

Letters to the Editor

LEPROSY, ONCHOCERCIASIS, DIETHYLCARBAMAZINE AND THE MAZZOTTI REACTION

Sir,

Whilst working in the Southern Sudan (1979–80) in an area endemic for both leprosy and onchocerciasis, we had the opportunity to examine a large number of patients with onchocerciasis and borderline leprosy, particularly borderline-lepromatous; BL on the Ridley–Jopling scale of whom we noted adverse reactions closely following the administration of diethylcarbamazine (DEC), both for the Mazzotti test and for treatment, as noted in a personal communication by Emilia Odé from the Cameroon in 1976.¹ These included skin and nerve lesions suggestive of reversal (upgrading) reaction, and they responded to appropriate treatment with steroids or other anti-reaction drugs. In view of the very large number of factors which may precipitate reactions in borderline patients, and the difficulty of ascertaining the full medical history, including drug intake, it is difficult to be sure that the drug was the sole cause in all instances, but the association was strongly suggestive in many of our cases.

A matter of additional interest in relation to these two diseases and the use of DEC is that in a few patients with lepromatous leprosy, we observed a suppression of the Mazzotti reaction, similar to that described by Meyers and Connor in 1975² and an absence of killing of microfilariae. A typical patient was a 45-year-old male, on regular dapsone treatment 100 mg per day for 2 years, with microfilarial counts as follows: eye 8/mg; scapula 153/mg; iliac crest 230/mg; calf 363/mg; upper arm 164/mg and lower arm 65/mg. A slight concurrent infection with *D. perstans* was also found in the blood. There was no rise of blood eosinophils and the urine and stool were negative for parasites. There was one onchocercoma palpable at the waist only. A Mazzotti test with 50 mg DEC produced absolutely no effect, but 3 hours later we recorded a microfilaruria, 10 out of 28 forms being alive in the sediment. Treatment was started with 50 mg DEC daily, increasing to 6 × 50 mg daily within a period of one week. From day 10, 8 × 50 mg was given daily up to day 24. Several examinations of blood revealed only an unchanged *D. perstans* infection and there was no eosinophilia either during or after treatment. Skin biopsies were taken 5 days and 17 days after the start of DEC treatment and we are grateful to the Armed Forces Institute of Pathology, Washington DC for the following reports.

5 day biopsy ‘... The most spectacular feature is the large number of microfilariae of *O. volvulus* in the upper dermis. None of these is degenerating or centred in a focus of inflammatory infiltrates. It is surprising none is degenerating after 5 days of DEC therapy. Nor do we see any evidence of any microfilariae migrating into the epidermis. These findings, however, are consistent with your clinical observation that there was no Mazzotti reaction and no microfilaricidal treatment response. Changes of leprosy are present with some inflammatory changes of nerves, occasional acid-fast bacilli in nerves and clumps of acid-fast bacilli in the walls of dermal vessels.’

17 day biopsy ‘... There are many microfilariae in the upper dermis. None is degenerated or surrounded by inflammatory cells. The remarkable feature is the absence of a reaction to the diethylcarbamazine. The number and distribution of microfilariae suggests that the DEC has had no effect.’

It must be recorded that this absence of response on Mazzotti testing and on treatment with DEC was not invariable in our lepromatous patients, but this case and a few others suggest that the subject may be worth further study. A similar observation has recently been made on a large series of patients in Bamako, Mali,³ in which it was also observed that onchocerciasis was commoner in patients with leprosy than in the general population.

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References

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