Letter to the Editor

REVERSAL REACTION OCCURRING 16 YEARS AFTER BEGINNING ANTIBACTERIAL TREATMENT – COMMENT

I would like to make some comments on the interesting case report Dr Rea published in the March 2004 issue of Leprosy Review: ‘Reversal reaction occurring 16 years after beginning antibacterial treatment’.

There are several reports of type I reaction that occurred after the end of treatment. This patient was examined in 1984 with a borderline eruption that appeared 1 year before, with a BI of 5.5. The patient was treated from 1984 to 1986 with DDS 100 mg/daily and rifampin 600 mg/daily, thereafter with sulphone alone until October 2002. ENL appeared in 1985 and it lasted until 1999. During this period, there were difficulties in distinguishing ENL from reversal reaction on clinical grounds. In June 1985, BI was 5.2; in June 1986, BI was 4.6; in June 1991, BI was 1.8 and in 2001, during the reversal reaction, it was negative.

This case presents some uncommon aspects; however, I agree that it cannot be considered a unique syndrome, since too many have been encountered in the past.

The author thinks it unlikely that the bacilli started to multiply. I would like to argue that it is well known that nearly all multibacillary patients harbour persisters, even after a long period of treatment. During the time of dapsone monotherapy, patients, after decades still on treatment, showed suddenly signs of reactivity. In some, bacilli could then be detected. Those patients were then considered as possibly drug resistant, although some continued dapsone awaiting the results of mouse footpad culture. In some, the reactivity settled and the bacilli disappeared. A possible explanation was given at that time: ‘it was possibly persisters, that for whatever reason started to multiply’. They were killed by the immune system, which recovered after treatment during a low bacterial load. The released antigens of the killed bacilli then elicited a hypersensitivity reaction (reversal reaction).

Another explanation was that the bacilli were still dapsone sensitive and were killed by the dapsone. However, at the time that only chaulmoogra oil was available as treatment, some of the patients were also cured after a reaction, having been known to harbour bacilli before the reaction.

Therefore, I think that in this borderline patient, the CMI under treatment had recovered to some extent. When persisters started to multiply, the patient reacted and was able to deal with these bacilli. This is confirmed by the fact that no bacilli could be found by the methods used. What is wrong with this ‘old’ explanation? One should not be too dogmatic with the current explanations of a RR.

I strongly feel that the patient relapsed, but was able to deal with the relapse herself.

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References