Dapsone-Induced Motor Polyneuropathy in a Patient with Leprosy

TO THE EDITOR:

Dapsone neuropathy is not a common occurrence in spite of the widespread use of this drug for the treatment of a variety of unrelated disorders. It was first reported by Saqueton, *et al.* ([4]) in a patient with pyoderma gangrenosum treated with dapsone 400 mg daily. Most other cases have been reported in patients with chronic skin diseases, such as dermatitis herpetiformis ([2]), subcorneal pustular dermatosis ([1]) and herpes gestationis ([5]), treated with high doses of dapsone. It has not been reported in leprosy cases ([3]). With a dapsone dose of 100 mg daily as given for treatment of leprosy, neuropathy is unlikely to occur, and even if it develops may remain unrecognized in the presence of the associated neuropathy caused by leprosy itself.

A 22-year-old male developed an asymptomatic, hypopigmented, dry, anesthetic, partly ill-defined bordered plaque of 3×2 cm on the lower left leg. There was no nerve thickening. Slit-skin smears from the patch and earlobe smears did not show acid-fast bacilli (AFB). A histopathological study of the biopsy specimen from the plaque revealed tuberculoid granuloma in the upper and mid-dermis. The epidermis was not eroded by the granuloma. Blood hemoglobin was 13 gm%, total leukocyte count 7000/cmm, and differential count neutrophils 60%, lymphocytes 34%, and eosinophils 6%. ESR was 12 mm/1st hr (Westegren). Blood sugar fasting level was 80 mg% and postprandial 140 mg%. The liver function and renal function tests were within normal limits.

A diagnosis of borderline turberculoid Hansen's disease was made and the patient was prescribed dapsone 100 mg daily and rifampin 600 mg once a month. Two months later while on multidrug therapy

(MDT), the patient reported with a tingling sensation and progressive weakness of the muscles of both hands and feet of 2 weeks' duration. Type 1 reaction was suspected and he was admitted in the ward and started on prednisolone 40 mg daily in addition to daily dapsone and once-monthly rifampin. In spite of having gross motor involvement, sensations of the extremities were normal and there was no associated thickening or tenderness of the nerves or signs of inflammation on the leprosy plaque. On further interrogation it was evident that the patient had been taking 100 mg dapsone 3× day as wrongly advised by a pharmacist.

A neurological examination showed wasting of the small muscles of both hands and feet. The hand grip was weak. Around the ankle he had only grade IV power. There was bilateral foot drop. All of the tendon reflexes on the upper limbs were absent, while on the lower limbs the knee jerks were just elicitable and the ankle jerks were absent. All sensations were normal. Rhomberg's test was negative. A nerve conduction study revealed diminished amplitude of compound muscle action potential with normal conduction velocity. Electromyography was suggestive of neurogenic atrophy. Sensory conduction studies did not show any abnormality.

A diagnosis of dapsone-induced motor neuropathy was made and both dapsone and rifampin were withdrawn. Prednisolone was gradually tapered off. Ten weeks after stopping dapsone, he recovered completely from his palsies. For leprosy he has now been treated for 4 months with clofazimine 100 mg daily, and the plaque is regressing well.

When a patient with leprosy develops palsies of the extremities while on treatment, one would usually consider the possibility of associated type 1 reaction. This made us start him on high-dose corticosteroids. But the absence of tenderness of the nerves, the absence of inflammatory signs of the leprosy plaque, and noninvolvement of the sensory system in spite of gross motor affection of the extremities made us suspect causes other than leprosy reaction for his neuritis. A history of taking dapsone in a higher dose gave a clue about the diagnosis of dapsone-induced motor polyneuropathy in our case. In demyelinating neuropathies, nerve conduction is markedly delayed, while in axonal degeneration the conduction is normal but the amplitude of evoked muscle action potential is reduced. The diminished amplitude of the compound muscle action potential with normal conduction velocity and the electromyographic evidence of neurogenic atrophy in our patient suggested an axonal type of neuropathy. The exact mechanism of dapsone neuropathy is not known. It produces a potentially reversible toxic polyneuropathy with its primary effect on the soma and axons of motor neurons. A dying back of motor neurons has been postulated ([2]). The patient recovered completely from his palsies on stopping dapsone. Whether the short course of corticosteroids he received with a wrong diagnosis of type 1 reaction had any influence on his rapid recovery from neuropathy is not known.

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REFERENCES

1. Fredericks, E. J., Kugelman, T. P. and Kirsch, N. Dapsone-induced motor polyneuropathy: a complication of prolonged treatment of subcorneal pustular dermatosis. Arch. Dermatol. **112** (1976) 1158–1160.
2. Gutman, L., Martin, J. D. and Welton, W. Dapsone polyneuropathy an axonal disease. Neurology **26** (1976) 514–516.
3. Jacobson, R. R. Treatment. In: *Leprosy.* Hastings, R. C., ed. Edinburgh: Churchill Livingstone, 1985, pp. 193–222.
4. Saqueton, A. C., Lorincz, A. L., Vick, N. A. and Hamer, R. D. Dapsone and peripheral neuropathy. Arch. Dermatol. **100** (1969) 214–217.
5. Wyatt, E. H. and Stevens, C. Dapsone induced peripheral neuropathy. Br. J. Dermatol. **86** (1972) 521–523.