CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of this JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Epidermal Langerhans' Cells in Subtypes of Leprosy

TO THE EDITOR:

The role of Langerhans' cells (LC) in antigen presentation has been well recognized in the pathogenesis of contact dermatitis ([3]). However little is known about their involvement in infectious disorders. We have studied 28 normal healthy individuals and 117 patients with different types of leprosy: 17 tuberculoid (TT), 30 borderline tuberculoid (BT), 15 borderline lepromatous (BL), and 55 lepromatous (LL). A skin biopsy was taken from the lesions; half of the tissue was processed for routine histological examination and the remaining tissue was processed to demonstrate LC ([4]). It was interesting to note that LC counts in TT patients (856 ± 154.75 LC/mm^2) were near normal (927.43 ± 103.87 LC/mm^2); whereas a gradual decline in the LC count was observed from the BT to the LL end of the leprosy spectrum (BT = 717.79 ± 188.0 LC/mm^2, BL = 490.27 ± 106.61 LC/mm^2, LL = 465.30 ± 234.0 LC/mm^2). Morphological alterations in LC, mainly in the form of increased granularity and at times lack of dendritic process, were observed only in some LL cases. In some of the BL patients at the time of downgrading, we observed large LC, suggesting that some kind of compensatory phenomenon might be going on. These findings need confirmation with electron microscopic studies.

The low counts of LC in TT and BT cases reported by Liu, *et al.* ([1]) are contrary to our observations. The possibility of defective epidermal separation could be responsible for such findings. We have also encountered similar situations in experiments where the dermoepidermal separation was not smooth, but on repeating the procedure in the same patient we found a higher count.

In our earlier communication ([2]) we tried to postulate a hypothesis about the involvement of these immunocompetent epidermal Langerhans' cells in the pathogenesis of leprosy. It is based on the observations of Ptak, *et al.* ([3]) who demonstrated that if trinitrophenylated substrate (TNP) is introduced, either by skin painting or given intravenously after conjugating it with epidermal extracts, it generates contact hypersensitivity. However, when TNP conjugated to peritoneal exudate cells was given through intravenous route, bypassing LC, it resulted in unresponsiveness for contact sensitivity since it favors activation of splenic suppressor cells. If such a phenomenon also occurs in leprosy, then it can explain the polar concept of leprosy.

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