Letters

Severe peripheral neuropathy due to lithium intoxication

Sir: Central nervous system damage due to lithium intoxication is well known, but evidence of peripheral nervous system damage is scanty. Three reports of individual cases have been made, in only one was the neuropathy severe, and all three cases recovered without apparent sequelae. We report a case of severe, persisting central and peripheral nervous system damage following lithium intoxication.

A 31-year-old man with a history of manic-depressive psychosis had been maintained on lithium carbonate 1500 mg/day. While working as a volunteer on an Israeli kibbutz, the dose was inadvertently increased to 1800 mg/day. He was admitted to a local hospital after the gradual onset of confusion, weakness, and tremulousness. Serum lithium level on admission was 3.63 mmol/l, and daily haemodialysis was instituted. However, he became comatose after two days and required assisted ventilation. Nephrogenic diabetes insipidus was diagnosed and treated with frusemide and salt restriction. He regained consciousness 11 days after admission, but could obey only simple commands, was hardly able to speak, could swallow liquids only, and remained bedridden. Urinary tract infections were treated with carbencillin and amikacin. Diarrhoea was treated with diphenoxylate and atropine. Serum lithium by the 18th hospital day had fallen to 0.34 mmol/l, and 8 weeks after admission he was transferred back to Australia. Examination revealed a wasted bedridden young man who was mute. He could obey very simple verbal commands, but any attempt to elicit speech or complex motor tasks caused extreme agitation accompanied by grinding of his teeth. Snout, sucking and bilateral grasp reflexes were elicited. Gag reflex was absent and he was unable to swallow. Upper limb movements were accompanied by gross intention tremor; there was marked hypotonia and distal muscle weakness, with complete left wrist drop. There was flaccid paralysis of the lower limbs and extensive decubitus ulceration. Deep tendon reflexes were diminished proximally and absent distally in arms and legs. Both pinprick and joint position perception were absent distally in all limbs. Haemoglobin was 11.4 g/dl, white blood count 15.7 × 10³, serum albumin 32 g/l, potassium 2.5 mmol/l. Other routine haematological and biochemical tests were normal. The cerebrospinal fluid contained no cells, but the protein level was elevated at 0.70 g/l. Serum B12, urinary heavy metals, and porphyrin studies were normal. An electroencephalogram showed diffuse theta and occasional delta waves. Computed tomography of the head showed a mild degree of cerebral atrophy. Nerve conduction studies confirmed the presence of a severe peripheral neuropathy: no sensory or motor responses could be obtained in the lower limbs, while in the upper limbs the amplitude of both sensory action potentials and compound muscle potentials was reduced. However, motor conduction velocity of both median and ulnar nerves was normal. Right sural nerve biopsy revealed moderate loss of myelinated fibres. Many fibres showed evidence of axonal degeneration: there was splitting of myelin lamellae, myelin breakdown products within macrophages, dense body accumulation within axons, and marked enlargement of the endoneuroal space. Biopsy material was also examined with the teased fibre technique and under the electron microscope, and these examinations confirmed acute axonal degeneration. Two months later he could walk in a frame with the aid of two people. He remained weak distally with absent distal tendon reflexes, but his speech and tremor had improved.

This patient showed many of the central nervous system effects of lithium intoxication. In addition he had a severe persistent peripheral neuropathy, which was shown to be due to acute axonal degeneration. Other known causes of this type of peripheral neuropathy were excluded by history or special investigations. Abnormal nerve conduction velocities have been recorded in manic-depressives and asymptomatic volunteers receiving lithium. It is becoming increasingly apparent that lithium intoxication can cause a peripheral neuropathy of varying severity. A mild neuropathy could be overlooked as the central nervous system damage is usually more dramatic. A peripheral neuropathy should be looked for in all patients on chronic lithium therapy, and may be more common and potentially more serious than previously thought.

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References


Acute autonomic and sensory neuropathy associated with elevated Epstein-Barr virus antibody titre

Sir: We report a rare case with neuropathy manifesting marked autonomic and sensory disturbances associated with high antibody titres of Epstein-Barr virus. On 9 March 1978, a 23-year-old Japanese woman noticed pricking sensation and paraesthesiae of the whole body. Muscle weakness of all extremities occurred rapidly and two days later she became bedridden. She had dysarthria, dysphagia, nausea, vomiting and hypersalivation. On the fourth day of illness, she experienced difficulty in respiration and urinary retention. Two months later, dysarthria, dysphagia, and muscle weakness gradually improved. She became able to walk 9 months after the onset, when she began to experience fainting spells upon arising. In spite of noticeable improvement of muscle power, severe sensory impairment of the whole body and diarrhoea persisted. She often burned her hand when cooking. The patient was transferred to our hospital 2 years and 8 months after the onset. There was no history of intoxication by drugs or food preceding the illness. Past history, family history and life history were non-contributory.
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