PostScript

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 refer to other reports 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), which was originally described by Hinchey *et al* in 1996.[2] This condition is characterised by vasogenic oedema in the posterior circulation territories of the brain, and is classically completely reversible. PRES involving just the pons has been reported by several groups[3] and, although usually associated with hypertension, it has been described in association with electrolyte disturbances in normotensive patients.[4] In contrast, pontine and extrapontine myelinolysis is usually not a clinically or radiologically reversible condition, and carries a poor prognosis. Indeed, it is likely that at least a significant proportion of reported cases of ''reversible'' CPM are actually cases of PRES. Recent studies, including one by our group, have illustrated the utility of diffusion-weighted brain MRI in establishing the diagnosis of PRES.[3 5] Increased signal of T2-bright pontine lesions on diffusion-weighted imaging (DWI) suggests acute myelinolysis or ischaemia, while decreased signal on DWI is indicative of vasogenic oedema—that is, PRES. This distinction has obvious prognostic implications. Pertaining to the case of Sugimoto *et al*, diffusion-weighted MRI sequences at the time of the first brain scan would have been informative in this regard.

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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.[1 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 frequent self-induced vomiting. Despite gradual correction of serum electrolytes, CPM developed. Lohr,[5] who reviewed the literature, reported that osmotic demyelination syndrome developing after correction of hyponatraemia was complicated by hypokalaemia 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.[6] A decrease in Na-K-ATPase activity during hypokalaemia may limit the ability of a cell to preserve and/or regulate its volume in the presence of increasing osmolality, which may predispose the cell to shrinkage and injury during correction of hyponatraemia. Therefore, patients, including ours, who developed CPM in association with changes in serum electrolytes and osmolality tend to develop vasogenic oedema as a complication, and may show low-intensity signals resembling those observed in PRES on DWI. On the other hand, CPM that is consistent with the classic concept (presence of underlying disorders such as chronic liver disease and tetraplegia) has been reported to show high-intensity signals in the pons on DWI, and an associated decrease in the apparent diffusion coefficient, suggesting cytotoxic oedema occurring in active demyelination.[7] Therefore, the diagnostic usefulness of the DWI signal value for differentiating CPM from PRES should be evaluated with due consideration of the heterogeneity of the pathological mechanism of CPM. In the future, it will be necessary to have a more specific diagnosis and classification of CPM (including its differentiation from PRES). This requires a comprehensive approach consisting of a detailed history and evaluation of the clinical course, clinicoradiological assessment, and neuropathological examination.

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BOOK REVIEWS

The post-traumatic vegetative state

Edited by Giuliano Dolce G and Leon Sazbon. Published by Thieme, Stuttgart, 2002, pp 158, €39.95. ISBN 1-58890-116-5

This study of the vegetative state (VS), substantially written by Dolce and Sazbon 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 prognostic importance of aetiology, 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 death, but rather as the result of a loss of physiological coherence between a number of brain systems (sensory,