MULTIDRUG THERAPY FOR PAUCIBACILLARY LEPROSY: WHO REGIMEN INADEQUATE?

Sir,

Multidrug therapy as recommended by the WHO study group (Technical series report 675, on Chemotherapy of leprosy for control programmes) is now the standard and accepted method for

leprosy control in many countries. The treatment schedule for paucibacillary cases consists of rifampicin 600 mg once monthly (supervised) and dapsone 100 mg daily (self-administered) for a period of 6 months. I would like to communicate my short experience in treating 18 paucibacillary cases of leprosy (7 cases of TT and 11 cases of BT) with this regimen. Before starting the treatment, confirmation of the type of the disease was made by histopathological and slit-skin smear studies. At the end of treatment period of 6 months, in 6 patients even though there was reduction in size of the lesions, the erythema and infiltration were still persisting and histology revealed tuberculoid granuloma in the dermis. In 3 other patients, though the skin lesions regressed completely leaving only atrophy and analgesia, histology showed dense collection of lymphocytes, young histiocytes and a few epithelioid cells in the upper dermis. Thus one-third of the patients who had multidrug therapy, were found to have clinical as well as histopathological evidence of persistence of the disease, while one-sixth showed histopathological activity in spite of good clinical recovery.

In the remaining patients there was neither any clinical lesion nor any histological evidence of leprosy after multidrug therapy for 6 months. But one of these patients, 11 months after completion of treatment, gradually developed erythematous and infiltrated plaque at the site of the previous lesion. There were no associated systemic features of toxicity. Slit-skin smears from the lesion were negative for acid-fast bacilli and histological features were suggestive of BT leprosy. Chemotherapy has been restarted in this patient. Another patient who had histological activity of the disease in spite of good clinical recovery at the end of treatment period, presented 7 months after completion of treatment, with erythema and infiltration which developed slowly and insidiously all around the margin of the residual analgesic and atrophic patch. There were no features of systemic toxicity. Slit-skin smear was negative for acid-fast bacilli and histology showed features of BT leprosy. Slow and insidious onset of the lesions at the sites previously affected by the disease, without development of either ulceration of the surface of the skin lesions or the constitutional features of systemic toxicity suggest that these two patients had relapse of the disease rather than having type I lepra reaction.

Although the number of patients studied here is only small, the persistence of clinical and histological evidences of leprosy in one-third of cases, even after multidrug therapy for 6 months and development of clinical and histological relapse in 2 cases suggest that either the regimen or the duration of treatment recommended by the WHO study group, for paucibacillary leprosy is quite inadequate. According to this regimen one has to stop chemotherapy at the end of 6 months. I do not understand the rationale for discontinuing the drugs at the end of 6 months even when the disease is active. In no other chronic bacterial disease is the antibacterial agent discontinued abruptly in the presence of active clinical signs and symptoms. Nothing is known about *Mycobacterium leprae* in paucibacillary lesions, after short course MDT, since foot-pad inoculation studies are not possible in such cases. A test is yet to develop to find whether the last organism in the lesion has been wiped out. One may argue that adequate cell-mediated immunity such patients possess may cause resolution of these 'residual' active lesions in the absence of further continuation of treatment. But this happens not only in partly treated cases but in the majority of indeterminate and tuberculoid cases without any treatment at all. But no one can predict in which patient spontaneous healing will take place or in which it will not.

According to the WHO regimen, after 6 months chemotherapy in paucibacillary leprosy, we are depending on Nature's mercy for further healing of the lesions. In the presence of availability of effective drugs for leprosy, why should the drug be discontinued abruptly even when the skin lesions are active, clinically and histologically. Whatever the type of leprosy or the number of bacilli in the lesions may be, the microorganism responsible for the leprosy in all patients is the same. Then why not the benefit of prolonged treatment as for bacilliferous cases or at least till the skin lesions become inactive be instituted in paucibacillary cases also? Two cases of 'relapse' noted in this study further support our view that the present regimen as recommended by WHO study group is quite

inadequate. We would like to hear from our professional colleagues about their experiences in treating paucibacillary cases of leprosy with this regimen.

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