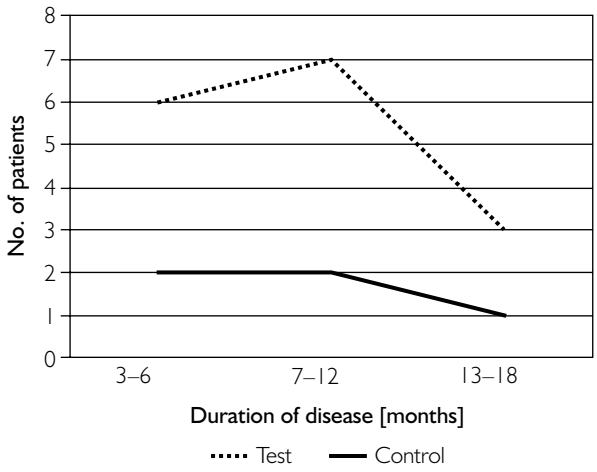
Improvements (grade)	2 months after treatment		4 months after treatment	
	Test site (N = 30), n (%)	Control site (N = 30), n (%)	Test site (N = 30), n (%)	Control site (N = 30), n (%)
1 (< 25%)	12 (40)	23 (76.67)	9 (30)	23 (76.67)
2 (26–49%)	6 (20)	5 (16.66)	5 (16.67)	2 (6.67)
3 (50–75%)	11 (36.67)	2 (6.67)	9 (30)	4 (13.33)
4 (> 75%)	1 (3.33)	0	7 (23.33)	1 (3.33)

Table 8. Efficacy of excimer light therapy according to duration of disease (at end of 4-month follow-up period)

Tabela 8. *Wyniki leczenia w zależności od czasu trwania choroby (4 miesiące po zakończeniu terapii)*

Duration [months]	> 50% hair regrowth (grades 3 and 4)	
	Test site	Control site
3–6	6 (50)	2 (16.67)
7–12	7 (46.67)	2 (13.33)
13–18	3 (100)	1 (33.3)

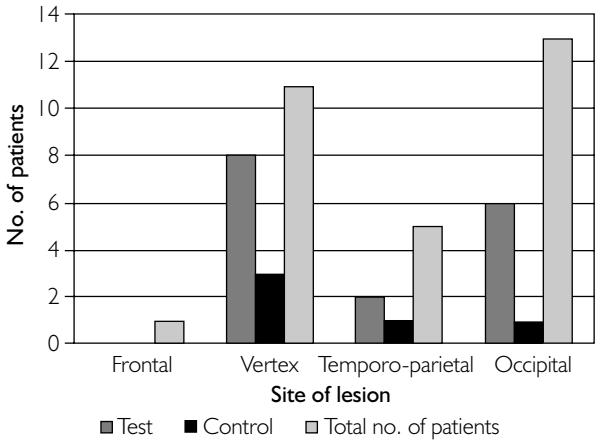
ment, successful hair regrowth (grades 3 and 4) was seen in 16 patients at test sites and in 5 patients at control sites. The difference was statistically significant ($p < 0.05$) (Table 7, Figure 6). There was no statistically significant difference in the response of treatment in relation to the duration of the disease ($p > 0.05$) (Table 8, Figure 7). The best response was seen at the vertex (Table 9, Figures 8 and 9 A–D). No serious side effect was seen in our study. None of the patients showed recurrence during the follow-up period.



The largest group of patients (7, 46.67%) with significant hair regrowth was seen among the subjects who had alopecia for 7–12 months.

Figure 7. Efficacy of excimer light therapy according to duration of disease

Rycina 7. *Wyniki leczenia w zależności od czasu trwania choroby*



The largest group of patients (13, 43.33%) had alopecia in the occipital region.

