Arachnoid cyst and the hemorrhagic complications of open decompression


The best treatment approach for intracranial arachnoid cysts is considered controversial in current neurosurgical practice. Most neurosurgeons prefer endoscopic or microscopic fenestration over shunt insertion as the method of choice for the initial treatment modality for symptomatic arachnoid cyst, and if this treatment modality fails, CSF shunting is considered as the next treatment option.

The main reason for favoring fenestration over shunting is to avoid ending up with a shunt-dependent patient. Despite this rationale for avoiding CSF shunting as the first treatment step for symptomatic arachnoid cyst, however, we have observed during our practice in treating this condition that open fenestration of arachnoid cysts is associated with high rates of complications and a high failure rate. In fact, this observation is also reported by others. For example, Choi et al. recently published a report of 75 patients who underwent surgical treatment (fenestration) for intracranial arachnoid cyst, with a 36-month median follow-up period. They reported a successful rate of only 28% (21 patients) and a total of 35 complications occurring in 28 patients (37%).1 Nevertheless, some authors documented a better outcome with intracranial arachnoid fenestration; however, it is undeniable that some complications of this kind of treatment are extremely serious (such as postoperative intraparenchymal hemorrhage).1,2

Intracranial arachnoid cyst is considered a congenital condition, although there are reports of intracranial cysts that developed spontaneously de novo or after trauma.7 Based on several observations, this condition may in fact represent a kind of CSF dynamic disorder. It seems that patients harboring an intracranial arachnoid cyst develop with time some new CSF pathways and a unique dynamic balance between the cyst and the other CSF compartments. This may explain why most arachnoid cysts are asymptomatic. It also explains why some cysts increase in size, decrease in size, or appear spontaneously; why sometimes the symptoms are just transitory; and why shunting sometimes leads to unusual CSF dynamic phenomena.4–7

If intracranial arachnoid cyst represents a CSF dynamic disorder, it is rational to assume that these lesions should also be associated with cerebral perfusion and cerebral autoregulation disorder (because both the CSF dynamic and cerebral blood flow are connected through the underlying physiology described in the Monro-Kellie doctrine).6 It has been shown that the Monro-Kellie doctrine operates not only to prevent critically high intracranial pressure (ICP) but also to prevent critically low ICP.6 This concept may be important in explaining the hemorrhagic complication reported by Auschwitz et al. The sudden decompression of the cyst (sudden removal of a large volume of CSF) might cause the ICP to become critically low, and as a result the blood is accumulated intracranially as a compensation mechanism. This whole process can be looked at as a kind of acute ICP homeostasis failure that happened because of the acute decompression of the cyst.

That being said, one of the important questions regarding the optimal treatment of intracranial arachnoid cyst is how much we can interfere with the well-established CSF dynamic balance during the course of the treatment. Thus, we also need to know the best way to ensure that our intervention remains under control. Shunting may be the best answer for these questions, especially with the advances in shunt technology (programmable shunt) and the advances in imaging guidance that may enable us to make the shunt procedure for treatment of intracranial arachnoid cyst even more minimally invasive, which may represent another important advantage over fenestration. Therefore, perhaps we should change our current popular paradigm of treatment strategies of intracranial arachnoid cyst and move from fenestration toward shunting as the initial treatment modality for the symptomatic intracranial arachnoid cyst, or maybe we should refine our surgical technique of open arachnoid cyst decompression in a way that respects the underlying abnormal CSF dynamic.

Yet, in addition to conducting well-designed randomized clinical studies for CSF shunting versus fenestration, we strongly believe that the ultimate answer regarding the best treatment for intracranial arachnoid cysts requires conducting more research efforts that aim to achieve a better understanding of the changes in CSF pathways that develop in patients with intracranial arachnoid cysts, and how these pathways are affected by fenestration and
shunting. This may give us more insights into why some patients develop symptoms, the reason for some serious complications after fenestration, and why some patients become shunt dependent.

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DISCLOSURE
The authors report no conflict of interest.

References

Hemorrhagic infarction following open fenestration of intracranial arachnoid cyst

TO THE EDITOR: Having read with interest the recent article by Auschwitz et al.1 (Auschwitz T, DeCuypere M, Khan N, et al: Hemorrhagic infarction following open fenestration of a large intracranial arachnoid cyst in a pediatric patient. J Neurosurg Pediatr 15:203–206, February 2015), we would like to make a few comments based on our own experience with the same pathology.3,4

First, endoscopic cystoventriculostomy is recommended in the treatment of symptomatic arachnoid cyst, and we think that this technique is the gold standard in the treatment of intracranial arachnoid cyst. Endoscopic treatment maintains the basic strategy of cyst fenestration into the lateral ventricle or basal cisterns without the invasiveness of open craniotomy. The procedure is simple, effective, and minimally invasive; it saves operative and recovery time; and it is associated with low morbidity and mortality rates.2 In our opinion, craniotomy is only indicated in cases of complicated arachnoid cyst with an intracystic hemorrhage, or with rupture into the subdural space causing brain compression and midline shift. We agree with the authors that craniotomy with fenestration produces a rapid reduction in intracranial pressure leading to changes in the cerebral circulation and resulting in cerebral hemorrhage.

Second, the authors believe that by implanting a shunt it is possible to progressively reduce intracranial pressure. We fully agree with this statement, but today we do not consider the shunt a good treatment option because it eventually presents numerous avoidable complications.5

Finally, the authors state that “subdural hematomas and hygromas have rarely been reported as complications of arachnoid cyst and subdural hematomas are widely reported throughout the literature.” However, in our experience it is not unusual for bruising to occur in low-intensity trauma, causing subdural hygromas or chronic subdural hematoma, especially in young patients.

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Response
We appreciate the comments of Salma et al. regarding our case report. Their theoretical discussion of a possible scenario that might occur with sudden decompression of the brain (i.e., drainage of a large cyst) is interesting; however, there are other pathological conditions, both acute and chronic, in which surgical treatment results in the same phenomenon—a rapid decompression of pressure in the brain—such as removal of large clots, abscesses, or drainage of a large tumor cyst. Fortunately the complication of hemorrhagic infarction, as seen in this scenario, is very rare, but that makes our ability to understand the risk factors difficult.

We have hypothesized that when an arachnoid cyst warrants treatment and when there are signs of significantly increased ICP present, using an approach that would al-