SHORT REPORTS

Hyponatraemia as a cause of reversible ataxia

Apart from ataxia induced by drugs or alcohol, reversible causes of cerebellar dysfunction are rare. We report on two patients with inappropriate antidiuretic hormone secretion as a result of bronchial carcinoma who presented with severe cerebellar ataxia that was fully reversible on correction of their serum sodium concentrations.

Case reports

CASE 1

A 64 year old man was admitted with a history of five weeks of unsteadiness and two days of slurred speech. His medical history was unremarkable. He had not been receiving any drugs, smoked five cigarettes a day, and rarely drank alcohol.

Neurological examination showed slight signs of confusion, which had not been noticed by his relatives. He had an obvious dysarthria and a noticeably ataxic gait and could not walk unaided. He had a bilateral intention tremor, and heel to shin coordination was poor. Fundi and cranial nerves were normal, and there was no nystagmus. Reflexes, strength, and sensation were also normal. Serum sodium concentrations were: 109 mmol(mEq)/l, potassium 4·2 mmol(mEq)/l, and urea 5·4 mmol(mEq)/l (32 mg/100 ml). Corrected serum calcium concentration was 2·16 mmol/l (8·6 mg/100 ml). Serum osmolality was 229 mmol(mosmol)/kg, and urinary osmolality was 628 mmol/kg. An *x* ray film of his chest showed left hilar lymphadenopathy. Bronchoscopy showed a tumour, which histological examination identified as a small cell carcinoma. The results of computed tomography of the skull, examination of cerebrospinal fluid, and an electroencephalogram were all normal. Thyroid function was normal, and results of treponemal serology were negative.

His fluid intake was restricted to 800 ml/day, and he received demeclocycline 600 mg/day. His serum sodium concentration rose over the next few days to 136 mmol/l, his cerebellar signs improved, and after one week there was no neurological deficit. He remained asymptomatic for three months but then deteriorated rapidly and died. An autopsy was not performed.

CASE 2

A 68 year old woman was admitted with a two week history of unsteadiness associated with a recent cough and anorexia. Medical history was unremarkable, and she was taking no drugs. She smoked 20 cigarettes a day and did not drink alcohol.

Examination showed poor heel to shin coordination and inability to walk unaided. There was some disorientation but no overt confusion. All reflexes were normal, and she showed no weakness or sensory loss. Fundi and cranial nerves were normal, and there was no nystagmus. Serum sodium concentration was 108 mmol/l, potassium 3·8 mmol/l and urea 4·7 mmol/l (27·2 mg/100 ml). Serum osmolality was 234 mmol/kg and urinary osmolality 702 mmol/kg. Corrected serum calcium concentration was 2·32 mmol/l (9·2 mg/100 ml). Radiography of the chest showed a mass in the right mid-zone with hilar lymphadenopathy. Cytology of sputum showed malignant cells of small cell type. A computed tomography scan of her skull and an electroencephalogram were normal. Thyroid function was normal and results of treponemal serology negative.

Her fluid intake was restricted to 700-800 ml/day, and her serum sodium concentration rose to 129 mmol/l with resolution of her ataxia. She remained well for six weeks but then developed pneumonia and died. Autopsy was not performed.

Comment

Hyponatraemia is associated with confusion and, at serum sodium concentrations of less than 120 mmol/l, with epilepsy and coma.[1,2] An almost pure cerebellar syndrome is not, however, a recognised feature. Ataxia coexisting with hyponatraemia has been reported in patients with carbamazepine toxicity and porphyria,[3,4] but in our patients ataxia was probably caused by the effect of the metabolic disorder on the central nervous system.

A subacute cerebellar degeneration is a recognised non-metastatic effect of bronchial carcinoma but is progressive and rarely reversible, even when the primary tumour is treated.[5] This therefore cannot explain the ataxia in these patients unless a subclinical, non-metastatic cerebellar syndrome had been greatly exacerbated by the low sodium concentrations.

We suggest that reversible cerebellar dysfunction should be recognised as a rare presenting feature of hyponatraemia in patients with inappropriate antidiuretic hormone secretion. Whether this occurs only when the syndrome is secondary to carcinoma is unknown.

