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The outpatient treatment of nerve damage in patients with borderline leprosy using a semi-standardized steroid regimen

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Summary Thirty-three patients with borderline leprosy who had developed recent (less than 6 months duration) loss of nerve function were treated with a semi-standardized course of corticosteroids, the average initial dose was 25 mg prednisolone daily, and the average duration was 5 months. Treatment was unsupervised and no patient was admitted to hospital. The results were assessed by tests of voluntary muscle power and of sensory function, of the 57 nerves studied, 38 showed marked improvement and none got worse. There were no serious side-effects. Patients were taught exercises to prevent deformity, and residual muscle weakness did not progress to contractures. Corticosteroid treatment is safe enough, and confers sufficient benefit to be used in standard dosage under field conditions.

It is common experience that a patient will present himself for treatment because of recent nerve damage (motor or sensory) or signs of incipient nerve damage ('aches and pains' or paraesthesiae), commonly of only a few months duration. In such cases there is a good prospect of improvement if effective treatment for neuritis is instituted promptly. This is even more true of those patients who develop nerve damage during the first years of chemotherapy.

In patients with borderline leprosy the nerve damage is caused by the cell mediated immune response to antigens of *Mycobacterium leprae*, and many patients with recent nerve damage show signs of actual or incipient Type 1 lepra reaction (reversal reaction) in their skin lesions. The natural history of this reaction (rapid onset, gradual subsidence over a period of months) suggests a logical pattern of steroid treatment.[1] But no 'standard course' has yet won general acceptance.

It is often difficult to treat patients with nerve damage under field conditions, indeed, there is a tendency to insist on hospitalization for steroid treatment. But patients will probably refuse unless they have severe painful neuritis. Moreover

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there are often few beds available and no effective referral system. If the beds are in a general medical unit, hospital staff will usually have little knowledge or interest in the management of leprosy neuritis.

It is not surprising that field staff, unauthorized to give effective treatment for neuritis and often unable to refer patients for such treatment, may consider it unimportant to look for signs of nerve damage. In this situation it would be helpful if there was known to be a standard course of corticosteroids, which was effective in improving most patients, and was seldom harmful when used under field conditions. This paper reports the results of an out-patient study using a semi-standardized course of prednisolone to treat patients with recent nerve damage. We hope it will contribute towards defining a standard course for field use.

Patients and methods

The study included all borderline leprosy patients registered during 1982 for treatment in Dhoolpet Leprosy Research Centre, Hyderabad, who had, by their history, developed signs of nerve damage within the previous 6 months. About half of them had received some previous treatment for leprosy, the rest were untreated.

INITIAL ASSESSMENT

This included clinical examination of the skin lesions and palpation of nerves. Slit skin smears for acid fast bacilli were taken in all cases, and skin biopsy to confirm the clinical classification in about half the patients.

Nerve damage was assessed by tests of voluntary muscle power (VMT) of muscles supplied by the facial, ulnar, median and lateral popliteal nerves.[2] Sensory tests (ST) in areas supplied by affected nerves were performed using graded nylon bristles;[3] tests for protective sensation (indentation of the skin by a ball-point pen tip) were also undertaken.

The VMT results for the ulnar nerve were scored by adding the figures (0–5 scale) for the 2 muscles tested, which were abductor digiti minimi and 1st dorsal interosseous. Other nerves, where only one muscle or group was tested, were scored by doubling the VMT figure. Thus the scores for all nerves could be directly compared.

TREATMENT

All patients received dapsone 50–100 mg daily as anti-leprosy chemotherapy. Treatment for neuritis was with prednisolone. The average initial dose was 25 mg daily; this was normally reduced by 5 mg daily per month. However, dosage was

adjusted for body weight, and also for severity of neuritis (the more severe the neuritis the higher the initial steroid dosage). Patients were advised to take the full daily dosage of both dapsone and prednisolone as a single morning dose. All treatment was unsupervised, on an out-patient basis.

HEALTH EDUCATION

This was undertaken by the doctor who saw the patient, and occupied 30 per cent or more of an average consultation. Points covered included:

Appropriate active and passive exercises for affected muscles. It was emphasised that treatment might prevent permanent weakness and sensory loss, but exercises were needed to strengthen muscles and prevent stiffness and contractures.

