Matters arising

Cerebellar syndrome in lithium poisoning

Sir: Tesio *et al*'s recent report on lithium-related cerebellar injury is of particular interest since cerebellar atrophy is illustrated by computed tomography (CT).¹ I wish to describe one additional subject in whom I equally believe cerebellar atrophy developed as the result of lithium toxicity. A 27 year old male with a history of schizo-affective disorder became progressively obtunded and developed generalised seizures and rigidity a few days after receiving haloperidol, diazepam and lithium carbonate for acute exacerbation of his psychosis. He had no history of drug addiction or heat exposure. Ventilatory support was required but episodes of hypoxaemia were never documented. No evidence of infection was encountered. Spinal fluid examination and initial CT of the brain were normal while the electroencephalograms showed diffuse slowing. Lithium blood level was 1.5 mmol/l. The patient regained consciousness in 3 days and was discharged from the hospital one month after admission. On neurological examination 6 months later, he exhibited scanning speech, coarse biplanar gaze-evoked nystagmus, pronounced ataxia of limbs and of locomotion and bilateral Babinski signs. Brain CT revealed 4th ventricular and basal cisternal dilatation and marked parenchymal cerebellar atrophy (figs 1, 2). His ataxia has progressively improved but after 3 years he still requires assistance in ambulation. The CT scan remains unchanged. Although this patient's blood lithium levels were not in the toxic

range, ample evidence has been provided to support the occurrence of lithium intoxication with "normal" serum concentrations.²⁻⁵ The clinical presentation of these individuals resembles that of neuroleptic malignant syndrome; in fact, often these patients have been receiving a combination of neuroleptics and lithium.³ Cohen and Cohen suggested a reaction of incompatibility between haloperidol and lithium causes the syndrome;⁶ in more recent publications it has been argued, however, that toxicity is solely due to lithium.⁷ Cerebellar ataxia as part of a diffuse encephalopathy is not uncommon in patients with neurological sequelae from lithium intoxication; by contrast, isolated cerebellar ataxia is comparatively rare.⁸⁻¹⁰ Residual cerebellar atrophy, demonstrated by pneumoencephalogram or CT, is also encountered exceptionally.^{1,3,5} These cases underscore the vulnerability of the cerebellum to lithium and the necessity of physicians to be aware of this potential complication even in the presence of "non-toxic" blood levels. Prompt discontinuation of lithium at first symptoms will prevent permanent sequelae.¹⁰ Jaime Rubio, MD, referred the patient.

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Recurrent subarachnoid haemorrhage due to spinal haemangioma

Sir: We have read with interest the letter by Van Hille *et al*¹ about the difficulties in the diagnosis and the rare presentation of spinal haemangioblastomas as subarachnoid haemorrhage. Recently we have had the opportunity to study a patient whose clinical outset was a spontaneous subarachnoid haemorrhage caused by a spinal vascular tumour.

A 15 year old boy was admitted because of sudden onset of headache, vomiting, backache and neck stiffness. On examination he had meningism with minimal weakness in the left arm and leg.

Lumbar puncture yielded yellowish fluid with 98 red cells/mm³. A cranial computed tomography (CT) scan was normal but spinal cord CT showed two hyperdense lesions at the cervical and dorsal levels. A total myelography was performed that made evident a spinal cord enlargement with a negative-image of abnormal vessels at C4-C5 and T3 levels. Selective spinal cord arteriography showed two vascular tumours at the above mentioned situation. Several complementary tests were made in order to rule out a Hippel-Lindau disease. All them were normal. A laminectomy was carried out and the dorsal tumour was removed. Eight months after that he had a new epi-

Fig 1 CT of brain showing 4th ventricular and cisternal enlargement and parenchymal cerebellar atrophy.

Fig 2 CT scan showing parenchymal cerebellar atrophy.