Figure 8. Efficacy of excimer light therapy according to sites of lesion on scalp

Rycina 8. *Skuteczność naświetlań w zależności od lokalizacji ognisk na głowie*

Table 9. Efficacy of excimer light therapy according to sites of lesion on scalp

Tabela 9. *Skuteczność naświetlań w zależności od lokalizacji ognisk na głowie*

Site	No. of patients ($N = 30$), *n* (%)	Test site > 50% re-growth (grades 3 and 4), *n* (%)	Control site (grades 3 and 4), *n* (%)
Frontal	1	0	0
Vertex	11 (36.67)	8 (72.73)	3 (27.27)
Temporo-parietal	5 (16.67)	2 (80)	1 (20)
Occipital	13 (43.33)	6 (84.62)	1 (15.3)

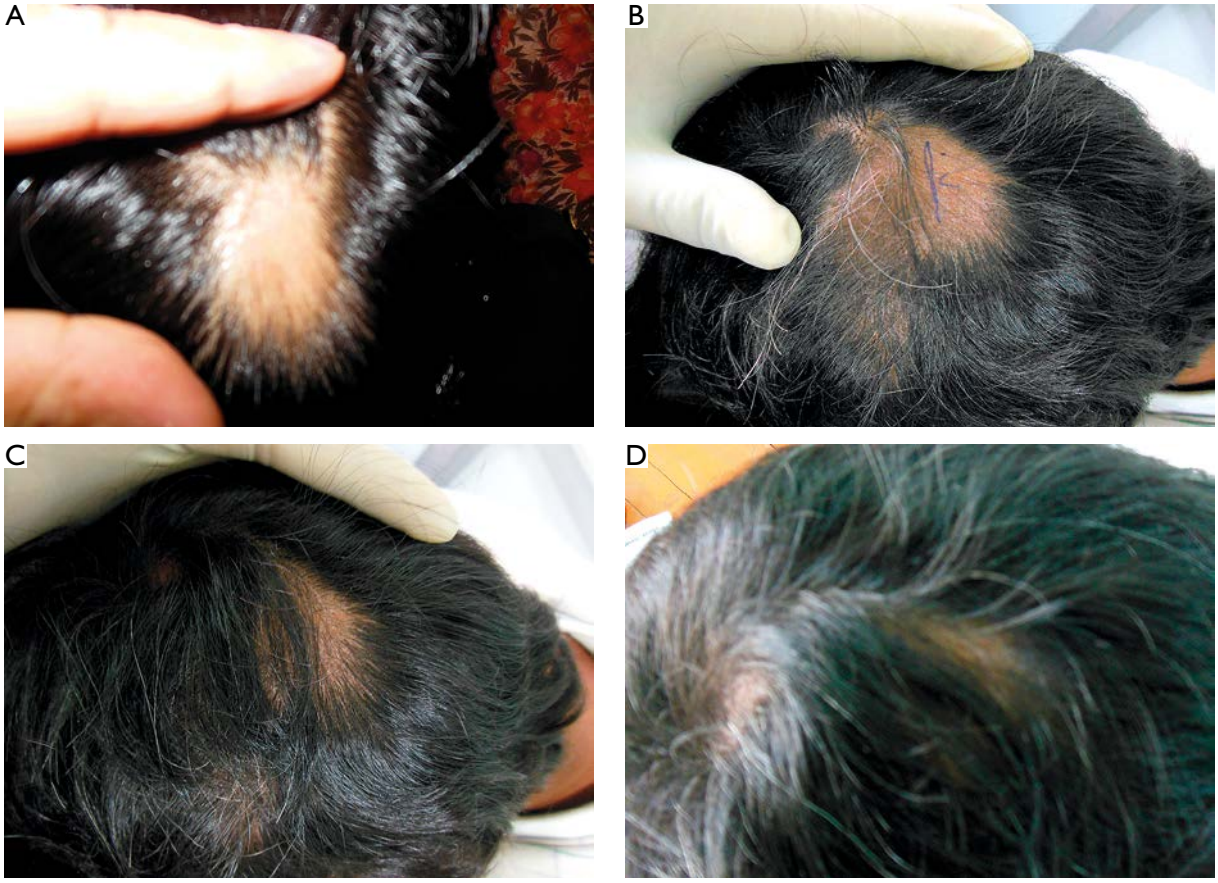


Figure 9. Efficacy of excimer light in AA: **A** – baseline, **B** – at end of treatment, **C** – 2-month follow-up, **D** – 4-month follow-up

