A Misleading Case of Central Pontine Myelinolysis

Risk Factors for Psychiatric Patients

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Central pontine myelinolysis (CPM) is an uncommon disorder initially described in alcoholic or malnourished patients. Recent reports suggest an aetiological association with abnormalities of serum sodium. A physically unwell non-alcoholic chronic schizophrenic patient, whose symptoms led to psychiatric referral, died of CPM. A review of the literature reveals that psychiatric patients may indeed be at risk for CPM.
British Journal of Psychiatry (1992), **160**, 550–552

In 1959 Adams *et al* reported a previously undescribed condition characterised by symmetrical loss of the myelin sheaths of all nerve fibres in the central part of the basis pontis, with sparing of the nerve cells and axon cylinders and non-involvement of the blood vessels, for which they coined the pathodescriptive term 'central pontine myelinolysis' (CPM). Their four patients, all of whom died, were either alcoholic or malnourished.

Although early experience suggested an invariably poor outcome for CPM, recovery was subsequently documented in patients who clinically seemed to show dissociation at the pontine level: brisk reflexes, extensor plantars, quadriparesis leading to quadriplegia, dysarthria, and dysphasia, giving a 'locked in' appearance (Estol *et al*, 1989). The severity of symptoms seemed to vary with the size of the lesion. Hemiparesis, spastic weakness of the arms, and oculomotor abnormalities were noted in milder cases, while small lesions in the mid-pons gave rise to few clinical signs. Confusion or stupor were often present (Arieff, 1985; Haibach *et al*, 1986).

Magnetic resonance imaging (MRI) and high-resolution computerised tomography (CT) now permit the diagnosis of CPM *in vivo* and have charted resolution of the pontine lesion (Ragland *et al*, 1989). Neuroradiological resolution may lag behind clinical resolution (Gerber *et al*, 1983).

Deranged serum sodium levels are currently understood to be of prime importance in the development of CPM. Hyponatraemia *per se* was initially implicated, but it has since been widely suggested that CPM is an iatrogenic disorder which may be precipitated by the rapid correction of a low serum sodium (Laureno, 1983). At least 60% of cases of CPM have a history of alcoholism (Arieff, 1985). Liver dysfunction may be of specific importance (Estol *et al*, 1989).

The mechanism of injury in CPM is not known. Messert *et al* (1979) emphasised the grid-like arrangement of fibres in the basis pontis and suggested that they might, therefore, be susceptible to 'strangulation' in the event of cerebral oedema. However, the hypothesis does not account for the 10% of cases (Wright *et al*, 1979) which show additional extrapontine involvement, and a role for oedema has not been established (Laureno, 1983).

Case report

The patient, W, was a 50-year-old divorced chronic schizophrenic living with her widowed mother. She had been admitted four times to the psychiatric unit, between 1967 and 1988, suffering from persecutory delusions and auditory hallucinations. Her first admission, however, had been in 1962 with a depressive illness which responded to a course of electroconvulsive therapy (ECT). Always a lady of some bulk, a cholecystectomy in 1984 had left her with a large incisional hernia. She gave no history of alcohol abuse.

W was admitted by surgeons with a one-month history of anorexia, vomiting, and diarrhoea. Her initial management was conservative, until she developed cellulitis in the region of her incisional hernia a week later. At laparotomy the next morning the terminal ileum was found to have perforated within the hernial sac; a right hemicolectomy was performed. Post-operative recovery was steady and W went home. Biochemical investigations during the admission had been unremarkable apart from an albumin level of 16 (normal range 35–52)g/l, which was corrected with parenteral human albumin.

W was readmitted by the surgical team six weeks later with persistent vomiting. On examination she was noted to be vague, slow, and uncooperative. She was apyrexial and dry. Her pulse was regular at 96 beats per minute, blood pressure 130/80 mmHg. Heart sounds were normal and the chest clear. Her abdomen was soft and non-tender, bowel sounds were normal, and the laparotomy scar had healed. She was taking lorazepam (1 mg t.d.s.), orphenadrine (50 mg t.d.s.), and fluphenthixol (80 mg i.m. every three weeks), which had last been given six days earlier.

Blood results were as shown in Table 1; plasma osmolality was 227 (280–305)mOsm/kg, urine osmolality 480 mOsm/kg, urinary sodium less than 10 mmol/l, urinary potassium 78 mmol/l.

Clinically and biochemically the patient was considered to be volume depleted. She received 1 litre of 0.9% sodium

Table 1
Electrolyte results

	Sodium: mmol/l	Potassium: mmol/l	Chloride: mmol/l	Bicarbonate: mmol/l	Creatinine: μmol/l	Urea: mmol/l
Normal range	136–145	3.5–4.5	102–111	22–30	50–100	2.4–6.8
On admission, 10.00 h	102	2.2	52	38	77	5.9
Day 2, 10.19 h	111	2.9	65	34	77	4.6
Day 3, 12.07 h	124	3.2	81	33	95	4.4
Day 4, 11.05 h	126	4.3	99	21	63	3.9
Day 9	130	2.9	98	25	71	4.2

chloride intravenous infusion plus potassium chloride (60 mmol) 8-hourly for two days, followed by 1 litre of intravenous sodium chloride 0.9% infusion plus potassium chloride (20 mmol) 8-hourly for a further two days (Table 1).

Thirteen days after admission, while being supported by two nurses, W was reported to have put herself to the floor and refused to walk further. This incident prompted a request for psychiatric review. She was seen by a senior house officer in psychiatry who knew her from clinic attendances. He found her to be lacking in motivation and apparently preoccupied; however, he could elicit no psychotic phenomena and she reported no affective symptoms. He reduced her depot by 25% in view of her apparent weight loss, and asked for a further referral should her condition alter.

