Hyponatremia and cerebral vasospasm following subarachnoid hemorrhage

Cerebral vasospasm resulting from aneurysmal subarachnoid hemorrhage is a problem that continues to plague both patients and physicians alike. Despite considerable research dedicated to the subject over the past several decades, cerebral vasospasm continues to be a leading cause of morbidity and mortality for those patients who survive the initial insult of a ruptured intracerebral aneurysm. While the ultimate goal should be the elimination of vasospasm altogether, significant effort has been focused on predicting which patients will develop symptomatic vasospasm, with the obvious hopes of limiting the severity and thus the deleterious effects of vasospasm. The authors of this study provide evidence that low sodium levels may be a harbinger for the development of cerebral vasospasm.\(^1\)

The idea that we may more accurately predict cerebral vasospasm by detecting drops in serum sodium levels is intriguing. The association between hyponatremia and cerebral vasospasm has been previously examined, however, the conclusions remain equivocal.\(^2\) What is important to remember is that there is a differentiation between radiological vasospasm and clinically symptomatic vasospasm, with up to 70% of patients experiencing...
radiographic vasospasm, while only 20-30% experience symptomatic vasospasm.\textsuperscript{[3]} The authors of this study used transcranial doppler (TCD) to detect the presence of cerebral vasospasm and thus their results suggest an association between hyponatremia and what can best be described as radiological vasospasm. Qureshi et al. found no correlation between low sodium levels and cerebral vasospasm, however they looked at what is perhaps the more relevant parameter of clinical vasospasm, using previously established criteria.\textsuperscript{[2,5]}

There are several limitations to this study as the authors discuss. This study is subject to the well established limitations of a retrospective, single institution series. Additionally, the definition of vasospasm with only TCD and the irregular application of TCD screening remain limitations. The paucity of data concerning sodium intake and administration of different fluids introduce potential bias among groups. The absence of endovascular management in this series may weaken the potential application of this data to contemporary patient populations where endovascular therapy is an important treatment modality for patients harboring ruptured intracranial aneurysms. It would be interesting to note if treatment modality and location of aneurysm (for example, anterior communicating artery) influence this observed association of hyponatremia with cerebral vasospasm.

The authors’ suggestion that there is a correlation between hyponatremia and cerebral vasospasm is interesting and potentially important and therefore provides impetus for further careful investigation.

R. Webster Crowley, Aaron S. Dumont
Department of Neurological Surgery, University of Virginia School of Medicine, Box 800212, Charlottesville, VA 22908, USA. E-mail: asd2f@virginia.edu

References