Lateral sinus thrombosis: antithrombotic therapy and the issue of management of cerebellar swelling

TO THE EDITOR: We read with great interest the article by Apra et al.1 (Apra C, Kotbi O, Turc G, et al: Presentation and management of lateral sinus thrombosis following posterior fossa surgery. J Neurosurg [epub ahead of print February 26, 2016. DOI: 10.3171/2015.11. JNS151881]) concerning sinus thrombosis after posterior fossa surgery. The authors retrospectively analyzed 180 posterior fossa surgical procedures for removal of different space-occupying lesions at a single institution between January 2008 and May 2014; they focused their study on cases of postoperative lateral sinus thrombosis (LST). The authors clearly elucidated the incidence and risk factors associated with this entity and properly concluded that well-tolerated postoperative LST in asymptomatic patients should be managed conservatively.

Management of sinus thrombosis following posterior fossa surgery is a topic of debate in the neurosurgical literature, and clear therapeutic guidelines do not exist. Therefore, we would really like to thank the authors for their contribution and for having shared their experience in the treatment of asymptomatic patients. What appears to be more intriguing is how to treat those rare cases of patients who become symptomatic and exhibit clinical worsening due to cerebellar swelling. Indeed, sinus thrombosis associated with acute cerebellar swelling represents a life-threatening condition that may induce rapid neurological deterioration that is reversible only by prompt and adequate treatment, such as emergency posterior fossa decompressive craniectomy.2,5,7

Our contribution to the discussion comes from the following illustrative case. A 55-year-old woman presented to our institution with a large left petroclival meningioma (Fig. 1A) causing brainstem compression. Surgery aimed at subtotal resection, and optimal brainstem decompression was performed through a retrosigmoid approach. In the early postoperative course, the patient did not present any new neurological deficit, apart from slight diplopia due to manipulation of cranial nerves IV and VI. LST was ruled out on MR images obtained on postoperative Day 1 (Fig. 1B). However, on the 2nd postoperative day, the patient developed headache, nausea, and photophobia, and her level of consciousness had deteriorated since the previous day; hydrocephalus was diagnosed, and a ventriculoperitoneal shunt was immediately placed. Despite shunting, she did not regain her neurological status of the first postoperative day and remained in a stuporous condition that was progressively worsening. New MRI and CT scans disclosed a left LST and severe cerebellar swelling with effacement of the fourth ventricle and basal cisterns (Fig. 1C–E). A wide posterior fossa decompressive craniectomy with brainstem decompression was immediately performed, allowing for reappearance of the fourth ventricle and basal cisterns (Fig. 1F); this procedure stopped neurological deterioration and likely saved the patient’s life.

Apra et al. confirmed the general feeling that antithrombotic therapy may increase hemorrhagic risk. In fact, they found that the incidence of surgical complications, second-look surgeries, and surgical bed hemorrhage was higher in the group of patients who received antithrombotic therapy than in those who did not (56.2% vs 27%, 37.5% vs 13.4%, and 18.8% vs 10.4%, respectively).1 Conservative management and a strict follow-up therefore appear to be the best option for asymptomatic patients.

However, in our view, attention should be focused on the symptomatic group. In fact, in symptomatic patients, the issue of emergency cerebellar decompression related to acute cerebellar swelling requires further debate. We believe that the real challenge for the surgeon is to recognize among the population of patients with postoperative LST those few who will develop cerebellar swelling, given the fact that posterior fossa sinus thrombosis and acute cerebellar swelling may amount to a vicious circle, where one condition leads to another, and vice versa. In such cases, the development of a posterior fossa “compartment syndrome” leads to a sudden local increase in intracranial pressure,2,4,6,10 which should be properly managed with very early surgical decompression in order to prevent brainstem damage (Fig. 1C and E). The use and the effects of antithrombotic therapy in these critical patients deserve maximal attention and debate.

Cerebellar swelling after posterior fossa surgery may develop even in absence of LST;4 in fact, the exact percentage of patients with postoperative LST who will develop cerebellar swelling is unknown. Certainly, LST appears to be a significant risk factor. But, if this is the case, should we look for LST with an early and dedicated postopera-
tive MRI sequence in all patients during the postoperative course? And, if yes, when, given that thrombosis may not be evident on MRI study obtained on the 1st postoperative day as happened in our case? Should we base this diagnosis on MRI findings only or should we consider any other examination?

Furthermore, if thrombosis is detected, should we immediately start high-dosage antithrombotic treatment aiming at sinus recanalization or should we give the patient a “usual” low dose of enoxaparin (e.g., 4000 UI once per day) and wait to see what happens to the cerebellum? Apra et al. showed that high-dosage antithrombotic therapy required a long period of time for sinus recanalization (272 ± 23 days). Is it of paramount importance to start it early? Moreover, what is the gold-standard dose of enoxaparin for postoperative posterior fossa surgery?

Conversely, if the course of LST is not a significant predictor of cerebellar swelling, should we avoid high doses of antithrombotic drugs to prevent hemorrhagic complications? The rate of second-look surgery for hemorrhagic complications in the series by Apra et al. was almost 20%; this seems much higher than our posterior fossa surgery series of the last 5 years or other series available in the literature, with an incidence around 5%. Could this difference be attributed to a different attitude toward the administration of antithrombotic therapy at our institution? Indeed, the safe/unsafe dose limit for antithrombotic therapy in neurosurgery is not clear.

Different attitudes and approaches to posterior fossa sinus thrombosis among high-volume centers highlights the importance of the topic addressed by Apra et al. and how posterior fossa surgery is still a “craft work” rather than a standardized procedure. Further studies involving many centers that share common protocols of postoperative imaging and management are required to further clarify these important issues.

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References