Rycina 9. *Skuteczność światła ekscymerowego w AA:* ***A*** *– przed leczeniem,* ***B*** *– po zakończeniu terapii,* ***C*** *– po 2 miesiącach obserwacji,* ***D*** *– po 4 miesiącach obserwacji*

DISCUSSION

Alopecia areata is a chronic inflammatory disease that involves hair follicles, and sometimes nails. T-lymphocytes are believed to play a critical role in the development of AA [12, 13]. Several AA treatments utilize topical immunomodulators in terms of topical corticosteroids or topical immune sensitizers [14, 15]. UV radiation in the form of UVA or UVB has been used successfully to modulate T-cell responses in various T-cell-mediated cutaneous disorders, including AA [16]. 308 nm excimer light is a recent addition to our repertoire of light-based therapy, and has been used, like other light sources, in T-cell mediated diseases such as psoriasis and vitiligo. It offers the advantage of selective irradiation of the affected areas while sparing the surrounding unaffected skin (targeted effects) [7]. With this device, UVB can be irradiated to only one or a few individual patches, with adequate protection of the surrounding healthy skin. So far, there have been only a few studies [7, 17–20] on the use of excimer light therapy in AA.

The course of AA is very erratic and impossible to predict [16]. In general, the younger the age of the patient at onset and more widespread the disease, the poorer is the prognosis [21, 22]. Spontaneous hair regrowth, however, has been observed more frequently in single AA patches [23]. In that respect, the choice of a single patch of AA for assessing a therapeutic modality may not be ideal. However, it is

noteworthy that the mean duration of the disease in our study group was 8.2 months and the majority of patients had received some form of treatment before enrolment in this study without noticing any improvement. This can be taken as justification for choosing single patches of AA for our study. Our light source was a xenon chloride excimer lamp which emits the total energy at a wavelength of 308 nm (regarded as a "super-narrowband" UVB light source). In contrast, narrowband UVB primarily emits polychromatic, non-coherent light. Two systems emitting high-energy monochromatic excimer light (MEL) have been developed: (i) laser technology and (ii) a new non-laser MEL technology. This new MEL is 17 times more powerful compared with a Philips UVB TL-01 source. The advantages over the laser system are low operating costs and the fact that a large area can be treated quickly. In contrast, the 308-nm excimer laser systems produce a small spot size which requires multiple treatments of adjacent areas to cover the lesional skin [24].

The choice of initial dose in most of the studies that have used a light source as a therapeutic modality is determined by MED [7, 17–20]. We, however, did not follow the MED protocol and chose an initial dose of 300 mJ/cm^2 without determining MED in our patients. The reason why we chose this approach was as follows: firstly it may not be easy to calculate MED in type IV and V skin, which is present in all our patients, and secondly, Gundogan *et al*. [17] also used the initial dose of 300 mJ/cm^2 in an AA patient who had type 1 skin and 600 mJ/cm^2 in a patient with type III skin. In each patient, we irradiated only one half of the patch, and the other half covered with a black rubber shield served as a control. We understand that this might not constitute an ideal control, but we believe that by doing so, we were able to bring more objectivity to our results. The irradiation dose was increased by 100 mJ/cm^2 at every visit, which was in contrast to a study conducted by Ohtsuki *et al*. [7] in which the increment was by 50 mJ/cm^2. The therapy was given twice a week (but not on consecutive days) for a period of 2 months (16 sittings). When the patient developed fine erythema, no further increase in the irradiation dose was made. The total number of irradiations and the duration of treatment have varied in different studies that have used excimer light for AA [7, 17–20, 24]. Our choice of 2 months of treatment was based on these studies. The maximum number of patients showed erythema at doses varying between 1200 and 1600 mJ/cm^2. Cumulative dose ranged from 14.7 J/cm^2 to 16.8 J/cm^2. This is in contrast to a study conducted by Ohtsuki *et al*. [7] in which the cumulative dose ranged from 0.75 J/cm^2 to 12.8 J/cm^2. These differences in the erythema dose and cumulative dose could be due to the differences in skin type. We assessed our results on the basis of serial photographs taken with standard settings at the beginning and end of treatment, and at 2 months and 4 months after treatment. Serial photography has been used as an assessment tool in many other studies also [7, 17–20, 24]. Recently, Ganjoo and Thappa [25] used a dermoscope as a tool for therapeutic evaluation. Scoring was done according to the regrowth of both vellus and/or terminal hairs as follows: < 25% hair regrowth (poor response – grade 1), 25–49% (fair response – grade 2), 50–74% hair regrowth (good response – grade 3) and 75–100% hair regrowth (excellent response – grade 4). Grades 1 and 2 were considered as "ineffective treatment/failure", and grades 3 and 4 were considered as "successful/effective treatment".

