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Persistence of Langhans' Giant Cells in Rapidly Downgrading Leprosy Lesions

TO THE EDITOR:

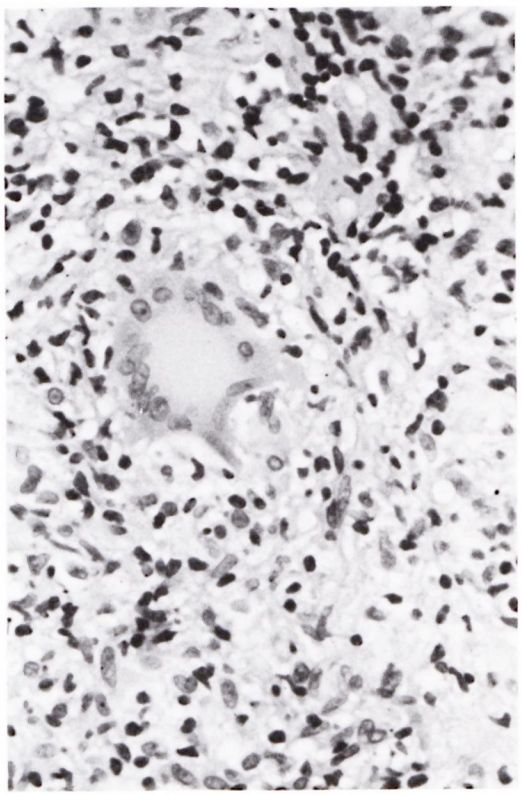
Inflammatory giant cell formation occurs in many diseases and is usually associated with granulomatous infiltration. Langhans' giant cells are a feature of the histopathological cell types found in lesions of tuberculoid and, to a lesser extent, borderline tuberculoid leprosy. They are not a feature in mid-borderline or lepromatous leprosy.

Cell-mediated immunity in borderline leprosy is unstable and, in a review of the outcome of reactions in 12 patients, it was shown that Langhans' giant cells were produced and persisted following upgrading reversal reactions but were not conspicuous in downgrading reactions ([1]).

We have recently observed Langhans' giant cells in the histopathology of rapidly downgrading leprosy.

Case A presented one month after the emergence of hypopigmented macules on his thigh and upper arm. A biopsy showed mid-borderline leprosy. Six weeks later he returned with an increase in the number of lesions, some of which were slightly erythematous. A biopsy of the left radial cutaneous nerve showed a cellular infiltration containing a few epithelioid cells, foamy macrophages, lymphocytes, and Langhans' giant cells. Acid-fast bacilli were also seen. With the exception of the Langhans' giant cells, the histological picture was that of borderline lepromatous leprosy (The Figure).

Case B was seen four months after the appearance of multiple hypopigmented lesions with poorly defined edges. The lesions had rapidly increased in number; some were marginally elevated but all had near normal sensation. Histopathology showed border-



THE FIGURE. Langhans' giant cell in an otherwise borderline lepromatous histological field from Case A.

line lepromatous to subpolar lepromatous leprosy with acid-fast bacilli. Langhans' giant cells were also present.

Case C was a patient who had been seen three times over a period of ten months. On her first presentation, she had typical hypopigmented anesthetic lesions of borderline tuberculoid leprosy. She was seen again four months later. During this period she had not taken her treatment, and the lesions were more inflamed and obvious nerve involvement was present. A biopsy showed borderline tuberculoid leprosy in reversal reaction. She was subsequently seen six months later. The disease had progressed and the lesions, which were more pleomorphic, were clinically borderline lepromatous. A biopsy confirmed this diagnosis but, in addition to the expected histological appearance, Langhans' giant cells were also seen.

The histopathology and the history in these three patients were quite similar, and all had borderline lepromatous or subpolar lepromatous leprosy. In addition, all had rapidly downgraded. Since these observations, we have seen two other patients with similar histopathology who were also considered as downgrading.

The presence of Langhans' giant cells in rapidly downgrading leprosy suggests that either these cells are capable of remarkable longevity or that the factors stimulating their formation remain present despite a diminution of cell-mediated immunity. Their persistance, together with the cellular types expected at the lepromatous end of the spectrum, may be a useful histopathological sign of rapidly downgrading leprosy.

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DDS-resistant Leprosy

To the Editor:

With reference to the paper by Almeida, *et al.* on "DDS-resistant Infection Among Leprosy Patients . . ." appearing in the September 1983 issue of the Journal (pp. 366–373), the following comments are offered:

Firstly, exclusion of 149 "not screened cases," 198 "absentees," and cases with less than 80% treatment from analysis introduces a bias in estimation. Taking overall treatment regularity as criteria for comparison does not appear to be correct since it does not discriminate the treatment regularity in the crucial initial period of treatment in a patient.

In their paper, "prevalence" is worked out as a percentage of the total number of cases "fully studied" over an unspecified period of time; whereas "incidence" is expressed as the average annual percentage of total person-years of treatment experience. It is like cutting the cake to suit the needs of the situation. If we cut a lamb into a certain number of pieces, we do not get that number of lambkins, only lamb chops considered to be a culinary delicacy. Use of person-years to work out rates in Tables 2 and 3 seems unnecessary and makes it difficult to apply statistical tests for comparison, in the way they are presented. "Prevalence" and "incidence" figures are projected in many papers on studies on DDS-resistance. Can someone elucidate the appropriate methodology for a study to find out prevalence and especially incidence rates of DDS resistance? We are in total disagreement with the interpretation of the findings in Table 3. Cases with less than 50% treatment as well as those with 50–79% treatment should have been included. If, after their inclusion, the results were found to be similar to what is projected in the table, the