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 binocular 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.6 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.11-13 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.

References


Recurrent subarachnoid haemorrhage due to spinal haemangioma

Sir: We have read with interest the letter by Van Hille et al1 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.

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Sode of meningism with tetraplegia that was attributed to rebleeding. He developed hydrocephalus and died with a nosocomial infection. The anatomical diagnosis was hemangioblastoma.

Spinal intramedullary haemangioblastoma and similar tumours present diagnosis problems clinically, radiologically and pathologically. The spinal haemangioblastomas are certainly a rare cause of spontaneous subarachnoid haemorrhage and died with a nosocomial infection. The anatomical diagnosis was hemangioblastoma.

CT Scan of the brain shows a hyperdense mass within the left parietal lobe, consistent with a haemangioblastoma. The differential diagnosis includes metastasis, choroid plexus papilloma, and meningioma.

The radiologist was able to confirm the presence of the mass as a haemangioblastoma.

References


Computed tomographic findings of brain and skull in myotonic dystrophy

Sir: With great interest we read the article by Avrahami et al about computed tomographic findings of brain and skull in myotonic dystrophy. However, I do not agree with their opinion that bones in the base of the skull and others of the body seem not to be involved. We have previously reported two myotonic dystrophy patients with ossification of the posterior longitudinal ligament causing transverse myelopathy, in that one patient with calvarian hyperostosis but another with not only calvarian hyperostosis but also abnormal ossification of the clivus, which was confirmed by necropsy. To our knowledge, ossification of the posterior longitudinal ligament has not been recognised in patients with myotonic dystrophy outside of Japan, of which there have been at least eight reports. Ossification of the posterior longitudinal ligament is a common condition in Japan, frequently incidentally identified, but sometimes causing myelopathy. Although the precise cause of ossification of the posterior longitudinal ligament remains unknown, it is suspected that it may be a part of manifestations of the generalised hyperostotic potential in patients, probably genetically transmitted, because of high incidence of association with ossification of the other ligaments of the spine and concurrence in the same families on national surveys of ossification of the posterior longitudinal ligament.  

Avrahami et al hypothesise that the cause of hyperostosis of the calvarium is secondary to microcephaly because the base of the skull is not involved. However, in our two patients with ossification of the posterior longitudinal ligament, one had even hyperostosis of a part of the base of the skull. Jequier has suggested that cranial hyperostosis might have been genetically determined in patients with myotonic dystrophy. In Japan, there has been a case of concurrence of myotonic dystrophy and ossification of ligaments of the spine in two siblings and additionally calvarian hyperostosis in one reported. Therefore, it is suspected that association with ossification of the posterior longitudinal ligament and hyperostosis of the clivus in myotonic dystrophy may be more than fortuitous. Further investigations are certainly needed to clarify whether patients with myotonic dystrophy have generalised hyperostotic potential.

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References


Korczyn and Avrahami reply:

We are grateful to Dr Kawamura for drawing our attention to his work, demonstrating ossification of the posterior longitudinal ligament in some Japanese patients with myotonic dystrophy. As Dr Kawamura notes, this phenomenon is common among Japanese, and the relationship to myotonic dystrophy in the cases described by him may therefore be fortuitous. Otherwise, this heterotopic calcium deposit is more likely to be related to basal ganglia calcification described by us than to the thickening of the calvarian bones. Clearly, however, further studies on calcium and bone metabolism in myotonic dystrophy are needed.

Notice

The World Federation of Societies of Biological Psychiatry

A Regional Congress will be held 2-7 April 1989 in Jerusalem. Information may be obtained from Professor RH Belmaker, Chairman, POB 983, Jerusalem 91009, Israel.
Recurrent subarachnoid haemorrhage due to spinal haemangioma.
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