



Introduction

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Alopecia areata (AA) is a frequent cause of hair loss. The origin of the disease is not fully understood. However, it is most important that it is a T-cell mediated autoimmune process. Genetic, immunologic and psychologic factors are important for the outbreak of the disease. Most patients show localized patches of acute hair loss, where regrowth observed spontaneously or with simple topical treatment within few months. In up to 15% of patients severe forms of disease can be developed with total scalp; alopecia totalis (AT) or scalp and body hair loss; alopecia universalis (AU). There are only few known risk factors for development of a severe form. Although spontaneous remission is possible in these cases, it occurs rarely and treatment is difficult. The unpredictable course of the disease is a major handicap for clinical trials and treatment recommendations (*Friedli and Harms, 2002*).

There is no permanent cure and there is no universally proven therapy for inducing remission. There are different modes of therapies for which partial success has been claimed but as soon as any of the therapies are stopped, AA returns. All the current modalities used are more effective in those with milder forms of the disease but much less effective in people with extensive hair loss (*Freyschmidt et al., 2001 a*).

Treatment is challenging and aims at the regrowth of hair in affected individuals. Intralesional corticosteroid

injections are widely used in milder disease. Topical anthralin and minoxidil may also be clinically efficacious. Topical sensitizers are sometimes employed. Various therapies for the disease may have efficacy in different patients, making a universal treatment difficult to implement(*Papadopoulos et al., 2000*).

Topical therapy using contact sensitizers has been practiced since the 1960s to treat conditions associated with an altered immunological state. Dinitrochlorobenzene, squaric acid dibutyl ester and diphenylcyclopropenone are most commonly employed in the therapy of AA(*Buckley and Vivier, 2001*).

Diphenylcyclopropenone has been reported to be an effective topical immunotherapy for extensive AA with a tolerable side effects(*Galadari et al., 2003*).