

Letters

Cerebellar syndrome in lithium poisoning: a case of partial recovery

Sir: Acute poisoning may occur during anti-depressant 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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L TESIO
GL PORTA
E MESSA

*Servizio di fisiokinesiterapia
e riabilitazione
Istituto Geriatrico "Pio Albergo
Trivulzio", via Trivulzio 15,
20100 Milano, Italy*

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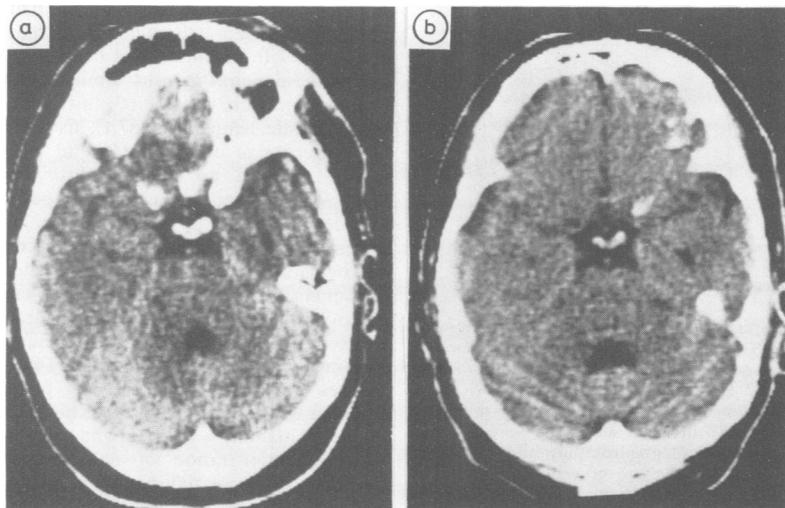
Development of Wolff-Parkinson-White syndrome in a patient with Friedreich's ataxia

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).

ataxia was made. He subsequently developed progressive scoliosis, pyramidal weakness and increasing ataxia and is now wheelchair-bound.

Six months before referral he had suffered an isolated episode of acute chest pain and loss of consciousness. A cardiac dysrhythmia was suspected but his ECG showed no change from earlier recordings. In May 1984 he began to experience fainting episodes at school and 6 months later presented as an emergency with acute chest pain and dyspnoea and was found to have a paroxysmal supraventricular tachycardia. This was treated with intravenous practolol. The resting ECG now showed the typical features of WPW Type B (fig). Treatment with propranolol and quinidine has prevented further symptoms to date.

Cardiac symptoms and signs occur in about one-third of patients with Friedreich's ataxia¹ but electrocardiographic abnormalities are more common,² with T-wave inversion in several leads, with or without other features of left ventricular hypertrophy, being reported in 30%³ to 90%⁴ of cases in different series. In a series of 115 cases, Harding and Hewer² found only one patient with a short PR interval and none with atrio-ventricular block. Supraventricular tachycardia was noted in eight cases and ventricular extrasystoles in two. Atrial fibrillation occurred in one patient;

both arrhythmias were associated with a poor prognosis.^{2,5}

While there is often evidence of asymptomatic cardiac involvement at the onset of neurological symptoms, the development of cardiac signs and symptoms prior to neurological dysfunction is rare.^{2,6-8} Berg *et al*⁶ described two sibs who were eventually found to have Friedreich's ataxia, who presented with cardiac failure at 3 and 5 years of age, and Nadas *et al*⁷ reported a 3½ year old child who had cardiomegaly, a murmur and ECG abnormalities at the time of presentation with unsteadiness. However, onset under one year of age as in the present case has not been documented previously.

Pre-excitation syndromes such as WPW are generally believed to be due to the presence of accessory functioning pathways between the atria and ventricles. In typical cases of WPW these accessory pathways, which are composed of functional myocardial cells (or rarely specialised conducting tissues), are believed to be vestiges of a more extensive atrioventricular ring found in the embryonic heart.⁹ However, the present case illustrates that such pathways are not necessarily congenital but can evolve as a result of a progressive cardiomyopathy. Our patient had a form of hypertrophic cardiomyopathy and this appears to be the commonest cardiac disorder in Friedreich's ataxia.¹ Concentric left ventricular hypertrophy occurs most frequently¹ but outflow tract obstruction due to asymmetric septal hypertrophy can develop and was demonstrated in nearly one-third of cases in one series;¹⁰ cardiac dysrhythmias, including WPW, are not uncommon under such circumstances.¹¹

R NEIL BALDWIN
RUSSELL JM LANE
Associated Unit of Neurological Science,
Walton Hospital,
Rice Lane,
Liverpool L9 1AE, UK

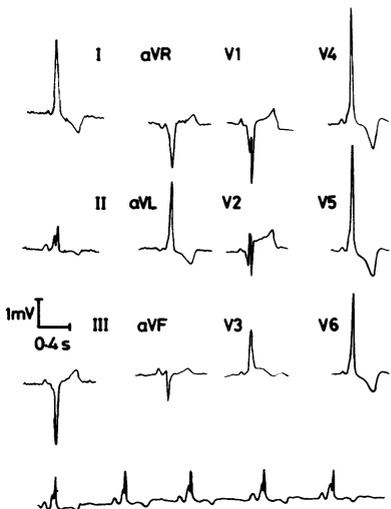


Fig Electrocardiogram recorded in November 1984, showing left ventricular hypertrophy and WPW Type B. The PR interval is 0.04 ms, delta waves are present and the QRS complex is widened (0.12 ms) with a negative initial deflection in V₁. Widespread T-wave inversion is evident.

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Flunitrazepam intoxication simulating a structural brainstem lesion

Sir: We wish to report a case of severe brainstem dysfunction provoked by an overdose of flunitrazepam.

A 60-year-old man was brought to the emergency room, unconscious, after being found unresponsive by his wife. He had a past history of depressive illness, but according to his wife his mood had been normal during the previous months. On admission, his blood pressure was 120/60 mmHg, pulse 68 per minute, temperature 37°C; his breathing pattern was irregular. General examination revealed no further information. Neurological examination revealed a patient in a deep coma, with no spontaneous limb movements and decerebrate posturing elicited by painful stimulation. He had bilateral unresponsive small pupils. Oculocephalic, oculovestibular, ciliospinal and corneal reflexes were absent. There was an increased tone in lower and upper extremities. Tendon reflexes were brisk and the left plantar response was extensor. There was no nuchal rigidity. Thiamine was given, and the intravenous administration of naloxone and hypertonic glucose did not alter his neurological status. Baseline blood tests, blood gases, chest radiograph, electrocardiogram and CT scan of the head were normal. Elec-