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LANGERHAN'S CELLS IN TINEA CORPORIS

I wish to make a few comments on the paper by Narayanan RB, Girdhar A, Levania RK et al entitled 'OKT 6 positive epidermal Langerhan's cells in tinea corporis' published in Ind J Dermatol Venereol Leprol, 1988; 54 : 247-250.

The opinions expressed and the inferences drawn by the authors do not seem to be supported by the presently available information. As mentioned by the authors, the absence of Langerhan's cells (LC) may hamper the uptake and processing of the fungus. Dermatophytes have not been shown to be taken up by LCs for processing; however, LCs are known to gather around fungal elements in the active margin of the lesion.[1] Absence of LCs has been advanced as the reason for reduced levels of CMI and also the absence of DTH to various antigens in patients with dermatophytosis. A good number of apparently healthy patients with dermatophytosis, are known to exhibit DTH response to trichophytin and CMI values are unaltered. Indeed a high proportion of persons with chronic dermatophytosis show immediate skin test reactions to trichophytin.[2] Conversely, positivity of an immediate test is not invariably associated with a chronic infection.[3] Evidently, the immune mechanisms in dermatophytic infections are more complex than can be explained on the findings reported by the author.

Absence of LCs in the epidermis if seen independently is bound to give a very incomplete and distorted information because they are known to migrate to the perivascular areas in the dermis which is their mode of functioning as is classically seen in allergic contact dermatitis.[4,5] So, if the authors had looked into the dermis also, they might have found the LCs actively doing their job. LCs are known to revert back to their original site and number after subsidence of the inflammatory process e. g. tinea infection.[6,7] It would have been better if the authors had discussed the findings in the light of total available information and not restricted themselves to the absence of LCs from the epidermis.

The reference to the similarity of findings of reduced population of LCs in such diverse conditions as psoriasis and sarcoidosis vis-a-vis tinea infection does not seem to be relevant. Because, there is more to disappearance of LCs rather than the cytotoxic factor released by the fungus as believed by the authors.

I, however, do appreciate the efforts of the authors to help us know more about the immune mechanisms involved in diseases like dermatophytosis which are so commonly seen.

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REPLY

We thank Dr Bhushan Kumar for his comments on our paper "OKT6 positive epidermal Langerhans cells in tinea corporis." The comments are interesting. Nevertheless, we would like to clarify as follows : (1) The term fungus used in the text refers to products or antigenic components and not the whole molecule. (2) Patients with tinea corporis may be grouped as responders or non-responders according to the skin test response with trichophytin and DNCB. Our results may be of relevance to the non-responders. (3) OKT6 antibody is a specific marker for LC. It is very useful in quantitation and assessing the anatomical distribution of LC. In our studies, we could not detect T6+ LC in the dermis of tinea corporis lesions. This was not mentioned as the attempt was to quantitate them in the epidermal tissue. Since our experiments were short and had a limited objective, it is not possible to comment on the disappearance of LC. We in fact did not address this question in our study. Further, we consider it not proper to make wide extrapolations from such limited observations. (4) Our aim of comparing the findings with diverse conditions like sarcoidosis, psoriasis, vitiligo was to find if in tinea corporis also, a state of immunosuppression occurs as in other conditions.

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