Correspondence

Treatment of SIADH with isotonic saline

Sir,

Drs Musch and Decaux report on the beneficial effects of isotonic saline infusion in a sub-group of patients with hyponatraemia due to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).¹ Though of scientific interest, there are considerable potential problems and dangers in such active treatment of SIADH, which the authors do not address.

The first concerns the recognized association between rapid correction of hyponatraemia in SIAD with osmotic demyelination syndromes (notably central pontine myelinolysis).² This complication is most likely when the rise in plasma sodium (Na) is greater than 10 mmol/24 h.³ Though in the series presented by Drs Musch and Decaux, the rises in plasma Na were generally small, there was a more responsive sub-group of patients with relatively low plasma osmolality levels, and more prolonged saline treatment could be potentially hazardous in such patients.

The second question is whether treatment is needed in these patients at all. Most cases of SIADH remit spontaneously, and even in chronic cases (due for example to untreatable malignancies), active management may not be necessary or wise unless plasma Na levels are especially low or the patients is deemed symptomatic from the hyponatraemia.⁴

Hyponatraemia is frequently benign and asymptomatic, and active management of hyponatraemia per se is usually unnecessary, as the condition often remits spontaneously. Management of the underlying disease process, where possible, is the prime target of management.

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References


Fatigue syndromes

Sir,

The paper by Pentilla et al. on cytokine dysregulation in post-Q-fever fatigue syndrome¹ not only provides evidence that when this syndrome follows Q fever it is causally related to rickettsial infection, but also illustrates the way in which methodologically nominalist definitions allow the disease terminology to be used in diagnostic statements concisely and without unwarranted implications.²

‘Chronic fatigue syndrome’, defined in clinical-descriptive terms, should convey no causal implication; when there is convincing evidence of a causal factor, the case belongs to a causally-defined subset of this syndrome, and in the diagnostic statement this factor should take precedence. ‘Post-Q-fever fatigue syndrome’, with the acceptable acronym QFS, conforms to this desideratum. The use of this term leaves no doubt that the findings are relevant to only one sub-set of CFS, with no implication that they are applicable to all cases of this syndrome. Of course, they lead to a series of further questions about this subset, such as the mechanisms by which the symptoms are produced and the possibility of specific therapeutic approaches. About these, testable hypotheses can be advanced.

This contrasts with the confusion caused by the thoughtless acceptance of the unjustified term ‘myalgic encephalomyelitis’ (ME) as synonymous with CFS. This term can properly refer only to some sort of inflammatory process affecting the brain and spinal cord and associated with muscle pain. Since no such change has been demonstrated in any instance, its use can imply only the belief that the central nervous system is affected by changes of this

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