We thank Mr D T Beswick and Mrs Pamela Vernon for their help in preparing this report.

1 Bartter FC, Schwartz WB. Syndrome of inappropriate secretion of anti-diuretic hormone. *Am J Med* 1967;**42**:790-806.
2 Detroyer A, Demanet JC. Clinical, biological and pathogenic features of the syndrome of inappropriate secretion of ADH. *Q J Med* 1976;**45**:521-31.
3 Ashton MG, Ball SG, Thomas TH, *et al*. Water intoxication associated with carbamazepine treatment. *Br Med J* 1977;i:1134-5.
4 Lipschutz DE, Reiter JM. Acute intermittent porphyria with inappropriately elevated ADH secretion. *JAMA* 1974;**230**:716-7.
5 Oelbaum MH, Statham R. Carcinoma of the bronchus presenting as cerebellar neuropathy. *Postgrad Med J* 1961;**37**:546-9.

(Accepted 8 August 1986)

Department of Neurology, Selly Oak Hospital, Birmingham B29 6JD
S M KELSEY, BSC, MB, senior house officer
A C WILLIAMS, MD, MRCP, consultant neurologist
D CORBIN, MB, MRCP, registrar in neurology

Correspondence to: Dr Kelsey.

Finger clubbing in the Zollinger-Ellison syndrome

A case of the Zollinger-Ellison syndrome with clubbing of the fingers is described. So far as we know this association has not been reported before. The pathogenesis of clubbing is examined with particular reference to this case.

Case report

A 15 year old boy was referred in 1983 with a three year history of diarrhoea and episodic epigastric pain. There had been a severe bout of haematemesis and melaena three months previously. There was no relevant past medical history or family history of illness, including pachydermatoperiostosis.

Examination showed no abnormality other than clubbing of all the fingers. Facial features and skin were normal. Results of a barium meal study and endoscopy were normal apart from an ulcer in the first part of the duodenum. Radiographs of chest and hands were normal. Serum gastrin concentration was 1350 pmol/l (upper limit of normal 30 pmol/l); vasoactive intestinal peptide, pancreatic polypeptide, glucagon, and neurotensin values were not raised. Full blood count and urea and electrolyte, glucose, calcium, and phosphate concentrations were normal. Thyroid and adrenal functions were normal.

Computed tomography detected a tumour in the superior aspect of the head of the pancreas, which was confirmed by a coeliac axis angiogram. At operation a 3 cm trilobulated tumour on the superior aspect of the head of the pancreas was removed. No metastases were seen. Histologically the tumour consisted of sheets and clusters of regular polyhedral cells with a little eosinophilic cytoplasm and regular vesicular nuclei, consistent with a G cell tumour of pancreatic origin. Postoperative recovery was uneventful. Within a year of operation the finger clubbing had disappeared. Three years later the patient was well and the serum gastrin concentration had remained normal.

Comment

Many intrathoracic conditions are known to be associated with finger clubbing, including bronchial and oesophageal carcinoma, mesothelioma, bronchiectasis, lung abscesses, empyema, fibrotic lung diseases, subacute bacterial endocarditis, and cyanotic heart disease. Extrathoracic causes of clubbing include tumours of the upper gastrointestinal tract, ulcerative colitis, Crohn's disease, achalasia of the cardia, polyarteritis nodosa, α chain disease, Hodgkin's disease, and cirrhosis of the liver. The causative mechanism of this phenomenon remains uncertain.

Similar changes may be induced in dogs by the surgical production of a cardiac right to left shunt. It may be that substances capable of producing these changes are produced by various organs of the body but are normally inactivated in the pulmonary vasculature. Shunting results in their presence in the general circulation with consequent clubbing. Ferritin, a substance inactivated by the lungs, has been found in arterial blood only of patients who have clubbing[1]; this has not, however, been confirmed experimentally. A similar mechanism affecting the liver might account for the association of clubbing with liver disease.

A neurogenic explanation has also been advanced. Increased vascularity of the diseased organ may initiate a neural reflex, the afferent limb of which is vagal. This is supported by reports that clubbing may disappear after vagotomy.[2]