obtained in this case. The papules and nodules in present case did not fit at least clinically into any of the other skin diseases.

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## WHY LESIONS OF MORPHOEA ARE OFTEN HYPERPIGMENTED?

*To the Editor,*

The lesions of morphoea are characterized by indurated areas of skin, which at first are faintly purplish or mauve in colour. After a few weeks or months, they lose their colour, especially in the central part and appear as thickened waxy ivory coloured areas with a characteristic lilac border.[1] In Indians with mostly type IV or type V skin colour, we rarely appreciate the purplish or mauve colour and lilac border in the lesions of morphoea. Instead, in most of our patients we observe mild hyperpigmentation over the morphoea plaques. In the standard text[1] these hyperpigmented patches are stated to be present at the very beginning of morphoea lesion(s) or at the site of resolving plaque(s). However, we see these patches mostly over the well developed plaques of morphoea. The pathomechanism of such hyperpigmentation has not been elucidated in the standard textbooks.[1,2]

We have been interested to look into this aspect and to find out the status of melanocyte and basal cell layer in the histopathological sections of morphoea lesions. On Fontana-Masson stained sections, we have found that there is increased melanocytic activity in the form of prominent melanocytes in the basal cell layer. It appears that there is increased melanin synthesis inside the melanocytes. There is no basal cell degeneration and melanin incontinence. The first author has been observing this histopathological phenomenon for the last 6 years.

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## CLEARANCE OF PLAQUE PSORIASIS FOLLOWING IRRITATION DUE TO CALCIPOTRIOL

*To the Editor,*

A 9-year-old girl presented with extensive plaque psoriasis of 3 months duration. In view of inadequate response to topical coal tar and steroid therapy, calcipotriol (50 µg/g) was started. Patient developed irritant reaction to topical medicament within a

week of commencement of therapy, manifesting as severe burning sensation locally associated with aggravation of lesional and perilesional erythema. Calcipotriol was discontinued for the fear of precipitating exfoliative dermatitis and patient was put on topical emollients. Surprisingly review after two weeks revealed complete regression of skin lesions with postinflammatory hyperpigmentation.

In clinical studies calcipotriol has been shown to be an effective and well tolerated treatment for psoriasis.[1] Lesional and perilesional irritation is the most common adverse effect reported with the drugs.[2] Clearance of lesions following irritant reaction could be attributed to increased tissue concentration of drug subsequent to enhanced blood supply following inflammation or reverse Koebner phenomenon in this case.

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## EVIDENCE FOR CYTOMEGALOVIRUS INFECTION AS THE CAUSE OF VITILIGO

*To the Editor,*

The exact cause of vitiligo has not been known. I have found that (1) the melanocyte in vitiligo was a rounded structure which (2) formed the first stage of cytopathic effect (CPE) of cytomegalovirus (CMV) culminating in the loss of melanocyte to produce vitiligo. This possibility was investigated by 3 experiments, namely, (I) cytological, (II) immunofluorescent study, and (III) therapeutic test.

Four patients were selected for the first study, 2 for the second and 10 for the third. Their ages varied between 30 to 40 years and they had been suffering from vitiligo for last 5 years or more. Group I and II consisted of males alone and patients of Group III were 9 females and one male. That the patches of vitiligo were not secondarily due to syphilis, fungus infection, psoriasis, Kala-azar and eczema etc was confirmed by pathological examination and clinical history. Diagnosis in each case was confirmed by reaction to dihydroxyphenylalanine (DOPA).

For the cytological and immunofluorescent (IF) tests, pure epidermis preparations,[2] from (a) less pigmented border of vitiligo spots and from (b) contralateral part of normally coloured skin were digested in normal saline and mounted on albuminised slides. One portion of slides from (a) and (b) was subjected to treatment with DOPA, H&E and methyl green pyronin stain to visualise reaction to DOPA, nucleus and nucleolus under the light microscope. The other portion of slides from (a) and (b) were challenged with rabbit antihuman IgG sera, specific for CH2 domain, both plain and FITC conjugated, by direct and indirect methods and examined under the fluorescent microscope. The digests of normally coloured skin served as control. The results of the cytological and immunofluorescent tests are presented in Table I, as found in I, II, III and IV stages of CPE of CMV in angular melanocyte.[4] Some basal cells showed CPE of CMV upto II stage with + IF test.

For conducting the therapeutic test, 4 patients were put on idoxuridine and 4 on acyclovir iontophoresis each, passing a direct