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.

R C Shukla
Lucknow

Acknowledgements

Thanks are due to Dako Patts for supply of sera free of cost and to Mr BA York, Director, Patents, Sandoz Pharmaceuticals, Switzerland for critical evaluation of the data on treatment and subsidy of Sw.Fr.3,000/- towards filing a patent in UK.

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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

sporotrichosis.[2,3] We report one such case of sporotrichoid mycobacteriosis.

A 37-year-old serving soldier presented with complaints of multiple nodules over right forearm of 6 months duration, appearing 3 weeks following trauma to right index finger. He had received pefloxacin therapy for a month, 3 months prior to presentation resulting in partial and temporary resolution of skin lesion. His trade involved grooming horses with history of frequent contact with mud and water. No significant past or family history was elicited.

General, physical and systemic examination revealed no abnormality. Dermatological examination revealed multiple, crusted erythematous mildly tender and indurated nodules and plaques varying in size from 0.5 to 3.0 cm in diameter in a linear distribution over extensor aspect of right forearm (Fig. 1). No lymphatic cords or regional lymph nodes were palpable.

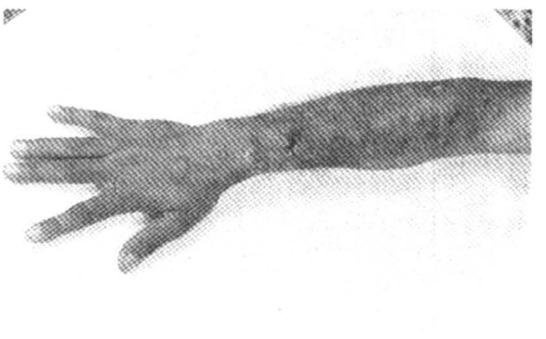

Fig. 1. Sporotrichoid skin lesions over right forearm.

Routine haemogram, urinalysis, blood sugar, LFT and serum ceratinine were within normal limits, ESR was 15mm/1st hour, Mantoux test was positive (16mm). Blood STS and ELISA for HIV were nonreactive. Skin biopsy revealed dense mononuclear infiltrate in upper dermis with few Langhans type of giant cells. No AFB, fungus or LD bodies were seen. Cultures for fungus, *Mycobacterium tuberculosis* and atypical mycobacteria showed no growth. Complete resolution of skin lesions was evident after 3 months of minocycline therapy, which is efficaceous in atypical mycobacterial infections.[4]

Appearance of lesions in a sporotrichoid pattern following trauma, histopathological features, and response to minocycline therapy in a person coming in frequent contact with mud and water is suggestive of atypical mycobacterial *(M marinum)* infection in this case. Failure to culture organism could be due to pefloxacin therapy received prior to presentation, as quinolones are documented to have activity against atypical mycobacteria.[5]

Gurcharan Singh, AK Malik,
Praveen Rodrigues
Bangalore

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SNEDDON-WILKINSON DISEASE

To the Editor,

Please refer to the article 'Sneddon-Wilkinson disease and arthritis' by S K Bose published in the Journal (1995 ; 61 : 231-2). I would like to share few of my observations on this disease.

Sneddon-Wilkinson disease (SWD) is no