Embolization of Orbital Arteriovenous Fistulas


Abstract

Purely intraorbital arteriovenous fistulas (AVFs) are rare, and their clinical management is controversial. The authors successfully treated a patient with an intraorbital AVF by transvenous embolization alone. An accurate distinction between arteriovenous malformation (AVM), which is characterized by the existence of a nidus, and an AVF, which has no nidus, is important and requires superselective ophthalmic artery angiography. Treatment of an intraorbital AVF by transvenous embolization can improve visual function.

The authors assert that transvenous embolization is the preferred method of treatment for orbital AVFs and advocate a percutaneous approach via the jugular and/or femoral veins. Direct surgical visualization and catheterization of the superior ophthalmic vein (SOphV) for the treatment of a carotid–cavernous fistula are well described,1 and Deguchi and colleagues note the utility of this approach in such instances. However, they state that direct access to the SOphV, which is usually markedly dilated in the presence of an orbital AVF, should not be undertaken because of the technical challenge of percutaneous direct puncture and the risk of orbital hemorrhage. They also suggest that venous tortuosity within the orbit would not permit adequate catheter access to allow embolization of the orbital AVF. We respectfully disagree with these statements and wish to draw the authors’ attention to our work in this area.

We recently reported a case of isolated orbital AVF treated with coil embolization via the SOphV approach.2 Percutaneous access to the AVF via the inferior petrosal sinus and facial veins was unsuccessful due to venous channel tortuosity; therefore, surgical identification and microcatheterization of the SOphV through an eyelid crease incision was performed. Under transarterial and transvenous roadmap guidance, the catheter was routed through the SOphV into the inferior ophthalmic vein (IOphV). The catheter tip was placed in the anterior IOphV, and the fistula was embolized with platinum detachable coils. Intraoperative transarterial angiography showed that there was no blood flow through the lesion. The patient’s diplopia and proptosis resolved, and complete closure of the fistula was confirmed on repeated angiography 3 months later. In contrast, Deguchi and colleagues were unable to reach the shunt in their patient and attempted to close the fistula by IOphV embolization; a second procedure was then required to close the SOphV upon recurrence of the patient’s symptoms.

We agree with Deguchi and colleagues that transvenous embolization of an orbital AVF is preferable to transarterial and/or surgical treatment because it appears to carry the lowest risk of vascular compromise and vision loss. However, we believe that the direct SOphV approach may permit microcatheter access directly to the lesion itself with a high likelihood of success and minimal risk of orbital complications. We recommend that the direct SOphV approach be considered as a means of definitive endovascular treatment of an isolated orbital AVF.

References


Response: We thank Dr. Subramanian and his colleagues for their valuable comments on our case report. We agree that the direct SOphV approach is one of the effective approaches for intraorbital and cavernous dural AVFs, but we do not think that the direct SOphV approach should be chosen prior to attempting the facial vein approach. When using the direct SOphV approach, blood flow through the SOphV is disturbed. As this is probably the sole source of drainage from this area, we fear the possibility of transient glaucoma and increased reflux to the cortical veins induced by the resulting elevation in SOphV pressure. We also hesitate to say that the SOphV approach is always easy: the dilated SOphV cannot always be found in the superficial layer under the eyelid, even with the aid of a microscope. Moreover, the direct SOphV approach has potential complications, such as hemorrhage resulting from surgical cut down, venous puncture, or rupture of the SOphV, as well as damage to the trochlea or other orbital structures, and infection.

Because the facial vein approach involves minimal disruption of drainage from the SOphV, we believe this approach should be attempted first. Subramanian and colleagues performed the direct SOphV approach after failure of the facial vein approach.

We think that the position of the tip of the author’s microcatheter in the IOphV is in the same location that we used for our catheter (as measured from the point of the inserted coils). In their case, because the AVF flowed directly into the IOphV, they were able completely to obliterate the lesion only by IOphV occlusion. In our case, the AVF flowed into the bridging vein around the optic nerve, which connected the IOphV and the SOphV. The approach route from the anterior SOphV was too acute and complex to reach the AVF shunting point under the occlusion of the direct connection between shunt point and SOphV in our patient.
Moreover, we used a stiff microcatheter. We undertook two interventions because of the stiffness of the microcatheter; this was not related to our choice of the facial vein approach.

