

Unfortunately the present study failed to show any effect of TA in CU. Good results in the preliminary studies (6) must have been due to a placebo effect, which often is recorded in CU (8). The lack of effect could either be because plasmin plays no part at all in the development of symptoms of CU, or because patients with only a slightly reduced C₁-esterase inhibitor level have an almost normal inhibition of plasmin, so that treatment with an inhibitor will not give rise to any noticeable change in the symptoms.

REFERENCES

1. Champion, C. H. & Lachmann, P. J.: Hereditary angio-oedema treated with E-aminocaproic acid. *Br J Dermatol* 81: 763, 1969.
2. Donaldson, V. H. & Evans, R. R.: A biochemical abnormality in hereditary angioneurotic edema. *Am J Med* 35: 37, 1963.
3. Hadjiyannaki, K. & Lachmann, P. J.: Hereditary angio-oedema: a review with particular reference to pathogenesis and treatment. *Clin Allergy* 1: 221, 1971.
4. Lundh, B., Laurell, A.-B., Wetterquist, H., White, T. & Granerus, G.: A case of hereditary angioneurotic edema successfully treated with E-aminocaproic acid. *Clin Exp Immunol* 3: 733, 1968.
5. Sheffer, A.-L., Austen, K. F. & Rosen, F. S.: Tranexamic acid therapy in hereditary angioneurotic edema. *N Engl J Med* 287: 452, 1972.
6. Zachariae, H.: Cyklokapron® treatment in hereditary angioneurotic edema. *Trans XX Scand. Congr. Dermatol.* p. 36, Stockholm, 1974.
7. Zachariae, H., Laurberg, G. & Hjortshøj, A.: Tranexamic acid (Cyklokapron®) in hereditary angioneurotic edema. *Ugeskr Laeger* 137: 1106, 1975.
8. Zachariae, H., Niordson, A.-M. & Henningsen, S. J.: Indomethacin in urticaria and histamine induced wealing. *Acta dermatovener (Stockholm)* 49: 49, 1969.

Treatment of Alopecia Areata with DNCB—An Immunostimulation?

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Abstract. Ten patients with long-standing areate type alopecia totalis were sensitized with 1-chloro, 2, 4-dinitrobenzene (DNCB). Following sensitization they

were painted once weekly on a 40×20 mm area of the vertex with DNCB in acetone, in concentrations adjusted to the allergic response.

After 7 weeks, growth of hair was seen in the painted area in 3 patients and after 8 weeks all over the scalp in 3 other patients.

Key words: Alopecia areata; DNCB; Immunostimulation

Alopecia areata is often combined with atopy, thyroid diseases, vitiligo, chronic mucocutaneous candidiasis, and the presence of specific autoantibodies.

At the Department of Dermatology, the Finsen Institute, Copenhagen, 60 patients with alopecia areata were screened clinically and immunologically and 10 patients with alopecia totalis were treated with 1-chloro, 2, 4-dinitrobenzene (DNCB) according to the method described by Rosenberg (1).

Close relatives of two-thirds of the 60 patients had alopecia areata, atopy, or autoimmune-endocrine diseases. Abnormal immunological reactions and conditions usually connected with reduced resistance to infections were found in two-thirds of the patients. A group of 10 patients with alopecia totalis (average duration 2 years) were sensitized with 1 mg DNCB in acetone (closed patch test). 14 days later a DNCB dilution series was applied and the weakest dilution in $\mu\text{g}/\text{cm}^2$ to give ++ reaction was recorded as the sensitization titre. The reactions to DNCB did not differ from sensitization titres in normal individuals. Thereafter a 40×20 mm area symmetrically over the centre line of the scalp was painted with DNCB in acetone. A



Fig. 1. Hair growth after twelve applications of 1-chloro, 2,4-dinitrobenzene in acetone.

bullous allergic inflammation developed within the next 2 days. The procedure was repeated every week for 12 weeks or until hair growth was seen. After 7 weeks of treatment with DNCB, growth of hair on the painted area was noted in 3 patients.

After 8 weeks, 3 more patients showed growth of hair, not only on the painted area, but all over the scalp.

Excision biopsy from the border of the treated area 2 weeks after the last DNCB application (Fig. 1) showed histologically a normal quantity of normal hair follicles throughout the biopsy. From the part of the biopsy without visible hair, the follicles were situated in the upper dermis as seen in alopecia areata. In the other part of the biopsy,

where hair growth had started, the follicles were situated in the hypodermis.

Direct and indirect immunofluorescence investigations proved negative. The histological finding suggests a normalization of follicle siting, but the mechanism seems obscure. However, the effect of the induced allergic inflammation, as well as the high frequency of immunological abnormalities in patients with alopecia areata and their families, favours the assumption that alopecia areata is a skinmanifestation of an altered immunoreactivity.

REFERENCE

1. Society Transactions. *Arch Dermatol* 112: 256, 1976.