same as the initial killing, at least during the course of treatment with RMP. In addition, unless one can demonstrate that 3 months of clofazimine can kill about 4 logs of organisms in a previously untreated lepromatous patient, it seems over-optimistic to assume that 3 months of daily RMP and clofazimine could be sufficient for the treatment as suggested by Dr Almeida.

Plenty of information has already demonstrated the seriousness of dapsone-resistant leprosy, and that its prevalence increases with time if dapsone monotherapy is continued. The later results from other areas were entirely consistent with the earlier assessments. The dapsone susceptibility of the M. leprae strains isolated from previously untreated patients had changed significantly: the primary dapsone-resistant M. leprae was virtually nonexistent before 1977; whereas the organisms from one-third to one-half of the MB patients diagnosed in the 1980s were resistant to dapsone. In addition, regular administration of dapsone in an adequate dosage could not effectively prevent the relapse, because after 20 years of supervised sulphone therapy, in which a majority of the patients had received the dapsone equivalent of at least 100 mg daily, the annual relapse rate still remained 1%. Therefore, it was clear that the attempts to control leprosy by dapsone monotherapy were failing, and the justification to further evaluate the 3-year dapsone monotherapy, as proposed by Dr Almeida, is weak.

References


COMMENT: ‘IMMUNOLOGICAL UPGRADING WITH COMBINED IMMUNOTHERAPY AND CHEMOTHERAPY IN A LEpromatous leprosy PATIENT: A CASE REPORT’

Sir,

The above article, which was published in Lepr Rev (1991), 62, 297–302, is intriguing. However, we feel that there are some points that should be discussed to avoid any possible misunderstandings.

Skin test conversion responses to killed vaccines prepared from cultivable mycobacteria, namely Mycobacterium avium (the ‘ICRC’ bacillus and Mycobacterium w) are well conceived,1,2 but have not been conclusively proved in experimental animals.3 Such a conversion may also occur through:
1. multidrug therapy (MDT); ^4, 5 2. repeated lepromin testing; ^6 3. spontaneous upgrading; and 4. injection of cytokines per se ^7, 8 or antigenic challenges that induce cytokine activation. More significantly, it has been documented that delayed-type hypersensitivity may not parallel specific immunity in mycobacterial infections. ^9, 10 Hypersensitivity and protective immunity may be directed towards separate mycobacterial antigens. ^11 These factors were not focused upon in the article. It is, therefore, imperative to enlarge the study, keeping in view the 3 groups, namely MDT alone, MDT and vaccine, and vaccine alone. Also these groups should be interchanged during trials. The findings in the present case should, therefore, be viewed with caution. We look forward with keen anticipation to the publication of the conclusive results of the study being undertaken by the authors.

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References

COMMENT: REVERSAL REACTIONS IN LEPROSY AND THEIR MANAGEMENT

Sir,

Dr Rose and Dr Waters in their Editorial (Lepr Rev, 1991; 62: 113–21) drew attention to the importance of the recognition of reversal reaction (RR) in the borderline group of leprosy and its management towards deformity prevention. I should like to make the following points on RR and its management.

The recognition of Type I, i.e. RR, downgrading reaction is a known clinical entity, more specifically observed in BB cases downgrading to BL and BL cases to LL cases. Such phenomenon is seen mostly in untreated BB cases, the 2nd and 3rd trimester of pregnancy, and in those with viral infections, which lower the cell-mediated immunity, are especially at risk. This RR downgrading is not strikingly symptomatic, but some signs are: oedema over the dorsal aspect of hand and foot; frequent appearance of new dermal lesions; and slow deterioration of the sensory and muscular