The facial vein approach is safe. Because the use of this approach does not require general anesthesia, experienced orbital surgeons, or high-resolution intraoperative angiography, we believe that this approach should be attempted first.

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Reference

Modeling the Risk of Unruptured Intracranial Aneurysms and the Benefit of Prophylactic Treatment

To the Editor: We read with interest the article by Yoshimoto on modeling the risk of unruptured intracranial aneurysms and the benefit of prophylactic treatment (Yoshimoto Y: A mathematical model of the natural history of intracranial aneurysms: quantification of the benefit of prophylactic treatment. J Neurosurg 104:195–200, February, 2006).

Abstract

Object. The International Study of Unruptured Intracranial Aneurysms (ISUIA) data raised new controversy regarding the threshold size that requires treatment. In particular, this study has been criticized for disagreeing with previous epidemiological data.

Methods. The author first used a Markov model to simulate the natural history of intracranial aneurysms, making three key assumptions based on prospective ISUIA data and other recent reports: that the rate of de novo aneurysm formation is constant after the age of 20 years; that unruptured aneurysms gain volume at a constant rate; and that unruptured aneurysms rupture at a volume-dependent rate. Next, he expressed outcomes for patients with unruptured aneurysms in terms of expected number of quality-adjusted life years (QALY) and compared two hypothetical cohorts, one receiving treatment and the other not being treated. These assumptions enabled the construction of a mathematical model with epidemiologically compatible findings. The benefits of treatment for unruptured aneurysms were highly influenced by aneurysm size and were calculated as −0.28, 0.25, and 1.07 QALY for patients having unruptured aneurysms with diameters of 7, 10, and 13 mm, respectively.

Conclusions. Under the author’s assumptions, the prospective ISUIA data may be consistent with epidemiological findings. Prophylactic treatment for unruptured aneurysms may produce some benefits in large aneurysms if acceptable treatment risks can be assured, but it is not likely to offer improvement over the natural history for patients with small aneurysms.

The model presented by Yoshimoto is particularly intriguing. Despite a number of simplifications, it provides a concept that may be helpful in decision making for individual patients.

One aspect of this report deserves comment. The author concludes from the conducted analysis that prophylactic elimination of large aneurysms may be beneficial. This conclusion is based on data from the ISUIA indicating that larger aneurysms carry a higher natural risk than smaller ones.1 The common clinical experience that the treatment risk also depends strongly on aneurysm size is ignored. Incorporation of this factor, which is also clearly proven by the ISUIA, may render the principal conclusion of the analysis questionable. The 10-year rupture rate of unruptured aneurysms measuring 13 to 24 mm appears to be 20%, and the 10-year rupture risk of giant aneurysms is approximately 30% according to ISUIA data. On the other hand, the risk of treatment-associated morbidity in large anterior circulation aneurysms (13–24 mm in diameter) is given as less than or equal to 25% for the different treatment modalities and age groups. In patients with giant anterior circulation aneurysms, the combined rate of surgery-related morbidity and mortality is given by ISUIA as greater than 30% in patients 50 years and older. There is no doubt that ISUIA data on the natural history of unruptured intracranial aneurysms as well as on treatment-associated complication rates are not very precise because of the small number of patient participants. We use the following rule of thumb to calculate the surgical risk (morbidity and mortality) for unruptured aneurysms at our institution: risk = 3% + (1% × aneurysm diameter [mm]). The risk of endovascular therapy is estimated as 50% of the surgical risk. These numbers are compatible with our institutional experience and with the ISUIA data. Our estimate is taken as an average and is modified for different age groups and aneurysm locations. The smaller risk of endovascular therapy for large aneurysms is balanced by their often incomplete elimination.

Although we are aware that no new information can be expected from a mathematical model, it should help us to form the data into an understandable concept. Because the natural and therapeutic risks of an unruptured aneurysm depend on its size, knowledge of the exact relation between these two factors is critical to defining the indications for treatment.

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Reference

Response: I appreciate the interest in my paper shown by Drs. Steiger and Hänggi. The ISUIA demonstrated that the rupture rate and treatment risk of unruptured intracranial aneurysms are size dependent, although a potential limitation of this study was the nonrandomized nature of the conservative, surgical, and endovascular cohorts; this led to asymmetries within groups. Indications for prophylactic intervention should clearly be determined by seeking a balance between the risks entailed by the natural history of the lesion and those associated with its treatment. In many cases, however, a high-risk natural history is associated with a high treatment risk.