2 Education on the risks of anaesthesia (if it was present) and the principles of hand and foot care.

3 Encouragement to take tablets regularly, and warning that prednisolone was dangerous if not taken according to instructions.

ASSESSMENTS DURING TREATMENT

Most patients were seen every 1–2 months during the period of steroid treatment, and every 2–6 months thereafter. Routine examination included palpation for nerve tenderness, and VMT and ST to assess the degree of improvement. Patients were asked to demonstrate how they did their exercises at home. Note was made of any symptoms that might be due to drug toxicity. Health education was continued according to the patients needs.

In most patients the steroid dosage was reduced month by month. However, if there was persistent or recurrent nerve pain or tenderness, or if function deteriorated, the steroid dosage was prolonged and/or temporarily increased.

Results

Forty-five patients were included in the trial, of whom 33 (classified clinically as BT-24, BB-2, BL-7) completed their steroid treatment and were available for follow up. Some had more than one affected nerve; the number of damaged nerves was 57 (BT-37, BB-5, BL-15). In about 80% of cases the final follow up assessment was more than 6 months after steroid treatment had been discontinued (Table 1).

A 'good' end result was defined as VMT power of 4 or 5 (i.e., a nerve score of 8–10). Table 2 shows the initial steroid dosage, number of patients, and results of treatment. About three-quarters of the nerves showed a good result. The degree

Table 1. Duration of follow-up period from start of treatment; 33 trial patients

Duration of follow-up (months)	Number of patients
6–9	3 (9%)
10–12	4 (12%)
13 or more	26 (79%)

Table 2. Initial dosage of prednisolone and end result of treatment of 33 patients (57 nerves)

Initial dosage of prednisolone (mg per day)	Number of patients	Number of nerves	
		Good result	Bad result
30	13	16	8
25	7	10	2
20	11	13	5
15	2	3	0
Total	33	42 (74%)	15 (26%)

of improvement (difference between initial and final scores) was 6 or more in two-thirds of the nerves (Table 3); none of them got worse. BB and BL nerves did as well as BT nerves.

The sensory status at follow up is shown in Table 4. It was normal or near normal in half the patients; only a quarter of them had lost protective sensation.

There were few toxic effects which could be attributed to the treatment. A few patients complained of epigastric pain which responded to antacids and reduced spice in the diet. A few developed infections of the hands or feet which were controlled by antibiotics. None developed signs of progressive tuberculosis, severe intestinal parasite infestation, diabetes or hypertension. The course was too short for osteoporosis to be a problem, and no patients complained of symptoms of adrenal insufficiency on stopping steroid treatment.

Table 3. Initial dosage of prednisolone and degree of improvement of 57 nerves

Initial prednisolone dosage (mg per day)	Number of nerves showing improvement in nerve score of			
	0–2	3–5	6–9	Total
30	7	1	16	24
25	2	2	8	12
20	4	3	11	18
15	0	0	3	3
Total	13 (23%)	6 (10%)	38 (67%)	57

Table 4. Results of sensory testing of 57 nerves

Sensory status	Number of nerves
Anaesthesia	15 (27%)
Protective sensation present	13 (23%)
Light touch absent	
Light touch felt (sensation normal or mildly impaired)	29 (50%)

Discussion

Nerve damage is not the only cause of deformity. Failure to do simple exercises leads to unnecessary stiffness and contracture formation; failure of reasonable hand and foot care will allow injuries and infections to cause further tissue loss and scarring. But the deformities which are commonly seen in patients who have been diagnosed reasonably early and treated (but without steroids) reasonably regularly indicate the need for more intensive measures to recognize and treat recent nerve damage as quickly as possible.

Not all improvement of function is attributable to recovery of nerve function. Motor units of a partially denervated muscle can hypertrophy, and sensory damage can to some extent recover by filling in from neighbouring nerve territories. Thus the management of a patient with nerve damage is not just a matter of prescribing corticosteroids; health education, centred round the need for regular exercises and hand and foot care is of comparable importance.

Nevertheless, steroids offer an increased prospect of reversing nerve damage and so obviating the need for a lifetime of burdensome exercises and precautions.

However, steroids are dangerous if misused, and in any case are only part of the overall management of neuritis (albeit the part which makes the rest really worthwhile). There is scope for discussion of what grades of worker could be authorized to use steroids. But effective treatment of nerve damage depends on steroids being available to use in defined courses by workers who have regular patient contact.

