## COMMENT: CLOFAZIMINE-INDUCED LYMPHOEDEMA

Sir,

In the light of a previous observation reported on clofazimine-induced lymphoedema<sup>1</sup> we would like to report our observations on 75 patients who are being treated at the Leprosy Mission Hospital, Shahdhara, Delhi.

Seventy-five patients were seen at random from June to December 1990 as they presented to the regular outpatient clinic for their monthly check-up and antileprosy treatment. Seventy-two of them had been on multidrug therapy (MDT) for paucibacillary or multibacillary leprosy for some length of time.

The patients were aged between 10 and 55 years, with a mean age of 30 years for males and 29 years for females. The types of leprosy seen among these patients were as follows: of the 60 male patients 26 had lepromatous leprosy, 28 had borderline–lepromatous leprosy, 5 had borderline–tuberculoid leprosy and 1 had tuberculoid leprosy. Among the 15 female patients 4 were lepromatous, 8 were borderline–lepromatous and 3 were borderline–tuberculoid.

None of these patients had any cardiovascular, renal, hepatic, filarial or nutritional disease prior to or after commencement of MDT.

Forty-five of these patients had pedal oedema, which we have found to be varied in nature and of possibly different aetiology. On clinical assessment and on reviewing their histories, the following observations were made:

- 1 Of these patients, 9 had either plantar ulcers or some foot deformity on the same foot on which the oedema was observed. This could possibly imply that the oedema in these patients might have been aggravated by the plantar ulcer or foot deformity.
- 2 Pedal oedema in both feet before starting MDT was present in 16 patients. Four of these patients were definite that the oedema had subsided after the onset of therapy. This could possibly imply that the oedema in these patients was a consequence of the disease process itself. Two of the 16 patients developed oedema of the hands after MDT was started, though there was no evidence of either Type I or Type II reaction.
- 3 Two patients with plantar ulcers on one foot had developed oedema of the opposite foot after MDT was started. This could have been due to a compensation in the stance, trauma, or clofazimine.
- 4 History of recurrent ENL was given by 8 patients who had been on steroids. One of these patients had also been on long-term steroids for ulnar neuritis. There is a possibility that in these patients the pedal (peripheral) oedema could be due to steroids.
- 5 What interested us the most was that there were 10 patients at least who did not have any pedal oedema before MDT, but who subjectively and symptomatically complained of a development of oedema of the feet after the onset of therapy. One patient showed oedema more in one foot than the other, though there was no obvious pathology of the foot.

It appears to us that the pattern of pedal oedema in these 10 patients was similar to that of the patients reported earlier.<sup>1</sup> We would like to suggest that the pedal oedema in these 10 patients could be due to a lymphatic stasis produced by clofazimine in the lymphatic channels.

Clofazimine has been shown to be deposited in the lymph nodes by various histopathologists,<sup>2</sup> <sup>4</sup> and such deposition was considered to be responsible for abdominal pain.<sup>5</sup> One report also mentions the development of persistent ard generalized oedema in a patient who was given clofazimine 100 mg daily, with prednisolone 10 mg.<sup>6</sup>

We intend to follow up these patients who have been studied in a randomized and retrospective manner. Radiological and biochemical studies are also being planned. But we would certainly be interested in receiving comments from leprologists, pathologists, pharmacologists or others on the

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possibility of clofazimine-induced lymphoedema, and we would also like to know if similar reports or observations have been made in any other centre.

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## References

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