This referral came ten days later, and she was seen by the consultant psychiatrist whose care she was under. By then she was bed-bound, immobile, and mute. She had been reviewed by the physicians, but no organic cause for her condition had been identified. It was felt that she could be suffering from a severe depressive illness or an acute form of her chronic schizophrenia. Since she was unable to tolerate oral medication or to give consent, a joint medical recommendation was signed under section 3 of the Mental Health Act 1983 with a view to proceeding with ECT. A radionuclide brain scan was performed to exclude any intracranial pathology. The scan suggested an ill-defined area of increased activity peripherally in the right posterior parietal area; a CT scan was arranged to exclude a localised subdural haematoma, but the neurosurgeons identified no subdural or surgically relevant lesion. W was reviewed by the team on call for neurology. They found her to be warm, well perfused and apyrexial. She had no neck stiffness. Pulse was regular at 84 beats/min, blood pressure 130/90 mmHg. Heart sounds were normal. There were some crackles in her left lower chest, and the abdomen showed colonic loading. She was generally oedematous and the skin overlying her buttocks was breaking down. Conjugate eye movements were roving. Fundal examination was normal. She made no response to threat but moved her lower limbs and right arm to painful stimuli. Limb tone was flaccid and reflexes diminished; plantars were down-going. The CT was reviewed, and considered to be normal. An electroencephalogram (EEG) gave a slow, low-amplitude tracing thought to be consistent with her long-standing psychiatric diagnosis and treatment.

She was mildly anaemic and her urea and creatinine levels were moderately raised. Porphyrin and vitamin B_{12} assays were normal. T_3 and total T_4 levels were acutely low, reflecting her systemic ill-health. She was again hypoalbuminaemic; liver enzyme levels were elevated. A prolonged prothrombin time corrected with intravenous vitamin K and a lumbar puncture proved normal.

She was restarted on intravenous fluids and her general condition improved as her urea and creatinine levels fell. She was treated for a proven infection of her urinary tract and a possible chest infection.

W was referred back to the psychiatrists and the section 3 application was completed. Then the formal neuro-radiological report on the CT scan was telephoned to the ward: an area of decreased attenuation within the central pons was consistent with a diagnosis of CPM. W was regraded informal. She died one month after the second admission.

Post-mortem findings were as follows. Both lungs were congested and had basal bronchopneumonic changes. The abdominal cavity and peritoneal surfaces showed multiple adhesions. The liver was markedly enlarged and the cut surface appeared diffusely fatty; there was no gross evidence of cirrhosis. Histological examination revealed well demarcated demyelination within the mid-pons; no evidence of demyelination was found elsewhere.

Discussion

In retrospect, our patient's psychiatric history acted as a confounding factor in this case, as did our unfamiliarity with CPM unless as a rare complication of chronic alcoholism. In particular, we might have wished for prior knowledge of the association between CPM and hyponatraemia or its therapy. It is that association which concerns us now – for while our patient's psychiatric past seems not to have been relevant, we believe that psychiatric patients are indeed at risk for CPM in as much as they are at risk for hyponatraemia.

Dilutional hyponatraemia may be caused by a number of psychoactive drugs via the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), which is characterised by hyponatraemia and hypo-osmolarity of the serum and extracellular fluid, with continued renal secretion of sodium. CPM can occur in patients with drug-related SIADH (Burcar *et al*, 1977), and SIADH has been described

in association with amitriptyline, nortriptyline, desipramine, clomipramine, dothiepin, lofepramine, tranylcypromine, thioridazine, fluphenazine, haloperidol, and carbamazepine.

A number of psychiatric conditions may be complicated by polydipsia, which may in turn lead to hyponatraemia. The literature has been thoroughly reviewed by Illowsky & Kirch (1988). Schizophrenia is the most frequent diagnosis. The prevalence of polydipsia may range from 6% to 17% of chronically ill patients, of whom 25% to 50% may develop symptoms of water intoxication. CPM has been described in a chronic schizophrenic whose hyponatraemia followed self-induced vomiting and subsequent water drinking (Haibach *et al*, 1986).

Finally, often malnourished and with a documented hyponatraemia, alcoholics are over-represented among cases of CPM (Arieff, 1985). They also constitute a group which tends to gravitate towards the psychiatric services.

We, therefore, recommend that patients with chronic mental illness, and those receiving psychoactive drugs, have periodic review of their electrolyte levels; electrolytes should always be measured on admission to hospital. This is especially important in chronic alcoholics. Where a psychiatric opinion is sought on a patient whose immediate past history includes deranged serum sodium levels, if not alcoholism or malnourishment, a diagnosis of CPM should be entertained.

Acknowledgements

The authors would like to thank those members of the several medical and surgical teams involved in the care of this patient, and also Dr P. R. Beck, Consultant Clinical Biochemist, Dr S. Beck, Consultant Pathologist, and Dr C. M. L. Smith, Consultant Neuropathologist.

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Chronic Superior Mesenteric Artery Syndrome in Anorexia Nervosa

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An 18-year-old woman presented with SMAS: an additional diagnosis of anorexia nervosa was later made. Both conditions should be considered when an adolescent presents with weight loss and vomiting.
British Journal of Psychiatry (1992), **160**, 552–554

The superior mesenteric artery syndrome (SMAS), or vascular compression of the duodenum, has been recognised for over 100 years as a cause of obstruction and gastric dilatation. The transverse portion of the duodenum lies on the vertebral bodies and is crossed anteriorly by the arch of the superior mesenteric artery, which is held away from the aorta by the retroperitoneal pad of fat. Narrowing of the aortomesenteric angle occurs when the retroperitoneal fat is lost in conditions such as burns, prolonged bed rest, rapid growth in children without corresponding weight gain, and anorexia nervosa, or when the body