Myasthenia gravis aggravated by pyrantel pamoate

Pyrantel pamoate is an antihelmintic agent used worldwide. 1 We report worsened myasthenia gravis by pyrantel pamoate in one patient.

In October 1989 a 72 year old diabetic man noticed mild bilateral palpebral ptosis when reading or washing. 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 dyspnoea. 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 ophthalmaparesis. Electrophysiological study revealed a normal area of compound muscle action potentials in the right abductor digitii 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. 25/50 of insertions showing increased jitter and no blockings were found. Haematological and biochemical tests were normal. Antinuclear antibodies were positive at a 1:510 titre with an homogenous pattern. Anti-gastric and -intestinal antibloody were negative. Antismooth muscle and antitimochondrial antibodies were positive at a 1/80 titre. CT of the thorax was normal. Antiactacholin 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 mussartinic symptoms appeared. Prednisone administration was started. Two weeks later he could tolerate solid food. Dyspnoea, weakness of neck extension and weakness of the extremities disappeared, but ophthalmaparesis persisted. From February 1990 he was treated with 70 mg every other day, without neuromuscular symptoms apart from ptosis and bilateral ophthalmaparesis.

We feel this is unique; this patient, ingestion of pyrantel pamoate aggravated a previously existing myasthenia gravis. Pyrantel exerts its antihelminthic action by blocking the worm’s neuromuscular transmission, producing a depolarising-type neuromuscular block. 11 In rabbits, parenteral administration leads to paralysis and death, 7 but toxic neuromuscular effects in humans have not been reported to date to our knowledge.

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Myxopapillary ependymomas arising from nerve roots of the spinal cord

Conventional pathogenesis suggests that myxopapillary ependymomas arise from ependymal cells lying adjacent to fibrous tissue. The large majority of myxopapillary ependymomas arise from the filum terminale where this arrangement applies. We report two cases of myxopapillary ependymomas arising directly from ependymal cells lying against the spinal roots. Ependymomas form 2–6% of all gliomas. 2 Myxopapillary ependymomas belong to a distinctive subgroup of ependymoma. They are virtually restricted to the cauda equina and are thought to originate from the filum terminale and conus medullaris. Myxopapillary ependymomas form 16% 9/53 to (21%) 8/37 of intraspinal tumours (six and two respectively). The myxopapillary antihelmintic and fibrous histology has led to the belief that the pathogenesis of myxopapillary ependymoma is related to ependymal cells lying against fibrous tissue mainly the filum terminale. 5

Ependymomas are 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, preservation of the left ankle plantar flexion and 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 no history of low back pain. 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. Myxopapillary ependymomas are known to occur in sympathetic ganglia and highly vascular tumours. There was a compact arrangement of ependymal cells and fibrous tissue around cores of hyaline acellular connective tissue rich in blood vessels.

The presentation of myxopapillary tumours is similar in many ways to myxopapillary tumours arising from the filum terminale or the conus medullaris, namely low back pain with myelographic block. The orthodox pathogenesis for myxopapillary ependymomas is from ependymal cells lying against fibrous tissue for example the filum terminal. Other proposed theories include myxopapillary ependymomas arising from ependymal rests in extra dural locations such as the sacrococcygeal region. 10 It has been observed 11 that ependymal rests occur in normal children, and occur where myxopapillary ependymomas arise in the same region, namely the dermis–subcutaneous junction. It may be that myxopapillary ependymomas arise in abnormal sites as a result of heterotopia, but heterotopia of tissue of the central nervous system in sites other than nasal is open to doubt. 12 Others 11 have suggested that the presence of ependymal cells against fibrous tissue is not essential for the formation of myxopapillary ependymomas, but a change seen in myxopapillary ependymomas. They suggest that this myxoid change is the result of anoxia due to vascular changes. A Giemsa stain, however, is the primary cause of myxopapillary ependymomas or the changes seen within them.
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