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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.

Should Large Lesions of Leprosy Be Considered As "Multibacillary" for Treatment Purposes Even If the Total Number of Lesions Is Less Than Five?[1]

TO THE EDITOR:

According to World Health Organization (WHO) recommendations: "five, or lesser number of lesions" of leprosy should be treated as paucibacillary (PB) leprosy and should be given 6 months treatment with rifampicin monthly and dapasone daily ([5, 6]). In this type of simplified classification, the size of the patches are not considered. However, we feel that size of the patch should be considered on deciding whether to treat a case as PB with two drugs for 6 months, or as multibacillary (MB) leprosy with three drugs for a year. Categorizing leprosy as PB or MB is particularly important in areas where treatment is commenced without any bacteriological and histopathological confirmation. Even in the time honored Ridley-Jopling Classification and its modifications, large patches of leprosy are considered as a feature, more commonly found in borderline, borderline tuberculoid, or subpolar lepromatous leprosy ([2, 4]), giving due consideration to the size of the lesions.

Histopathologically, in tuberculoid leprosy there are tubercles composed of epithelioid cells. This is due to the process of destruction of lepra bacilli by histiocytes ([3]). The granulomatous reaction thus produced is the result of a combination of the presence of bacilli and the host response. Considering that sensory impairment and pathological hypopigmentation in leprosy are due to this host response by the body in the fight against the leprosy bacilli, it is likely that, the larger the lesions of leprosy, the higher the number of bacilli that cause the pathology. A granuloma which originates due to one or more bacilli in a given area can only cause a very limited spread of its effects, e.g., focal sensory loss in the affected area. The fact that inoculation of atypical mycobacteria causes a granuloma in the immediate vicinity of the inoculation, and that it spreads very slowly, suggests that proliferation of bacteria are necessary to cause a larger lesion. This also means that if there is no proliferation of bacilli in tuberculoid leprosy, there can not be evolution of a small patch, to become a large patch. This concept is further supported by the fact that even in a Type I leprosy reaction, there is no real lateral spread of a leprosy lesion, though the existing lesions temporarily become inflamed. This suggests that a pure immunological response without an increase in bacilli is unlikely to cause a lesion to spread peripherally, to produce a large hypopigmented patch. However, it is known that lowering of one's cell mediated immunity is important in promoting the spread of leprosy lesions. In this situation, the patient's ability to destroy the multiplying lepra bacilli is impaired, allowing the lesion(s) to enlarge; as in the case of a tuberculoid leprosy (PB) lesion or lesions in an untreated patient, evolving towards the "lepromatous pole" (MB) over several years.

Unless many individual cutaneous nerve

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fibers are affected by separate bacilli, there can not be sensory impairment in a large area, involving the total area of the hypopigmented macule. This is different to distal sensory impairment due to a nerve trunk involvement, for example, sensory impairment along ulnar nerve distribution. Furthermore, in monitoring tuberculoid or borderline tuberculoid leprosy, peripheral extension of a lesion is considered to be a feature of failure of treatment or relapse.

Although relapses of leprosy after treatment are reportedly uncommon, many authorities feel that they may be underestimated ([1]). If an MB case is misdiagnosed and treated with dapsone daily and rifampicin monthly as a PB case, that patient receives only 6 doses of the bactericidal drug rifampicin before stopping the treatment. This would be totally inadequate. Some authorities even believe that MB treatment should be continued for 24 months rather than the WHO recommended 12 months ([1]). In countries where leprosy is still highly prevalent, follow-up after discharge from active treatment is unsatisfactory. Therefore many relapses or suboptimal treatments may go unnoticed for many years.

Considering the above facts, we feel that where a large patch (more than 10 cm in diameter) of leprosy is present, irrespective of the size of the other lesion or lesions, the patient should be treated as MB and given treatment at least for 12 months. Just as "5 or less leprosy macules are considered as PB" (as recommended by the WHO) is an arbitrary limit, "the dimensions of a lesion" is also an arbitrary measurement, for places where microbiological and histopathological services are unavailable. It should also be emphasized that counting lesions can be erroneous if the whole body is not carefully checked by the healthcare worker. A person may have 5 easily visible lesions, but there may be another small lesion or lesions in unsuspected places such as nasal cleft, a toe, or an elbow posteriorly. In such a situation, a patient would receive only PB treatment. However, it is highly unlikely that a large patch (>10 cm) of leprosy would go undetected by the patient or the clinician or the health care worker.

In our experience with cases of leprosy in the last two decades (mostly when working in Sri Lanka) we have encountered several instances where MB cases had been categorized and treated as PB, by others, especially by public health workers, due to their strict categorization according to the "number of patches." These cases were subsequently given the MB treatment regimen. In hospital settings, facilities for biopsy and smears with microbiological evaluation are available and clinicians do no go by the number of patches alone for treatment.

Long term follow-up of patients with large macules of leprosy treated with the standard WHO treatment regimens would be necessary to ascertain whether relapse rate is higher in this group of patients. Personal experience suggests this group has more relapses or non responders.

A consensus on the duration and type of treatment for large macules of leprosy would be desirable for places where histopathological and microbiological facilities are not available. It appears prudent to treat such cases with MB treatment regimen.

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