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ALOPECIA AREATA IN IDENTICAL TWINS

To the Editor,

Two 28-year-old, identical twins developed alopecia areata (AA) of the beard region simultaneously 2½ years ago. Both had a lesion each, one had involvement of the right cheek, while the other had the lesion on the left cheek. The use of potent topical corticosteroid (fluocinolone acetonide 0.1%) led to complete regrowth of hair in both patients within 3 months. One of them developed a new lesion in the same location 3 months later which responded to same treatment again. The other patient developed a new lesion on the same side of the face 2 years later, which had shown partial regrowth of hair after 2 months of treatment with topical fluocinolone acetonide 0.1% Cutaneous examination revealed patchy hair loss with no skin atrophy or any other surface changes.

Detailed history revealed no stress factor that could have contributed to the development of alopecia in these patients. Neither of them had history of any other autoimmune disease.

The development of AA has been observed in several members of the same family.[1] The incidence of family history of AA has been reported in upto 27% of the patients. There are only a few reports of AA occurring in identical twins.[2-4] The occurrence of AA in the members of the same family especially in the identical twins, supports the hypothesis that the patients of AA are genetically predisposed to developing this disease.

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LEVAMISOLE IN VITILIGO OF EYELIDS

To the Editor,

The treatment of vitiligo has been disappointing and is indeed an arduous challenge for dermatologists. Although no therapeutic panacea exists, a variety of treatments benefit innumerable patients. Currently, the major therapeutic measures for vitiligo include psoralens and corticosteroids, topically and/or systemically, either singly or in various combinations.[1,2] Because melanocytes are indolent and slow responders to all current treatment modalities, treatment must be continued for 6 to 12 months for an optimal response.[3] We wish to share our experience with oral levamisole, an immunomodulator, and topical hydrocortisone butyrate cream used for treating vitiligo involving eyelids.

Since 1993 we have been treating our cases of vitiligo with oral levamisole 150 mg (50 mg for children) on two consecutive days every week combined with topical 0.1% hydrocortisone butyrate cream applied twice

daily. We have registered 250 cases of vitiligo having different presentations, 17 of whom also had involvement of the eyelids. During follow up we observed that 15 of the 17 patients (88.23%) had complete repigmentation of their lesions over eyelids in a period ranging from 6 to 8 weeks. In subsequent visits for treatment of other lesions ranging from 1 to 3 years they did not show any recurrence of the disease over the eyelids. Hence, we conclude that oral levamisole combined with topical hydrocortisone butyrate cream may be the treatment of choice for eyelid vitiligo irrespective of age of the patient and duration of the disease.

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GENESIS OF CUTANEOUS DEPIGMENTATION

To the Editor,

Theories[1] on the aetiology of leucoderma abound but data about its initiation, progression or recession is extremely sparse. This report seeks to understand the causes and progress of cutaneous depigmentation.

Each chemical (2% w/v or v/v in Et. OH): hydroquinone (OH. C_6H_4. OH), p (benzyloxy)phenol (C_6H_5. CH_2O. C_6H_4. OH) p-hydroxypropiophenone (OH. C_6H_4.CO.CH_2.CH_3) butylphenol (C_4H_9.C_6H_4.OH) and amylphenol (C_5H_{11}.C_6H_4.OH) having a common hydrogen donor group (-OH), when appllied topically for 20 days on the vitiligo patients (6 in active stage and 4 in steady stage) and 10 controls without any skin disease, caused pigment dilution on the spots involved, progessing slowly with the repeat application of the chemical. In both groups depigmentation may be the consequence of a reaction between the colour determinant and the chemical in epidermis. The response after the cessation of the chemical application was different; original status of the test spots was restored in control groups after 20 to 30 days, but the acquired transition persisted in the patients with vitiligo. These observations clearly indicate the loss of a mechanism in the vitiligo patients. Further except cosmetic defect, the vitiligo patients show soundness in clinical status like those of healthy individuals. Also both these groups respond alike to the chemical structures with antigenic determinants and are capable to rid off these non-self structures through complement fixation, suggesting that the biomechanisms to neutralise the toxins with and without antigenic determinant(s) are active in the control groups whereas the mechanism to deal effectively with the candidate chemical structures, which fail to elicit antibodies, is lost in the vitiligo patients. Further in controls, skin colour is conditional upon the molecular viability of the colour determining melanolipoprotein as envisaged by Sawhney,[2] and sustainment of the threshold limits of toxin concentration in skin matrix. The bioactive inherent disposition mechanism armed with a neutralising protein does this job through conjugation of the intruders. However the skin faculty is not endowed with the ability to perform protein synthesis,its demand is met through the likely events which include the hepatic synthesis of such neutralising protein and its subsequent transfer via plasma to the