there was no fever; the patient was stuporose and dysphasic. Memory was severely impaired but spatial skills appeared to be preserved. There was visual inattention and a hemiparesis on the right with absent joint position sense in the right hand. There were frequent right-sided focal motor seizures.

A CT scan of the brain revealed only a small lacunar infarct in the left lentiform nucleus, an electroencephalogram showed left-sided slow waves and bilateral frontal intermittent rhythmic delta activity. The CSF contained 15 red cells/μl and no leucocytes, protein 0.81 g/l and glucose 14.1 mmol/l.

The patient was given intravenous acyclovir, phenytoin and insulin. After 5 days, the CSF contained 3 lymphocytes/μl, protein 0.46 g/l and glucose 5.6 mmol/l (blood glucose 12.5 mmol/l). High affinity antibodies to Herpes simplex virus type 1 were detected in the CSF (and not the serum) by ELISA and antibody heterogeneity was confirmed by agarose gel isoelectric focusing and antigen immunoblotting with immunoperoxidase staining on polyvinyl difluoride membranes. The patient made a good recovery over the next two weeks.

This case demonstrates that a raised CSF cell count may be absent in advanced HSE. Previously, one other case without a CSF pleocytosis on the first, second and eighth days of proven HSE was reported. In our patient, both CSF specimens were taken even later after the onset of HSE.

HSE is a serious disease; without treatment, many patients die and few survivors are neurologically intact. Antiviral therapy is effective in HSE but results are better with early treatment. For this reason, when HSE is suspected, empirical treatment with acyclovir is advisable without waiting for laboratory confirmation which cannot be achieved in the early stages of the illness. In this situation, we wish to emphasize that the absence of a cellular response in the CSF does not exclude HSE, even after more than two weeks from the onset.

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References


Cerebellar ataxia due to hyponatraemia

Sir,

Hyponatraemia, a common electrolyte disorder in the medical setting, has protein causes and clinical manifestations, but cerebellar symptoms and signs are extremely rare. We describe two cases of reversible cerebellar ataxia, induced by severe hyponatraemia.

Case 1

A 35 year old male patient was admitted with progressive weakness, pallor and purpura of 3 months duration shown to be aplastic anaemia. On the 7th day of his hospital stay he developed severe watery diarrhoea and became drowsy. He had received 80 mg intravenous frusemide with blood transfusion the previous night. Examination revealed moderately severe volume depletion, slurred speech, bilateral intention tremors, past pointing, impaired knee–heel test and ataxia. Serum sodium was 96 mmol/l. Other biochemical investigations and computed tomographic scan of the head were normal. His hyponatraemia was corrected over a period of 3 days, during which his cerebellar signs rapidly diminished and disappeared over the following 2 days.

Case 2

A 15 year old boy presented with intermittent fever, anorexia and recurrent vomiting for 10 days, unsteadiness of gait and head bobbing of 2 days duration prior to the admission. He had received a 7-day course of ampicillin before admission. He was febrile, moderately dehydrated and had soft splenomegaly. Neurological evaluation revealed striking dyssynergia, marked truncal ataxia, impaired coordination, broad-based ataxic gait and scanning speech. Investigations showed a positive Widal test, serum sodium 110 mmol/l, serum potassium 3.5 mmol/l and sterile blood culture. He was given intravenous ciprofloxacin and volume depletion was corrected with saline. His neurological symptoms rapidly improved within 24 h of hospitalization. He achieved complete neurological recovery within 3 days and became afebrile on the 5th day after admission.

Various authors have highlighted aphasia, hypo- and hyperreflexia, generalized rigidity, hemiparesis, focal weakness and unilateral Babinski sign in hyponatraemia reports. Ataxia with hyponatraemia has been described with the use of carbamazepine, in acute intermittent porphyria and associated with bronchogenic carcinoma. Indeed, a pure cerebellar syndrome has only been described in the last setting, thus re-emphasizing the rarity of this manifestation. In both the cases described in this report, gross cerebellar signs were observed, though absence of nystagmus was noteworthy. Volume depletion was the triggering mechanism of hyponatraemia, a fact not described earlier in connection with hyponatraemia and cerebellar syndrome. Although cerebellar ataxia has been noted as a rare feature of enteric fever, it is slowly reversible over weeks to months, if at all, making it an unlikely possibility in the second case. Thus, in an appropriate clinical setting, it may be worth
considering hyponatraemia as a cause of reversible cerebellar dysfunction.

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References


Spontaneous flail chest in multiple myeloma

Sir,

Spontaneous flail chest is rare in multiple myeloma but poses a difficult therapeutic problem.1–3 We report prolonged survival of a patient after hemibody irradiation. He presented at age 59 with a 2 month history of chest pain. On examination there was a large anterior flail segment, with paradoxical movement of the sternum and 4th to 9th ribs bilaterally; a chest X-ray revealed multiple bilateral rib fractures. Full blood count and renal function were normal. Abnormal findings confirming myelomatosis were a serum calcium of 2.7 mmol/l (albumin 36 g/l), a faint M-band in serum, 0.1 g/l of kappa light-chains in urine, widespread lytic bone lesions, and 41% atypical plasma cells in bone marrow. There was no respiratory failure. Ten milligrams of melphalan and 40 mg prednisolone were given daily for 3 days but his pain worsened. He was therefore treated with a modified upper (600 cGy in single fraction), followed by lower, hemibody irradiation4 and then 9 further courses of melphalan and prednisolone. Four months after starting treatment there was reduced mobility of the flail, radiological bone healing, improved spirometry and under 2% plasma cells in bone marrow. His chest is deformed but stable 4 years after diagnosis.

Although skeletal involvement is common in multiple myeloma, reports of spontaneous flail chest are rare. Our experience accords with previous descriptions:1–3 despite its alarming presentation, this complication does not necessarily predict a poor prognosis and justifies an aggressive approach to therapy.

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References


NACT Training Package

This is an open learning study pack which aims to improve the teaching skills of clinical tutors and other tutors. Topics covered include junior doctors' educational skills and their provision, the funding and management of resources, managing change, and the operation of postgraduate centres. The development of the package was funded by the Department of Health and produced with the help of the Open University and the Joint Centre for Educational Research and Development in Medicine.

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doi: 10.1136/pgmj.68.797.230

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