At the end of the treatment period (16 irradiations), none of the patients showed a grade 4 response at the test and control site. Only one patient showed a grade 3 response at the test site. This poor response at the end of the treatment period was in contrast to a study conducted by Ohtsuki *et al*. [7], who reported that 4 out of 7 patients (57%) with a single AA patch showed > 50% hair regrowth at the end of the treatment period. Al-Mutairi [19] reported that after 24 sessions of excimer laser, complete regrowth of hair was seen in 14 out of 22 pediatric patients (63.6%) with AA.

After 2 months of the follow-up period, 12 patients (40%) showed successful hair regrowth (grades 3 and 4) at the test site and 2 patients at the control site. This difference in the hair growth between the test site and control site was statistically significant ($p < 0.05$). This trend continued at 4 months of the follow-up period, showing successful (grades III and IV) hair regrowth at the control site. The delayed response seen in our study was slightly surprising, but if we consider the mechanism of action of excimer light in AA, this can be explained. By inducing T-cell apoptosis in patients with AA, excimer light might remove the inhibitory influence of T-cells on hair growth. Once this inhibitory influence of auto-reactive T-cells is removed, hair will re-enter anagen, and this will take some time.

There was no statistically significant difference in response to therapy between test sites and control sites in relation to the localization and duration of AA. This is in contrast to other studies which have reported that longer duration of the disease has a negative impact on response to the treatment [25].

Most of the patients accepted the therapy well. We did not observe any serious side effects such as severe erythema, blisters, crusting and necrosis, although 5 patients had to drop out due to persistent symptomatic erythema.

Most of the studies have looked for recurrence during the follow-up period [7, 17–20, 24]. We however did not observe any relapse/recurrence at that time – on the contrary, better hair growth was observed during this period. Our follow-up period was only four months, which was shorter than in most of the studies on this subject [7, 17–20, 24]. A longer follow-up period might have thrown more light on the reliability of regrowth of hair.

CONCLUSIONS

Our study suggests that excimer light has the potential to stimulate hair regrowth in patches of AA. The delayed response in hair regrowth was striking and should be borne in mind. Whether excimer light will find a place in the therapeutic armamentarium of AA can only be answered if larger double blind controlled multicentric trials are performed. In the future, critical evaluations must be performed with regard to the optimal time of treatment, dosage, and the corresponding effects on clinical improvement.

Conflict of interest

The authors declare no conflict of interest.