The present study was based on a 'city centre' clinic, and patients, though managed as out-patients, were always seen by a doctor. We did not aim to demonstrate the field use of steroids. But the study has shown 3 important preliminary points:

In the dosage we employed, steroids were safe for out-patient use. We took no special steps, for instance, to exclude tuberculosis in patients who looked well and had no cough. We did not advise patients to avoid work, even if it was manual and involved the risk of injuries and infections. Patients were warned that the tablets were dangerous in high dosage; we seldom found them taking too many, and were not pestered to continue prescribing them after the end of the course of treatment. On the other hand, the few patients who developed recurrent painful neuritis knew that they would be prescribed additional dosage to control their symptoms, and would not have to try to buy extra tablets for themselves elsewhere.

2 In the dosage we employed, steroids were effective. Most patients showed improvement, and most ended up with useful hands and feet. Even those with persistent weakness usually did not develop contracture deformities, and so avoided the stigma of being obvious 'lepers'. Although much of the benefit must be attributed to the steroid treatment, the health education, which was an integral part of the management, played an important role in its success.

3 Our patients were not angels. They were sometimes late for appointments, and no doubt sometimes forgot to take their tablets. Results such as ours can reasonably be expected in out-patients with unsupervised treatment.

The results of a similar study[4] have been reported.

This used a longer course of prednisolone, starting at higher dosage (40 mg daily for 2 weeks, 30 mg daily for 2 weeks, then 25–20–15–10–5 mg daily, reducing the dosage monthly. The whole course lasted for 6 months, and patients were admitted to hospital for months 1 and 2. The results of their study were much the same as ours (see Table 5, where the two studies are compared using the scoring system[4] for VMT's). This suggests that the results of both studies are about the best that can be obtained, and that lower dosage than we used, which would be more suitable for the field use of steroids, might still give worthwhile benefits.

We did not use a rigid dosage schedule in this study, and more work will be needed to define a standard course. The patients attending Dhoolpet Leprosy Research Centre are self-selected, and probably have more severe disease, and so

Table 5. Number of treated nerves and VMT results of nerves that improved, comparing results of Touw *et al.*[4] with those of the present study

	Touw *et al.*[4]	Kiran *et al.*
Ulnar nerves		
Number	53	34
Number improved	32 (60%)	23 (68%)
Average initial VMT score*	18	26
Average final VMT score*	42	46
Median nerves		
Number	40	10
Number improved	27 (67%)	10 (100%)
Average initial VMT score*	36	26
Average final VMT score*	48	48

* Only nerves which showed improvement are included in these groups. VMT score is as in Touw *et al.*[4]

probably risk more severe and prolonged neuritis, than patients in a normal field programme. It is therefore probable that a standard dosage schedule will start at a lower dosage than we usually used. But if the treatment is to do more good than harm the dosage must be high enough, and the course long enough, to relieve the symptoms of neuritis in most cases. A course which commonly fails to do this is unlikely to do any good at all.

Further studies are needed to define a course of steroids for field use which adequately balances benefits and toxicity. But in the meantime consideration should be given to the training needs of those who use steroids to treat neuritis. We suggest the following. The doctor or field worker using steroids to treat neuritis must know:

1 How to diagnose borderline leprosy (BT to BL on the Ridley–Jopling scale) and distinguish it from lepromatous.
2 How to palpate nerves and recognize when they are enlarged and/or tender.
3 How to test for protective sensation of the hands and feet.
4 How to do VMT's of the abductor digiti minimi, abductor pollicis brevis, and dorsiflexors of the foot.
5 How to teach hand and foot care, particularly the treatment of minor injuries and recognition of infection at an early stage.
6 How to teach maintenance exercises for the hands, particularly to prevent finger and thumb web contractures and strengthen the extensor muscles of the fingers.
7 The symptoms and signs of damage to nerves commonly damaged in leprosy,

including sensory loss, and how to obtain accurate information about them from the patient.

8 The standard treatment regimen used to treat patients with evidence of recent nerve damage.

9 The signs of steroid toxicity and how to treat patients who develop them.

10 How to recognize inadequate response to treatment.

11 How and where to refer patients in whom treatment appears to be ineffective.

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