

DDS 100 mg Daily, the Cornerstone in the Management of Reversal Reaction

TO THE EDITOR:

A retrospective study was performed to prove our hypothesis that the improvement in treatment response reported in the article "Reversal reaction, the prevention of permanent nerve damage. Comparison of short and long-term steroid treatment" (1) was not due solely to longer steroid treatment but mainly to the increase of the dapsone (DDS) dosage. Our hypothesis is based on

the fact that in the above mentioned article the patients with the better treatment response received not only longer steroids but also DDS 100 mg daily, whereas the patients with the poor treatment response received DDS 200 mg weekly or less besides the short-term steroids (2). Our retrospective study was designed to find borderline leprosy patients in reversal reaction with nerve damage who had been treated for

some time with DDS 100 mg daily only. The diagnosis of reversal reaction was made according to criteria described in the already mentioned article, i.e., raised swollen skin lesions, tender nerves, or evidence suggesting recent deterioration in nerve function.

Testing of voluntary muscle power with the voluntary muscle test (VMT) was used for the assessment of nerve function (3), and nerve damage was expressed as a VMT deficit. However, compared with the previous study, a modification in the scoring of the VMT deficit had to be made because not all six muscle groups were tested for the function of the ulnar and median nerve in every patient. Sometimes only the abductor digiti minimi muscle, the first dorsal interosseus muscle, and the abductor pollicis brevis muscle were tested. So to make comparison possible it was decided to express improvement or deterioration of the VMT deficit, not as an absolute score but as a percentage of the average initial VMT deficit of the three muscles tested.

In the period studied, 1974–1980, 23 case histories were found of patients in reversal reaction with an impressive improvement of their VMT deficit when they did not receive any prednisone. The reasons for the patients not receiving prednisone were: refusal of the patient to start prednisone treatment as an inpatient, medical contraindications for prednisone, or deterioration of nerve function under previous prednisone treatment.

Thirteen of these patients never received prednisone. These patients were followed up for periods varying from 2 to 15 months. Their average VMT deficit showed an improvement of 58%. When one adds this result to the results given in the former study, three different treatment schedules can be compared. DDS 200 or less mg weekly plus short term steroid treatment showed no improvement after 6 months and 32% improvement after 12 months. DDS 100 mg daily without steroid treatment gave 58% improvement after a mean period of 7 months. DDS 100 mg daily plus long-term steroid treatment showed 60% improvement after 6 months and 75% after 12

months. One particular patient of our 13 patients is a very good example of the importance of DDS 100 mg daily in the prevention of permanent nerve damage. He was treated for about 3 years with DDS 5 mg daily and showed gradual deterioration of the VMT deficit. Increase of the DDS dosage to 100 mg daily resulted in a complete disappearance of the VMT deficit. These findings show clearly that the prevention of permanent nerve damage is not the value of long-term steroid treatment but due mainly to increase of the DDS dosage.

Another argument in favor of our hypothesis that the value of long term prednisone treatment is not proven is the case histories of the remaining 10 patients. These, all borderline lepromatous leprosy patients, were treated with corticosteroids because of neural damage for a mean period of 17 months. In that period their VMT deficit deteriorated by 144%. After the prednisone was withdrawn but DDS 100 mg daily was continued, their VMT deficit dramatically improved. Ten months later the VMT deficit was even 39% less than before the corticosteroids were started.

These 23 case histories clearly illustrate that a controlled trial is needed to prove the value of long-term steroid treatment in reversal reaction.

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