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

Table I. Summary of cytological and IF characteristics of CPE of CMV of melanocytes in I to IV stages of degeneration and of basal cells upto stage II of degeneration. Abbreviations are, N=nucleus, NL=nucleolus, IB=Inclusion body, RD=DOPA reaction. All measurements are in U.CF=Contrast from, FDP=Fine dendritic processes;DP=dendritic process

Stages	Angular melanocyte				Nucleus			Special features
	RD	Whole cell	Body	Shape of body	Size	Shape	Location	
I	+	70x12	12x10	rounded	8x10	egg round	Central	anterior notches in N lost (CF), N is marginated (N)
II	±	80x12	12x12	rounded	8x12	oval elongated vacuole	eccentric	NL at wall of N. Bizzare form of NL in N. IB+
III	-	100x55 cytomegaly	Bald, FDP lost Elongated	tubular	10x12	tubular,into root of DP large vacuoles	whole cell	IF+ in II or III stage seen in trident, straight melanocyt. Basal cell IF+
IV	-	giagatic	Fractionated			Bizzare or in twos		Lost by fractionation or necrosis

current of 10 V at 3-4 mAmp for 15 to 20 minutes for 60 days, introducing psoralen ultraviolet ray A range therapy (usually designated as PUVA) after 15 to 20 days, when the vitiliginous spots had regained normal colour. Two patients were kept for control. All the 8 patients were completely cured without any relapse for the last 5 years (UK Patent No. 2251380, published on 13.7.1994). The treatment by iontophoresis was restricted to an area of 3"x2" at one time. CMV infection of vitiligo explained occurrence of family history in 30% and Kobner's phenomenon.

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SPOROTRICHOID MYCOBACTERIOSIS

To the Editor,

Reports on atypical mycobacterial infection of skin have been appearing with increasing frequency in medical literature.[1] The majority are description of solitary granulomatous lesions of skin. However in several instances lesions developed in an ascending proximal fashion strongly suggesting