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,

motor, and reticular) that must work in concert to generate awareness.

There are two notable points of disagreement. The first may be semantic. The authors argue that although contact with the ''outer world'' is lost in the VS, contact with the ''inner world'', mediated by phylogenetically ancient systems, survives. 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 recovering awareness after spending one year in the VS, and the even lower probability of a good functional outcome, the authors describe the decision to withdraw nourishment in such patients as ''an act of active abandonment''. They later compare such 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.

A Zeman

The epidemiology of schizophrenia

Edited by Robin M Murray, Peter B Jones, Ezra Susser, Jim van Os, and Mary Cannon. Published by Cambridge University Press, Cambridge, 2003, pp 445, £55.00 (hardback). ISBN 0-521-77540-X

As a trainee in psychiatric epidemiology 40 years ago, I fervently wished for two things; firstly help to carry out endless statistical calculations and, secondly, a scientifically valid definition of schizophrenia. To a considerable extent computers have fulfilled the former wish. Sadly, neither they nor anything else has come close to achieving the latter, as the present volume repeatedly demonstrates.

This book is well edited, on the whole well written, and its contributors are eminent in their field. It is impressive in the width of its coverage and it provides a good overview of the discipline's status at the beginning of the new millennium. Then why, after having read through to the end, do I put the book down with a considerable sense of sadness?

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 combines 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 over-inclusive and so vulnerable to vagaries 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 disorders that make up such a heterogeneous whole.

How can anyone leave this book feeling happy when one reads (p 44) that ''Based on meta-analyses of a large literature on schizophrenic outcomes, improvement in the course or outcome from beginning to end of the 20th century are modest.''? What a condemnation of psychiatry and of society! But how can psychiatry expect society to provide proper amenities for sufferers when we cannot even describe adequately what it is we are dealing with?

I hope that readers of this book from the wide range of neurosciences may be inspired to collaborate in the introduction of cutting edge technologies into the investigation of schizophrenia. To misquote the old song which said ''What is this thing called love?'', it must be a first priority to find out ''What is this thing called Schizophrenia?''

A Munro

Handbook of neurological rehabilitation, 2nd edition

Edited by Richard J Greenwood, Michael P Barnes, Thomas M McMillan, and Christopher D Ward. Published by Taylor & Francis Books Ltd, Hove, 2003, pp 725, £120.00 (hardback). ISBN 0-86377-757-0

This is the second edition of the book that many UK rehabilitation physicians have come to regard as the bible of neurological rehabilitation. The book is primarily targeted towards neurologists, but has a much wider readership in multiprofessional rehabilitation teams. In addition to the four editors, there are some 93 contributors from a range of different professional backgrounds. Among them are many of the most respected authors in the field of rehabilitation and neurology.

As in the previous edition, the book is laid out in three main sections: principles of practice, exploring the epidemiological, social, and political principles of practice in rehabilitation as well as mechanisms of neurological recovery; assessment and treatment of functional deficits, covering the characteristics and remediation of physical, cognitive, affective, and behavioural disorders; and specific disorders, highlighting the use of rehabilitation techniques in the context of specific disease including head injury, stroke, spinal injury, progressive neurological disorders, and disorders of muscle and the peripheral nervous system.

For those familiar with the first edition (published in 1992), it has to be said that some chapters could have benefited from a slightly more radical face lift. In the section on environmental control systems, for example, the use of illustrations (unchanged from the previous edition) showing equipment now obsolete for some years belies some of the exciting developments in the field of computers and assistive technology that have occurred during the past decade. On the other hand, Derick Wade's completely revised chapter on stroke now provides a masterly summary of the evidence for effective management, assembled into clear evidence tables, for those readers who wish to explore the background literature.

In a world where we increasingly turn to the internet for reference and education, it is easy to overlook the advantage of far more instantaneous access to information afforded by a well written textbook. This book constitutes an excellent source reference, which no one with a serious interest in rehabilitation should be without.

L Turner-Stokes