References

1. **Norris D.:** Alopecia areata: current state. J Am Acad Dermatol 2004, 51 (suppl 1), S16-S17.
2. **Wassherman M., Guzman-Sanchez D.A., Scott K., McMichael A.:** Alopecia areata. Int J Dermatol 2007, 46, 121-131.
3. **Martinez-Mir A., Zlotogorski A., Gordon D., Petukhova L., Mo J., Gilliam T.C., et al.:** Genome wide scan for linkage reveals evidence of several susceptibility loci for alopecia areata. Am J Hum Genet 2007, 80, 316-328.
4. **Tosti A., Bellavista S., Iorizzo M.:** Alopecia areata: a long term follow-up study of 191 patients. J Am Acad Dermatol 2006, 55, 438-441.
5. **Safavi K.H., Muller S.A., Suman V.J., Moshell A.N., Melton L.J.:** Incidence of alopecia areata in Olmsted County, Minnesota. Mayo Clin Proc 1995, 70, 628-633.
6. **Sundberg J.P., Silva K.A., Li R.H., Cox G.A., King L.E.:** Adult-onset alopecia areata is a complex polygenic trait in the C3H/HeJ mouse model. J Invest Dermatol 2004, 123, 294-297.
7. **Ohtsuki A., Hasegawa T., Komiyama E., Takagi A., Kawasaki J., Ikeda S.:** 308-nm excimer lamp for the treatment of alopecia areata: clinical trial on 16 cases. Indian J Dermatol 2013, 58, 326.
8. **Mehta B.A., Raka A., Bharmbhatt V.:** Comparative study of various regimens in alopecia areata. Int J Dermatol 2012, 9, 1.
9. **Rangwala S., Rashid R.M.:** Alopecia: a review of laser and light therapies. Dermatol Online J 2012, 18, 3.
10. **Olsen E.A.:** Investigative guidelines for alopecia areata. Dermatol Therapy 2011, 24, 311-319.
11. **Ghersetich I., Campanile G., Loti T.:** Alopecia areata: immunohistochemistry and ultrastructure of infiltrate and identification of adhesion molecule receptors. Int J Dermatol 1996, 35, 28-33.
12. **Marwah M., Nadkarni N., Patil S.:** Hover'ing over alopecia areata: histopathological study of 50 cases. Int J Trichol 2014, 6, 13-18.
13. **Panda M.:** Clinico-epidemiological profile and therapeutic response of alopecia areata in a tertiary care teaching hospital. J Pharm Sci Res 2014, 6, 169-174.
14. **Majid I., Keen A.:** Management of alopecia areata: an update. Br J Med Pract 2012, 5, 530.
15. **Otberg N.:** Systemic treatment for alopecia areata. Dermatol Therap 2011, 24, 320-325.
16. **Dombrowski N.C., Bergfeld W.F.:** Alopecia areata: what to expect from current treatments. Cleve Clin J Med 2005, 72, 758-768.
17. **Gundogan C., Greve B., Raulin C.:** Treatment of alopecia areata with the 308-nm xenon chloride excimer laser: case report of two successful treatments with the excimer laser. Lasers Surg Med 2004, 34, 86-90.
18. **Raulin C., Gundogan C., Greve B., Gebert S.:** Excimer laser therapy of alopecia areata side by side evaluation of a representative area. J Dtsch Dermatol Ges 2005, 3, 524-526.
19. **Al-Mutairi N.:** 308-nm excimer laser for the treatment of alopecia areata in children. Pediatr Dermatol 2009, 26, 547-550.
20. **Al-Mutairi N.:** 308-nm excimer laser for the treatment of alopecia areata. Dermatol Surg 2007, 33, 483-487.
21. **Seetharam K.A.:** Alopecia areata: an update. Indian J Dermatol Venereol Leprol 2013, 79, 563-575.
22. **Wadhwa S.L., Khopkar U., Nischal K.C.:** Hair and scalp disorders. [in:] IADVL Textbook of Dermatology. 3rd edition. R.G. Valia (eds). Bhilani Publishing House, 2008, 902-903.
23. **Talpur R., Vu J., Bassett R., Stevens V., Duvic M.:** Phase I/II randomized bilateral half-head comparison of topical bexarotene 1% gel for alopecia areata. J Am Acad Dermatol 2009, 61, 592-599e.
24. **Spencer J.M., Hadi S.M.:** The excimer lasers. J Drugs Dermatol 2004, 3, 522-525.
25. **Ganjoo S., Thappa D.M.:** Dermoscopic evaluation of therapeutic response to an intralesional corticosteroid in the treatment of alopecia areata. Indian J Dermatol Venereol Leprol 2013, 79, 408-417.

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