Motor polyneuropathy and nystagmus associated with chloroquine phosphate

J. S. MARKS
B.M., B.Ch., M.R.C.P.

Department of Rheumatology, Wrightington Hospital, Wigan

Summary

A 49-year-old woman with rheumatoid arthritis developed a motor polyneuropathy and nystagmus after 5 months' treatment with chloroquine phosphate. The peripheral neuropathy resolved after the chloroquine was discontinued.

Introduction

Neuromyopathy due to chloroquine phosphate has been previously described (Loftus, 1963) and most reports have stressed the importance of myopathic features (Hughes, *et al.*, 1971). Ocular complications, particularly retinopathy, are well recognized (Scherbel *et al.*, 1965), but the occurrence of nystagmus has not been described. This paper reports the development of a motor polyneuropathy associated with nystagmus during treatment with chloroquine phosphate.

Case report

A 49-year-old woman with a 6-month history of classical sero-positive rheumatoid arthritis was started on chloroquine phosphate 250 mg daily after failing to respond adequately to ibuprofen 600 mg daily. Four months later she developed painless weakness of the legs without pyrexia which progressed over 10 days to complete paralysis. Examination showed lateral nystagmus but no other cranial deficits. There was slight weakness of triceps, wrist and hands and a flaccid paralysis of the legs. Sensory testing was normal but deep tendon reflexes were absent and the plantar responses were flexor. The patient's general condition was good apart from very mild inflammatory polyarthritis not associated with vasculitis or rheumatoid nodules.

Investigations showed ESR 33 mm in the first hour; Hb 13.2 g/dl and WBC $6.3 \times 10^9/l$ ($6300/mm^3$). Biochemical tests including muscle enzymes were normal. Tests for urinary porphyrins were negative. CSF contained 1.3 g/l (130 mg/100 ml) protein and

no cells. Virus cultures on CSF and stool samples and viral serological tests were negative. Tests for rheumatoid factor were positive but the anti-nuclear factor test was negative. Electrodiagnostic studies showed evidence of denervation and a slight reduction in motor conduction velocity in the popliteal nerve at 37.5 m/sec.

The chloroquine phosphate was discontinued after 18 weeks and replaced with prednisolone 60 mg daily. Recovery of muscle power was noted after one month. After 2 months the nystagmus was less, she had normal power in her arms and was walking well with a frame and the tendon reflexes had reappeared in her arms.

Discussion

This patient had a motor polyneuropathy with slight elevation of CSF protein. The acute onset and clinical features were in keeping with the Guillain-Barré syndrome but the presence of nystagmus suggested a 'toxic' cause for the neuropathy and the improvement that followed the withdrawal of chloroquine indicates that this drug might have been responsible. Electromyography showed a reduced interference pattern and fibrillation potentials in foot muscles but motor nerve conduction velocity was little affected, suggesting that chloroquine induces axonal degeneration rather than segmental demyelination. Peripheral neuropathy should be recognized as one of the rare hazards associated with chloroquine therapy.

References

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