LETTERS TO THE EDITOR

COMMENT ON WHO REGIMEN FOR PAUCIBACILLARY LEPROSY

Dr. K. Pavithran in his letter to the editor titled, "WHO regimen for paucibacillary leprosy, recommended dosage duration inadequate?" IJDVL, 1985; 51 : 294, has inferred that "These observations indicate either the regimen recommended by WHO for paucibacillary leprosy is to be reconsidered or the duration of treatment recommended as six months is quite inadequate and needs further evaluation."

In this regard, we would like to comment that the inference drawn from the observation of ten patients made by the author is misleading, inadequate, and incorrect; and this is so because the inference is drawn from the clinical and histopathological evidence of persistence of the disease in the patients. However, this was expected in some cases even by the people who recommended the WHO regimen for the same. The WHO while recommending this regimen had taken the relapse rate as the crucial criteria for deciding the duration of treatment. Hence, the real deciding factor for commenting on WHO regimen and its duration is the relapse rate. The author did not either mention or consider the criteria of relapse rate in his study. We would suggest that in such case studies, the relapse rate should be considered before passing any adverse comments on the WHO regimen.

In this connection, I would like to mention two recent trials involving WHO regimen.

(1) Rose P : A short course MDT in Guyana, Leprosy Review, 1984; 55 : 143-147. It is mentioned under the discussion "Two hundred and forty six of the 303 patients under review completed treatment during 1982, and 57 during the first five months of 1983. It is certainly too soon for a comprehensive review of results but on the evidence available so far the WHO recommendations for the paucibacillary patients are effective. There have been neither relapses, nor reactions among three indeterminate (I) and 108 TT patients at risk and a relapse rate of 1% amongst the 192 BT patients is acceptable. However, these 192 patients include many who had already had some previous treatment and it is possible that the relapse rate may in future rise when all the patients are new patients."

Experience so far suggests that there is no need to fear that patients released from treatment will not report back on deterioration; on the contrary, patients frequently return for treatment of incidental skin complaints and for reassurance where macules, though fading, are still present. Perhaps one of the most important benefits to be expected from the new regimen is that the release of large numbers of satisfied patients, after such a relatively short period of treatment, will have a tremendously beneficial impact on case finding. "A further account of progress in the treatment of paucibacillary and also multibacillary cases will be published later."

It was noted later in the same trial that in paucibacillary patients the continued improvement after withdrawal of treatment has been particularly impressive.

(2) In Malwi, the field trial of short course of chemotherapy in paucibacillary leprosy cases commenced in May 1983, and 505 patients have been included in the study trial. The classification of leprosy was based on clinical findings and supported by slit skin smears. Acceptability and tolerance of the drugs have been excellent and the side effects minimal, attendance rates have been consistently high. Late reversal reactions occurred in three patients after the chemotherapy was discontinued. No case of relapse has yet been reported in the follow up period.

Hence, from the evidence available so far, the WHO recommendations for paucibacillary patients are effective. In conclusion, we feel that the inference drawn by the author was inadequate, incorrect and misleading.

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REPLY

Dear Sir,

Reference comments by Kiran et al on my letter to the editor regarding WHO regimen for paucibacillary leprosy, I do agree that the number of patients studied here is only small; this is the reason why this inference was communicated as a letter to the editor. Three of the 10 patients who received the short-course MDT for 6 months for paucibacillary leprosy, showed clinical activity of the disease and 2 others had histopathological evidence of tuberculoid granuloma. According to WHO regimen for paucibacillary leprosy, one has to stop the chemotherapy at the end of 6 months. This is the point that needs clarification. I am scared to stop chemotherapy abruptly when erythema and infiltration of the skin lesions and thickening and tenderness of the nerves persist. I do not understand the rationale for stopping the drugs at the end of 6 months even when the disease is active. In no other chronic bacterial infection, the antibacterial agent is stopped abruptly in the presence of active clinical signs and symptoms. Nothing is known about the *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 be developed to say whether the last organism in the lesion has been wiped out. One may argue that the adequate CMI that such patients possess may bring about resolution of the residual 'active' lesions in the absence of further continuation of treatment. But this happens not only for partly treated case but in majority of the indeterminate and tuberculoid cases without any treatment at all. But no one can predict in which patient spontaneous healing will take place and in which it will not.

According to the WHO regimen for paucibacillary cases, after 6 months of therapy, we are to depend on the nature's mercy for further healing of the lesion. In the presence of availability of effective drugs for leprosy, why should the drug be stopped when the lesions are clinically and histopathologically active. Whatever the type of leprosy or the number of bacilli in the lesion, the organism responsible for the disease in all patients is the same. Then why not provide the benefit of prolonged treatment as for bacilliferous cases or at least till the lesions become inactive, in paucibacillary cases also. I don't think it is easy to convince the patient that the residual active lesions he sees after 6 months therapy, will clear spontaneously. Are we justified in releasing them from control at this stage?

Dr. Kiran has stated that clinical and histopathological persistence of the disease at the end of treatment period, was expected by people who recommended the WHO regimen for paucibacillary leprosy. But in the technical series report 675 of WHO on chemotherapy of leprosy for control programmes 1982, nothing is mentioned about this. It is gratifying to see the recommendation by IAL, 1983 that for paucibacillary cases, pulse doses of rifampicin are to be given for a minimum of 6, at monthly intervals or till clinical inactivity occurs.[1] It is also interesting to note the recent report from Bombay, which shows that among 498 patients who had MDT for paucibacillary leprosy, at the end of 6 months 41% (202 patients) had still clinically active lesions, though they showed features of regression.[2]

I do not think that the relapse rate need be mentioned in my communication since the study

focussed on the observations at the end of 6 months of therapy. Only on further follow up of cases one can find out the relapse rate. In short, in the presence of clinical and histopathological evidence of persistence of activity of the disease even after the short course MDT, I don't think it is wise to stop the antileprosy drugs abruptly and leaving the disease process to the mercy of nature for further healing, when effective drugs are available against this disease.

References

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HYPERTRICHOSIS FOLLOWING PUVASOL— A POSSIBLE MECHANISM OF ACTION

The pathogenesis of hypertrichosis following PUVASOL therapy in vitiligo[1,2] remains unknown. The photoperiod (the duration of daylight) has been shown to have a strong influence on the moulting cycle in a number of animals.[3] It has been postulated that the increase and decrease in the photoperiod influences the moulting cycle through the eyes, the hypothalamus and the hypophysis, which then directly modifies the follicullar activity through the thyroid and adrenals, and indirectly through the gonads.[4] Ocular changes are anticipated in PUVA therepy where 8-MOP gets deposited in the lens. This binding followed by UV light may bring about alteration in the moulting cycle (due to alteration in the effective photoperiod) with resultant hypertrichosis. Studies on the effect of PUVASOL on moulting cycle in animals, anagen and telogen ratio before and after PUVA, assessment of hormonal levels in patients with hypertrichosis following PUVA may throw further light on the *modus operandi* of PUVA. However, hypertrichosis following topical PUVASOL is not explained by this mechanism.

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References

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