LETTERS TO THE EDITOR

Recurrent spontaneous accessory neuropathy

Isolated spinal accessory nerve palsy of obscure or spontaneous origin is an uncommon but well recognised entity.1 We describe the first reported instance of recurrent spontaneous accessory palsy.

At the age of 23 the patient developed an aching discomfort in the right shoulder and neck. This resolved after a few days but after several weeks he became aware of weakness of shoulder elevation. Examination revealed weakness of the right trapezius muscle. Nerve conduction studies of the accessory nerve, recorded in the trapezius, revealed latencies of 4.3 ms on the right and 2.9 ms on the left (normal < 3.2 ms). Electromyography showed large motor unit potentials and reduced recruitment in the right trapezius only. After four to five months the condition improved and the size and strength of the right shoulder returned to normal.

He remained well and continued to work as a policeman until he was 31, when he again developed an ache in the right shoulder after exercising with five-pound dumb-bells. The ache persisted for two to three weeks but was never severe and did not interfere with his sleep. Naproxen provided some relief. About three weeks after the onset of pain, the patient began to notice weakness of his right shoulder and weakness of shoulder adduction. Two weeks later the pain had entirely resolved but he was now aware of the right shoulder hanging lower than the left. He had no sense of loss or pain other than in the shoulder. The other limbs and spinal function remained normal.

Examination revealed a fit man with marked wasting of the right trapezius muscle and moderate weakness of shoulder elevation. There was right scapular winging of the trapezius type, which was present with the arm at rest and accentuated by lateral elevation of the arm. The right sternomastoid muscle was normal. No wasting or weakness was observed in the other right shoulder girdle muscles and the remaining cranial nerves and other limbs were normal. General examination was normal and there was no sign of injury or abnormality on the lateral side of the neck.

Cervical radiographs and myelography of the foramen magnum were normal. CT scan of the neck and skull base revealed no abnormality. The cerebrospinal fluid (CSF) was clear and colourless, with four lymphocytes and a protein of 0.34 g/l. Serum glucose, full blood count, sedimentation rate, C-reactive protein, antinuclear factor, and routine biochemistry were normal.

Nerve conduction studies of the accessory nerve, recording trapezius, revealed a considerable decreased amplitude of the compound muscle action potential on the right, but the distal latencies were similar and normal on both sides (right = 3.2 ms, left = 3.1 ms). Electromyography revealed fibulation potentials and large motor unit potentials with reduced recruitment in the right trapezius, and normal findings in the serratus anterior, infraspinatus, deltoid, biceps, trachialis and extensor digitorum communis.

Our patient experienced two similar episodes, separated by eight years, of shoulder ache followed by right trapezius weakness and wasting. On both occasions and electromyography examination found no abnormality except in the right trapezius, implying the presence of a lesion affecting the accessory nerve distal to the innervation of the sternomastoid. He appeared to make a complete recovery after the first episode, only to suffer a recurrence eight years later. Investigation has not shown an underlying cause and we therefore believe he is the first reported instance of a recurrent accessory nerve palsy.2

Due to its complicated course, the accessory nerve may be damaged by a wide range of processes affecting the cervical spinal cord, cervical spinal canal, foramen and skull base, carotid sheath, or posterior triangle of the neck. The nerve is particularly vulnerable as it crosses the posterior triangle, where injury occurs most often following radical neck dissection, resulting in weakness and wasting of the trapezius.

Spillane described three patients in 1949 who had medical evidence of isolated accessory neuropathies, the cause of which was obscure.3 To make a complete recovery after the second episode coincided with light exercise with weights, these were small (5 pounds) and seem unlikely to have produced a stretch injury of the accessory nerve.4

Pesticide toxicity and motor neuron disease

Pyrethrins are neurotoxins that kill insects by paralysis—"knock down". This is thought to be secondary to an effect on sodium channels in motor neurons. Mammals are spared from this effect as, unlike insects, they can metabolise pyrethrins in the hepatic microsomal system to non toxic compounds.1

We previously reported a case simulating motor neuron disease (MND) closely associated with over-exposure to permethrin and chlor dane based insecticides.2 The chlordane component is metabolised to various compounds (including oxychlordane and epoxychlordane) via the cytochrome P450 enzyme system.3,4 The metabolites of chlordane as permethrin are metabolised by different routes (fig). Following esterase cleavage, two products are formed which are further metabolised then eliminated. In mammals,5 metabolite A is excreted in the urine as a glucuronide conjugate. Metabolite B under goes oxidation and hydroxylation to yield mainly C and D, with other compounds. Conjugates of minor metabolites with glycine,6 glutamic acid6 and taurine6 have been noted. However, the elimination of metabolites C and D in the rat is by sulphate conjugation7 which is the major metabolic pathway for permethrin.

This is of interest since we recently reported8 that MND patients have a defect in their ability to convert cysteine to inorganic sulphate and also show a poor capacity to form the sulphonate conjugate of paracetamol. In the light of these observations, the 4'-Hydroxy-3-phenoxy benzoic acid (meta bolite C) or and/or 4'-Hydroxy-3-phenoxy benzoic acid (metabolite D) may be important for the neurotoxicity that resulted in our patient suffering from his MND-type illness. Some environmental chemicals generally

7. \[\text{Equation}\]
8. \[\text{Equation}\]
Values are expressed as the mean (±1 SEM) for data obtained using seven animals.

*p < 0.01 compared to vehicle treatment, Dunnett's multiple comparison test.
Pesticide toxicity and motor neuron disease.

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