Myasthenia gravis aggravated by pyrantel pamoate

Pyrantel pamoate is an antihelmintic agent used worldwide. We report worsening of myasthenia gravis by pyrantel pamoate in one patient.

In October 1989 a 72 year old diabetic man noticed mild bilateral palpebral ptosis when reading. He was blind in the right eye from diabetic retinopathy. In mid November of the same year, he complained of diarrhoea. A stool specimen was positive for Ascaris lumbricoides and a single 1000 mg dose of pyrantel pamoate was taken orally with breakfast on 26 November. Several hours later he became fatigued when chewing and also when walking. The following week he could not chew meat and bread and he noticed dysphonia. Neurological examination on 4 December showed bilateral palpebral ptosis with fatigability, limitation of abduction of both eyes and limitation of adduction of the right globe, hypophonia, weakness of neck extension and weakness of abduction of both arms.

Administration of 2 mg of edrophonium chloride reversed the ophthalmoparesis. Electrophysiological study revealed a normal area of compound muscle action potentials in the right abductor digiti quinti muscle both at rest and after 15 seconds of maximal voluntary effort. Supramaximal repetitive stimulation of the right ulnar nerve at 3 Hz and 30 Hz were within normal limits.

Stimulation single fibre electromyography of the right extensor digitorum communis muscle showed a mean jitter of 40 μs (upper normal limit 25 μs) at 50% of insertions showing increased jitter and no blockings were found. Haematocutaneous and biochemical tests were normal. Antinuclear antibodies were positive at a 1/540 title with an homogeneous pattern. Anti-DNA antibodies were negative. Antismooth muscle and antimyot cholesterol antibodies were positive at a 1/80 title. CT of the thorax was normal. Antiacetylcholine receptor antibodies were not determined.

He was treated with 180 mg/day of pyridostigmine bromide without alleviation of his symptoms. There was no improvement in spite of increasing the daily dose of pyridostigmine and muscarinic symptoms appeared. Prednisone 60 mg/day was started. Two weeks later he could tolerate solid food. Dysphonia, weakness of neck extension and weakness of the extremities disappeared, but ophthalmoparesis persisted. From February 1990 he was free of symptoms. There was no effect on the knee on the same side. Myelogram showed complete block at spinal level lumbar 2. The second case was a 44 year old male who had experienced low back pain for three years. There was neither a sensory deficit nor a motor weakness along the lower part of the back. Myelogram revealed a block at spinal level thoracic 12.

Macroscopically these were large encapsulated tumours. The myxopapillary ependymoma in case 1 arose from the first sacral root and in case 2 it arose from the second lumbar root. Treatment for both cases was excision of tumour and resection of the nerve root. Histologically, the tumours were discrete, rarely, in subcutaneous sacrococcygeal tissues;14 the lateral ventricle;12 and the cervico-thoracic cord.1 The pathogenesis for myxopapillary ependymomas arising in such a manner is less clear.

The first case was a 48 year old male with a one month history of low back ache and left leg sciatica with dragging of his left foot. There was weakness of the left ankle dorsiflexion, elevation of the left foot while he sat on the own on the same side. Myelogram showed complete block at spinal level lumbar 2.
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