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ESPS Peer-review Report
Name of Journal: World Journal of Clinical Cases
ESPS Manuscript NO: 2742
Title: Desmopression for Hyponatremia Overcorrection: an Effective Adjunct Treatment for Reversing Rapid Overcorrection and Preventing Osmotic Demyelination Syndrome.
Reviewer code: 01200577
Science editor: Zhai, Huan-Huan
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	
[ ] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[Y] Minor revision
		[ ] No records	[ ] Major revision

COMMENTS TO AUTHORS

I think that the paper is interesting and mostly well described. However, there are some aspects to clarify before accepting it for publication. In particular: Clinical case I'd like to know: - How hypokalemia was treated in ED and if there were ECG alterations. - The volume of dextrose administrated and how the authors evaluated the correction rate of hyponatremia (by a specific formula?) Finally, I'd like to notice that chlorpromazine and thorazine are the same drug.. Discussion I think that this section lacks of a complete discussion on the pathogenetic mechanisms underlying the clinical picture. In fact, the authors focused their attention on osmotic demyelination syndrome and its prevention, but it could be equally useful to discuss the causes of hyponatremia in this patient, which, in turn, may explain the subsequent clinical evolution. In particular, it is important to underline that at admission the patient presented a clinical picture suggestive of SIAD (i.e. inappropriate anti-diuresis syndrome), which, in the author hypothesis, should have been triggered by vomiting, a known cause of SIAD (see also Ellison DH, N Engl J Med. 2007 May 17;356(20):2064-72. Esposito P, Nephron Clin Pract. 2011;119(1):c62-73). As SIAD seems a possible diagnosis of the initial clinical presentation, the following evolution is suggestive of central diabetes insipidus, a disorder caused by suppression in ADH secretion. This eventuality was further proved by the quickly effect of desmopressin, which, indeed is a part of the therapy of diabetes insipidus. Therefore, the authors should take into consideration the role of ADH as a main pathogenic mechanism. I think that such discussion could make the paper more complete.