CORRESPONDENCE

Central pontine myelinolysis associated with hypokalaemia in anorexia nervosa

I read with interest the article by Sugimoto et al.1 The authors report the case of a 31-year-old man with an eating disorder and hypokalaemia who was noted to have asymptomatic increased T2 signal in the central pons on brain magnetic resonance imaging (MRI), in the setting of intensive intravenous fluid rehydration. He was thought to have asymptomatic central pontine myelinolysis (CPM). Six months later, a repeat brain MRI revealed that the pontine lesion had disappeared. The authors make only a passing reference in the literature of asymptomatic and reversible CPM. However, I argue that a far more plausible diagnosis for this patient is posterior reversible encephalopathy syndrome (PRES). This condition is characterised by vasogenic oedema in the posterior circulation territories of the brain, and is classically completely reversible. PRES involving just the pontine myelinolysis associated with hypokalaemia in anorexia nervosa. J Neurol Neurosurg Psychiatry 2002;73:83–4.

Authors’ reply

We thank Dr Keswani for his interesting comments. Unfortunately, we did not do diffusion-weighted imaging (DWI) of this patient. CPM has been regarded as a disease with poor prognosis associated with underlying alcoholism or malnutrition, and it is diagnosed by neuropathological examination. Recently, however, cases with associated abnormalities in serum electrolytes and osmolality, in particular, following rapid correction of hyponatraemia have been brought to attention. With advances in diagnostic imaging such as MRI, patients with reversible or asymptomatic CPM have been increasingly reported.2 On the other hand, PRES, which consists of reversible vasogenic oedema in the posterior circulation territories, is usually associated with hypertension.3 As Dr Keswani suggested, patients with a lesion localised in the pons and few clinical symptoms have been also reported.4 Thus, the concept of CPM, which was originally a neuropathological entity, has gradually expanded while the concept of PRES, a clinicoradiological entity, has recently been proposed. This may cause some diagnostic confusion in pathologic conditions at the borderline between the two diseases, or in those with features of both diseases. DWI is expected to be useful for evaluating the pathophysiological conditions and outcome of these two diseases such as the progression of demyelination and the characteristics of the oedema.

Our patient with anorexia nervosa had electrolyte abnormalities, mainly hypokalaemia (with an almost normal serum sodium level), due to oral fluid restriction and induced vomiting. Despite gradual correction of serum electrolytes, CPM developed. Lohr, who reviewed the literature, reported that osmotic demyelination syndrome developing after correction of hyponatraemia in 89% of patients. Hypokalaemia has been found to be associated with a decreased concentration of sodium- and potassium-activated adenosine triphosphatase (Na-K-ATPase) in endothelial or glial cell membranes, as shown in skeletal muscle.5 A decrease in Na-K-ATPase activity of widespread cortical death, but rather as a nontimed in the VS (the likelihood of regaining awareness being higher after trauma, in younger patients, and falling as time passes); and its underlying pathophysiology—the VS is no longer regarded simply as a state of widespread cortical decerebration, but rather as the result of a loss of physiological coherence between a number of brain systems (sensory, motor, and autonomic). This study of the vegetative state (VS), substantially written by Dolce and Saizbon with contributions from a number of colleagues, offers an interesting Mediterranean contrast to Brian Jennett’s recent survey of the condition that he and Fred Plum christened in 1972 (Jennett B. The vegetative state. Cambridge University Press, 2002).

There is broad agreement on several key points: the clinical definition of the disorder; the diagnostic importance of dexamethasone, age, and time spent in the VS (the likelihood of regaining awareness being higher after trauma, in younger patients, and falling as time passes); and its underlying pathophysiology—the VS is no longer regarded simply as a state of widespread cortical decerebration, but rather as the result of a loss of physiological coherence between a number of brain systems (sensory, motor, and autonomic). This study of the vegetative state (VS), substantially written by Dolce and Saizbon with contributions from a number of colleagues, offers an interesting Mediterranean contrast to Brian Jennett’s recent survey of the condition that he and Fred Plum christened in 1972 (Jennett B. The vegetative state. Cambridge University Press, 2002).

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A statement on the penultimate page of text (p 436) encapsulates the reasons for my unhappiness. The author says: “However, history has shown that risk factor epidemiology can sometimes enter cycles of uninformative replications.” He is talking about one area of research, but in chapter after chapter the number of similar conditional statements combine to paint a very negative picture of overall progress in the epidemiology of schizophrenia. I do not wish to seem discouraging in my opinions, but a general sense of tasks scarcely begun pervades much of the volume, allied with some rather “pie in the sky” generalisations about the possible outcomes of future research.

The feeling of incompleteness comes as no surprise. Despite many years of multidisciplinary study, the nature and essence of schizophrenia still elude us. I would contend that this is inevitable because we lack objective markers and the illness category is so large, involving gradients of individual viewpoint and cultural pressure. In truly scientific terms, the disorder as currently conceived is virtually unresearchable, or at least research can only yield relatively banal results.

The epidemiology of schizophrenia gives a highly praiseworthy and up to date account of a multitude of concomitants of schizophrenia, whatever “schizophrenia” actually means. As a mile post of where research on these concomitants has reached, it deserves to be in every psychiatric library. But how can one come to conclusions about a “whatever”? It is only when the book touches upon modern advances in neuroinvestigative techniques and their potential application to the study of schizophrenia that my hopes begin to rise. In my personal reckoning the problems of this enormously important topic of schizophrenia will never be resolved as such. Instead we shall, if we are fortunate, see a gradual whittling away of the concept as scientists and clinicians learn to identify and measure the underlying abnormalities that are hidden by the confusing welter of concomitants. This is compatible with the standard view of the VS if the form of “contact” that the authors have in mind is basic metabolic and circulatory homeostasis mediated by the hypothalamus. That the authors have something more than this in mind is suggested by the remark that some patients in the VS enjoy “a relatively spared emotional life and contact with the inner world”. This intriguing if disquieting suggestion, and the evidence for it, is never fully explored.

The second point of disagreement is more sharply defined. While accepting the very low probability of recovery awareness after sleep deprivation, they later compare this decisions to the elimination of “lives without value” in Nazi Germany. The international debate over the proper management of patients who have little or no prospect of recovery from the VS is clearly far from over.

Although this volume contains a great deal of useful information, it is occasionally marred by puzzling passages that are likely to reflect linguistic problems.