Psoralen has been mentioned to be effective in a variety of conditions like alopecia areata, xeroderma pigmentosa, psoriasis, mastocytosis and even in fungal infections. Various postulations regarding its mechanism of action in the treatment of alopecia areata have been put

forward only recently. More recent articles in the international journals mention about this. I am not competent to comment on its effectiveness in arresting the frontal regression of hairs in males.

Dr. K. Pavithran

LOCALISED HYPERTRICHOSIS DUE TO ORAL 8-MOP

The exact mechanism of iatrogenic hypertrichosis is unknown, though several drugs (diphenylhydantoin, minoxidil, streptomycin, cortisone and penicillamine) are known to cause this.¹ That psoralens are capable of inducing hair growth is not a recent observation. El Mofty in his book 'Vitiligo and Psoralens' (1968) had mentioned the usefulness of topical psoralens for the treatment of alopecia areata. Instances of oral psoralen induced hypertrichosis were also reported.² The recent publication of reports on topical psoralen induced hypertrichosis by Pavithran³ prompted me to communicate my observation of a case of oral 8-MOP induced hypertrichosis.

A 12-year-old girl with a single lesion of vitiligo of 4 years' duration was seen in 1977. She had earlier received several medications which included oral TMP for a few months and then topical psoralen solution and later 8-MOP solution. But there was no change in the lesion. The hairs in the lesion were depigmented. She was put on 20 mg of oral 8-MOP/day and 20 minutes exposure to sunlight in December 1977. In about 6 months, a definite hypertrichosis of depigmented hairs was noticed. There was some repigmentation at the periphery of the lesion and a few grey hairs had turned black in this region. Hypertrichosis of black hairs also was observed in the region surrounding the patch of vitiligo (Fig. 1).

Whether or not there is actual proliferation of melanocytes in vitiliginous areas after psoralen therapy, is not clear though it is suggested that

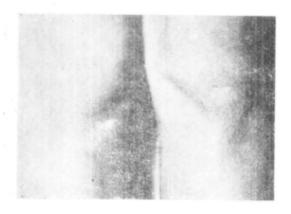


Fig. 1. Hypertrichosis of both white and black hairs in and around the lesion of vitiligo.

increased arborization of the dendrites, increased melanosome melanization and transfer, and increased tyrosinase activity occur. The rapidly dividing basal cells are inhibited by the photoadduct formation of psoralens with thymine (not histidine as mentioned by Pavithran)3 of the DNA in psoriasis. This does not apply to melanocytes, but theoretically should apply to hair matrix cells. The fact that there is no alteration in the hair growth in a majority of cases of vitiligo on psoralens, suggests that hair matrix cells are not affected. Similarly, the mechanism of hypertrichosis in an occasional case cannot be suitably explained. Repeated or long continued inflammatory changes involving the dermis may result in the growth of long and coarse hair.1 This probably is responsible in psoralen induced hypertrichosis though we should be seeing hypertrichosis much more frequently

as the minimum phototoxic dose always induces Observation of hypertrichosis of erythema. depigmented hairs without perifollicular repigmentation and hypertrichosis of black hairs in the region surrounding the vitiligo lesion in our case question Pavithran's suggestion that the stimulus which activates melanocytes could be responsible for stimulation of the follicular matrix cells. These are two entirely different processes. In any case, it would be interesting to explore further to find out the exact mechanism involved in iatrogenic hypertrichosis, especially due to psoralens. In our case, since there is excessive growth of depigmented hairs, the term hyperleucotrichosis may be preferred as it indicates the correct picture.

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Reply

It is quite interesting to note the letter from Dr. RPC Naik. The statements given in the letter with reference numbers 1 and 2 have already been mentioned in my article Hyper trichosis by topical psoralen. I thank Dr. Naik for pointing out the mistake of writing histidine instead of thymine with which psoralen forms photo-adducts. The observation of hyperleucotrichosis without perifollicular hypermelanosis, as noted by Dr. Naik is not a point

against the postulation put forward by me in the article. The number of melanocytes in the depigmented patches of vitiligo may be normal or sometimes reduced. In some patches, they may even be completely destroyed. Dutta and Mandal have found a poorer repigmentation response in those patches with achromotrichia. It is possible that in the case mentioned by Dr. Naik, the melanocytes in the hair follicles of the patch were destroyed by the immunological process. Hence, psoralen induced stimulus produced hyperleucotrichosis without melanin pigmentation in the patches.

I do agree with Dr. Naik that keratinisation and melanization in the hair follicle are two different processes. But the study by Ridi et al³ strongly supports my suggestion. They demonstrated marked hyperplastic changes (increased thickness of horny layer, acanthosis and intact hair follicles) in the beta irradiated skin of rats which were fed preliminarily with 8-methoxy psoralen. In the control normal rat skin which was not given 8-MOP; after beta irradiation there was no acanthosis and hyperkeratosis and the hair follicles were destroyed. Though E1 Mofty has mentioned about the beneficial role of psoralen solution in the treatment of alopecia areata, he has not mentioned hypertrichosis as a complication of topical psoralen therapy of vitiligo. In alopecia areata, psoralen causes regrowth of hairs in an alopecic patch. I do not think this regrowth of hair after treatment can be named as hypertrichosis. The immunopathology of alopecia areata has recently been well studied and it has been postulated that PUVA non-specifically suppresses the immune responses against the hypothetical hair associated antigen. To the best of my knowledge hypertrichosis after topical psoralen has not been reported except the one already cited in the article. I once again thank Dr. Naik for his valuable comments.