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Photochemotherapy for Alopecia Areata

A. L. Claudy and D. Gagnaire

Department of Dermatology, Hôpital Bellevue,
F-42023 St Etienne Cedex, France

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Abstract. Ten patients with alopecia areata (plaque type and totalis type) were treated with oral photochemotherapy. Successful results were obtained in 7 cases. No

side effects were noted. Hair growth was related to the energy delivered and was not dependent on the duration of the disease. No relapses occurred after discontinuation of the treatment. The hypothesis concerning the mechanisms of hair regrowth are discussed.

Key words: Alopecia areata; 8-MOP; Photochemotherapy

Alopecia areata (AA) is a disease of unknown origin. Alopecia totalis and alopecia universalis are the most severe forms, and the prospect of spontaneous hair growth are very poor. Up to the present, therapy has consisted of corticosteroid drug administration, either intralesional, topical, or systemic (9). New treatment modalities have been recently proposed, using sensitization to dinitrochlorobenzene (DNCB) (1, 3, 6), and topical photochemotherapy (8, 10). We present data obtained from 10 patients with AA treated with oral photochemotherapy.

PATIENTS AND METHOD

Ten patients were studied—8 men and 2 women. Their ages ranged from 11 to 48 years. 5 patients had AA with multiple plaques and 5 had alopecia totalis. The duration of the disease ranged from 2 months to 16 years. Informed consent was obtained from all participants.

Light source. Hexagonal stand-up light boxes containing 84 fluorescent tubes mounted side by side in a vertical plane were used as UVA source. These tubes have a continuous spectrum of high intensity irradiation between 320 and 400 nm with a peak emission at 365 nm. The energy delivered was, on average, 8–8.5 mW/cm² at a distance of 50 cm.

8-methoxypsoralen (8-MOP). Ten-milligram capsules of 8-MOP were given 2 hrs prior to UVA exposure, according to the patient's weight (from 10 mg for 25 kg or less, up to 60 mg for weights greater than 90 kg).

Table I.

M. P = multiple plaques, A. T = alopecia totalis

Case no.	Age	Sex	Clinical form	Duration	Energy (J/cm ²)	No. of irradiations	Hair growth (%)
1	48	M	M. P	6 mo.	84	14	90
2	22	M	M. P	6 mo.	406	36	90
3	44	M	M. P	2 mo.	444	50	100
4	11	M	M. P	1 yr.	312	46	0
5	27	M	M. P	8 mo.	430	50	90
6	30	F	A. T	16 yrs.	218	24	0
7	12	F	A. T	11 mo.	392	40	0
8	27	M	A. T	3 yrs	784	80	90
9	32	M	A. T	10 yrs	685	69	90
10	18	M	A. T	10 yrs	729	65	100

Exposures and UVA dosages. These patients were irradiated three times a week, allowing 2 days' rest after each exposure. The dosage of UVA was increased by one J/cm² on the first and third exposure.

RESULTS

Three out of 5 patients with alopecia totalis had a clinically substantial hair regrowth (more than 90% of the previous bald area). 4 out of 5 patients with multiple plaque AA achieved a total hair growth. Redness of the scalp invariably occurred prior to hair growth. The energy required for the initial response was from 85 to 120 J/cm². Satisfactory results were obtained with higher energy for alopecia totalis (685–784 J/cm², mean 732) than for multiple plaque AA (84–444 J/cm², mean 341).

Three patients failed to respond to PUVA treatment. The energy delivered to these patients was lower than that delivered to the other patients. We decided to discontinue the treatment, when no sign of hair growth could be observed. Failure to respond did not seem to be related to the duration of the disease, since these 3 patients had had AA for 16 years, one year, and 11 months, respectively.

We have been able to discontinue the PUVA treatment without any recurrence of the disease after more than 6 months' follow-up. No side effects were noted.

DISCUSSION

Immunologic mechanisms are thought to play a pathophysiological role in AA. Mononuclear cells surround affected hair follicles, antibodies directed against various autoantigens may be present (2), and an imbalance between the B-T lymphocyte system and/or between the T helper-T suppressor cell system has recently been suggested and thought to be responsive for AA (4, 6). Corticosteroid drugs have been recommended, and often successfully used, for their immunosuppressive effect. On the other hand, restoration of an active T lymphocyte pool obtained by sensitizing patients with DNCB has been proposed, with impressive results (1, 3, 6). In 1974, Rollier et al. (8) used 8-MOP and sunlight to induce hair growth and more recently Weissman et al. (10) proposed topical photochemotherapy, with satisfactory preliminary results.

On the premise that AA may be influenced by immunological factors and that PUVA is supposed to induce a modification in both the *in situ* and the circulating lymphocyte population (7), we decided

to treat AA patients with oral photochemotherapy. Our preliminary results are so far encouraging, since 7 of 10 patients had a hair growth on more than 90% of the previous bald area. The energy required in our study to induce hair growth was greater than that needed in Weissman's study on 5 patients (10).

The mechanisms by which hair growth occur is still unknown. Although we cannot exclude the possible role of a primary irritant effect of PUVA, it has been demonstrated that a decrease in a subset of thymo-dependent lymphocytes takes place during oral photochemotherapy (5, 7). It is conceivable that PUVA non-specifically suppresses the immune response against the hypothetical hair-associated antigen responsible for AA.

Photochemotherapy for AA is still in the experimental stage and its benefits should be carefully weighed against the possible risks associated with its use. We recommend that PUVA should only be resorted to when classical treatments have failed.

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