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NEWS AND NOTES

SUPPLEMENT

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CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Hypoalbuminaemic hyponatraemia: a new syndrome?

SIR,—The article by Dr P Dandona and others is certainly debatable (2 November, p 1253). They describe six patients with hyponatraemia—three in some detail—and conclude that the association with hypoalbuminaemia is new. However, it is difficult to decide exactly what is new.

Is the association of hyponatraemia and hypoalbuminaemia new? This combination is normally associated with clinically detectable oedema and is well described[1]; the physiological explanation is available and quoted in the article. If the suggestion is that certain patients with hypoalbuminaemia, but without oedema, develop hyponatraemia through an unknown mechanism care should be taken to exclude patients with other potential non-osmotic stimuli to antidiuretic hormone. All three detailed cases have an additional explanation for non-osmotic stimulation.

Is the association of hypoalbuminaemia, hyponatraemia, and cerebral symptoms, corrected by restoration of plasma sodium, new? In two of the detailed cases (cases 1 and 2) hyponatraemia was not a feature until after admission, suggesting iatrogenic induction by inadequate monitoring of fluid balance. Subsequent symptoms of confusion are then perhaps not surprising if induction of the hypo-osmolal state was rapid.[2]

Is the response of plasma sodium to albumin infusion new? The restoration of normal plasma sodium has previously been observed in patients with the nephrotic syndrome.[3] It is only a palliative treatment if steps are not taken first to reverse the underlying physiological cause of water retention.

The association between hypoalbuminaemia and hyponatraemia through inspection of laboratory records is tenuous. While suggesting the association should be evaluated critically, the authors ignore their own advice. There is no more value in causally associating hyponatraemia with hypoalbuminaemia than say hyponatraemia with a raised erythrocyte sedimentation rate.

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1 Anderson RJ, Chung H-M, Kluge R, Schrier RW. Hyponatremia: A prospective analysis of its epidemiology and the pathogenic role of vasopressin. *Ann Intern Med* 1985;**102**: 164-8.
2 Arieff AI, Llach F, Massry SG. Neurological manifestations and morbidity of hyponatremia: correlation with brain water and electrolytes. *Medicine* 1976;55:121-9.
3 Davison AM, Lambie AT, Verth AH, Cash JD. Salt-poor human albumin in management of nephrotic syndrome. *Br Med J* 1974;i:481-4.

SIR,—The syndrome described by Dr P Dandona and others is identical with that complicating parenteral nutrition and leading to excessive tissue oedema and multiple organ failures.[1] Any of their cases would also meet the criteria for the transurethral resection syndrome, and their syndrome is also little different from the "Pitressin and glucose" syndrome known in obstetrics. Any of these syndromes may present as shock lung, brain oedema, or heart, renal, or hepatic failure. Detecting a change in osmolality requires careful timing before endogenous osmolar "adaptation" takes place. Although some of these patients, and those of the authors, are described as "hypovolaemic," they are not fluid depleted. The increase in weight in such patients, due to fluid retention, may be up to 4-7 kg.[1-3]

Another version of the syndrome, in which hypoproteinaemia may be associated with normal sodium and osmolality, is that which occurs as a result of excessive saline infusion. Such infusion may be inappropriately given during surgery to correct hypotension that may or may not be caused by blood or fluid loss. This syndrome may present as shock lung or diffuse capillary bleeding occurring during or soon after surgery. In spite of massive fluid overload, the central venous pressure may drop and remain low after a transient rise.[3]

This is a point that is hard to sell: hypervolaemia may lead later to hypovolaemia, hypoalbuminaemia, and a low central venous pressure because of the escape of plasma into the interstitial space. It is a well documented response to saline loading in animals,[4] as well as volume overload in humans.[1-3] The development of diuresis, natriuresis, hyponatraemia, hypervolaemia, hypovolaemia, and hypo-osmolality depends not only on the type of fluid infused but also on the ability of the heart and kidneys to handle such massive fluid overload.

The high level of antidiuretic hormone may occur earlier due to pain, infection, drugs, or anaesthesia, and as a response to the early hypovolaemia. This is a syndrome of the "oedematous hypovolaemic hypo-osmotic" patient. Its true pathology is due to alterations of the cellular and the capillary membranes, and the best management is prevention. Before discussing further pathogenesis or management of such syndromes let us straighten up some physiological issues such as Starling's law and osmolality. This may transfer the management of these syndromes from a "hit and miss" situation into a scientifically well understood medicine.

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1 Watters DAK, Chamroonkul S, Griffith CDM, *et al.* Changes in liver function tests associated with parenteral nutrition. *J R Coll Surg Edin* 1984;**29**:339-44.