

Cerebellar syndrome in lithium poisoning: a case of partial recovery

Sir: Acute poisoning may occur during antidepressant treatments with lithium salts. Unpredictable precipitating factors include fever, low food intake, erroneous measurement of serum concentration, or suicide attempts. Fifteen per cent of the victims die, while 10% have persisting after-effects, the most frequent of which are cerebellar syndromes.¹⁻³

A 51 year old man ingested in a few hours 24 g of lithium carbonate (80 tabs), 5 litres of beer and 20 mg of a benzodiazepine (lorazepam, 20 tabs), with suicide intent. In 24 hours coma developed, with hyperthermia, neck stiffness, tetraplegia, double incontinence, massive haematuria, and respiratory failure. Intubation and peritoneal dialysis were required. Two days after poisoning serum lithium ion level was 3.7 mmol/l, three to four times the therapeutic concentration. A CT scan was normal (scan a in the figure). Consciousness reappeared 1 week after intoxication, when lithium concentration was 0.25 mmol/l.

There were generalised weakness and incoordination, horizontal bilateral nystagmus, double vision on upward gaze, dysarthria with slurred and scanning speech, but neither sensory deficits nor abnormal plantar responses. Forty five days after intoxication a second CT scan showed an enlargement of the 4th ventricle, indicating cerebellar atrophy (scan b in the figure).

The patient improved: within 4 months strength and coordination of the upper limbs were restored; speech recovered remarkably. Nevertheless, ataxia of the lower limbs and ataxia/hyperreflexia of the trunk still made standing and locomotion impossible. The patient had to learn to handle skilfully two forearm crutches: thus, 8 months after poisoning he could stand and walk. Nystagmus, double vision and tendon areflexia also persisted.

Two years after poisoning the clinical picture was unchanged.

This case confirms that cerebellar atrophy is the main late sequela of acute lithium poisoning. Nevertheless, it also confirms⁴ the possibility for a partial recovery of speech and motor functions.

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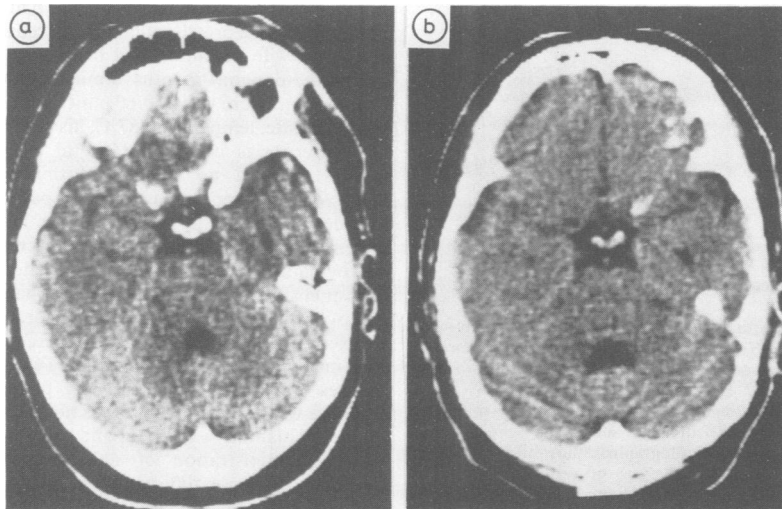
Development of Wolff-Parkinson-White syndrome in a patient with Friedreich's ataxia

Sir: The features of cardiac disease associated with Friedreich's ataxia have recently been reviewed¹ but we are unaware of a previous report of Wolff-Parkinson-White syndrome (WPW) in this condition, or of the development of cardiomyopathy in Friedreich's ataxia in the first year of life.

In 1969, a male infant presented at 5 months of age with dyspnoea. A harsh systolic murmur and precordial thrill were detected and he was thought to have a ventriculo-septal defect. At 5 years of age he was noted to become cyanosed while crying. His ECG at this time showed left ventricular hypertrophy (LVH) with widespread T-wave inversion, a normal PR interval (0.18 ms), and cardiomegaly on chest radiographs. Cardiac catheterisation in 1976 showed no valvular disease or septal defect, but a raised left ventricular end-diastolic pressure was found. A diagnosis of "non-obstructive hypertrophic cardiomyopathy" was made.

Two years later at 10 years of age, he was referred to this unit for the evaluation of ataxia. His mother had first noticed unsteadiness of gait and a tendency to fall frequently when he had started walking at around 13 months of age. This had progressed and he had never been able to participate in games or sports. Examination revealed sensorineural deafness in the left ear, mild cerebellar ataxia of limbs and gait, slight dysarthria but no nystagmus. The tendon reflexes were depressed in the arms and absent in the legs, and the plantar responses were extensor. He had bilateral pes cavus. There was no family history of similar problems.

Motor nerve conduction velocities in the right ulnar and lateral popliteal nerves were slightly reduced at 48 and 40 m/s respectively and sensory nerve action potentials were not recordable. Glucose tolerance was normal. A diagnosis of Friedreich's



CT scan after lithium poisoning: normal findings after 48 hours (a); enlargement of the 4th ventricle, indicating cerebellar atrophy, after 45 days (b).