Brain sag

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Alaraj and colleagues1 report on 5 patients who, following the surgical repair of ruptured anterior communicating artery (ACoA) aneurysms aided by spinal drainage with or without fenestration of the lamina terminalis, demonstrated clinical “brain sag” with classic CT features: elongation of the midbrain and effacement of the basal cisterns. In all cases the angiogram obtained during the “sag episode” showed inferior displacement of the basilar artery when compared with the “pre-sag” angiogram, and in 3 cases, this displacement was so severe that the basilar artery kinked. Vertebral artery kinking was seen in some cases as well. In 4 of the 5 cases, brain sag was recognized in a timely fashion, and patients responded favorably to conventional maneuvers, most notably placement in the Trendelenburg position. In 3 of the 4 patients who improved with positional changes together with a blood patch or clamping of the spinal drain, a “post-sag” angiogram showed marked improvement in the displacement of the basilar artery. The fourth patient whose condition improved did not undergo post-sag angiography, as it was not deemed clinically necessary. The authors are careful to note that this angiographic picture was not seen in a similar population of patients lacking clinical brain sag who were being studied for vasospasm.

While the authors’ series is small, it is carefully documented and provides dramatic evidence that the clinical symptoms of brain sag may not be solely due to mechanical displacement of the brainstem. Instead, regional posterior circulation cerebral ischemia may be a contributing factor. The fact that a surprisingly high percentage of their patients also exhibited early signs of vasospasm leads one to wonder whether brain sag is more common but becomes symptomatic only when there is concomitant vascular compromise. My initial impression, based on routine examination of early postsurgical CT scans, is that it is decidedly rare to see dramatic CT evidence of sag in an otherwise stable patient, although a more systematic review is needed. Such a review should clearly focus on ACoA aneurysms treated with spinal drainage.

At my institution, our initial impression was that the combination of lamina terminalis fenestration and spinal drainage predisposes to brain sag,2 but maybe it is more a function of the subarachnoid dissection in this region given that ACoA aneurysm rupture often may have already rendered the ventricles in communication with the suprasellar cistern. In any case, since we recognized this phenomenon some years ago, we have kept all patients with spinal drainage and lamina terminalis fenestration flat in the immediate postoperative period, and we are quick to place a patient in the Trendelenburg position in the rare instance that it is needed. Using this approach we rarely need to resort to blood patch, and no patient has exhibited obvious irreversible deficits from the sag episode.3 That said, we have not looked to see whether patients with brain sag were more likely to develop delayed cerebral infarction from vasospasm or neurocognitive symptoms with delayed follow-up. While assuming the Trendelenburg position, many patients may have experienced increased systemic blood pressure, as vasospasm may have been in the differential diagnosis. Whether induced hypertension led to a more rapid reversal of symptoms was not examined.

In conclusion, the proceeding paper is important because it documents for the first time the angiographic features of brain sag and leads us to reexamine what we think we know about its underlying pathophysiology, as well as how best to prevent it, treat it, and determine its true sequelae.

Disclosure

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References


Editorial

See the corresponding article in this issue, pp 586–591.
Editorial

Response

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We thank Dr. Connolly for his insightful comments regarding our paper. He proposes the intriguing possibility that the vascular displacement visible on angiography in patients with brain sag may itself contribute to the symptom complex associated with this phenomenon. Although certain findings, such as unilateral third nerve palsy, correlate more specifically with mechanical compression, the contribution of posterior circulation ischemia to brainstem dysfunction is certainly an additional consideration. With regard to the management of this condition, we have also adopted a preventative strategy postoperatively, keeping patients with intraoperative spinal drainage (including those with attempted but unsuccessful spinal drain placement) flat for the first 12–24 hours. With this simple modification in the postoperative protocol, anecdotally we have encountered a marked decline in the incidence and severity of this complication. The key to managing brain sag remains prompt recognition and immediate maneuvers to reverse the condition once suspected.

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