EDITORIAL

Dyselectrolytemia as a Predictor of Prognosis in Subarachnoid Hemorrhage: In the Clink or Still in the Dock?

Harsh Sapra

Indian Journal of Critical Care Medicine (2019): 10.5005/jp-journals-10071-23313

Subarachnoid hemorrhage is an acute neurological emergency, which despite available treatment, carries an in-hospital mortality as high as 18–20% and a great deal of morbidity in terms of poor functional outcome. While the primary disease severity in combination with the neurological complications, such as rebleeding and delayed cerebral ischemia, has been a major contributor to mortality, medical complications have been extensively proved to be adding to the number of patients who die or are left with neurologic sequelae. Medical complications like fever, anemia, and hyperglycemia have been the proven culprits for the poor disease prognosis.[1] However, dyselectrolytemia as a predictor of poor outcome has been in the dock for quite some time now, and the prosecution still continues. The reason for this has been the conflicting results of dyselectrolytemia on subarachnoid hemorrhage (SAH) prognosis in different clinical studies conducted worldwide.[1–4]

In the present issue, we present a retrospective study on the impact of dysnatremia and dyskalemia on the prognosis in SAH patients. The authors have concluded that hypernatremia, but not hyponatremia, in SAH is associated with poor outcomes, and that neither hypo- nor hyperkalemia has a significant effect on the patient's prognosis.

Many studies conducted in the past have reported a definite adverse effect of electrolyte abnormalities on the outcome in SAH patients. In 2002, Qureshi et al. retrospectively analyzed 298 patients across 54 neurosurgical centers in North America, to determine the prognostic significance of serum sodium concentration abnormalities. They concluded that hypernatremia after SAH was independently associated with poor outcomes, although neither hypo- nor hypernatremia had any association with the risk of symptomatic vasospasm.[5] In contrast to these findings, Chandy et al. reported an association between hyponatremia and cerebrovascular spasm in SAH. They reported that hyponatremia (fall in sodium levels >4 mEq/L from the admission levels) was associated with a significantly greater risk of developing vasospasm and might precede cerebrovascular spasm by at least one day, a finding later supported by many studies.[6]

Alimohamadi, in 2016, described that even though hyponatremia was the most common electrolyte abnormality after aneurysmal SAH, it was hypernatremia in the subacute phase that was significantly associated with poor outcome. These researchers emphasized that the time course of development of such abnormalities might also be important in their eventual impact on the outcome and that dyselectrolytemia occurring in the subacute phase correlated with poor outcomes.[7] Besides dysnatremias, they also stressed that both hypokalemia and hypomagnesemia were predictive of adverse outcomes, a finding not very widely supported by previous studies.[8]

Clinical experience points to the fact that occurrence of hypernatremia is ominous in neurosurgical patients. The secondary brain injury that ensues due to the cellular dehydration, myelin damage, and neuronal death is irreversible, and the kidney injury accompanying raised sodium levels contributes to overall patient morbidity. Thus, hypernatremia in a patient with SAH clearly indicates a poor outcome.

The question of whether the timely and appropriate management of these abnormalities may improve the treatment results of SAH has yet to be answered. No particular therapeutic modality or protocol in either preventing or treating hypernatremia has been studied so far.[9] Most studies demonstrating a relationship between hypernatremia and poor outcome are retrospective in nature, and so is the present one. Additional research is therefore needed to evaluate if maintaining normonatremia actively improves neurological outcome in SAH.

Department of Anesthesiology, Medanta: The Medicity Hospital, Gurugram, Haryana, India

Corresponding Author: Harsh Sapra, Department of Anesthesiology, Medanta: The Medicity Hospital, Gurugram, Haryana, India, Phone: +91 9650898577, e-mail: harshsapra@hotmail.com

How to cite this article: Sapra H. Dyselectrolytemia as a Predictor of Prognosis in Subarachnoid Hemorrhage: In the Clink or Still in the Dock? Indian J Crit Care Med 2019;23(12):545–546.

Source of support: Nil

Conflict of interest: None

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