Distal arterial occlusion for dissecting aneurysms

ROBERTO C. HEROS, M.D.
Department of Neurosurgery, University of Miami, Florida

The report by Nussbaum and his associates describing 3 patients with a ruptured dissecting aneurysm of the PICA treated by distal PICA occlusion is important because it presents a therapeutic alternative in cases in which other procedures—a direct approach to the aneurysm, proximal occlusion, or aneurysm trapping with or without bypass surgery—are not practical options. Clearly, patients with these lesions would present to most neurosurgeons, even neurosurgeons with a specialized vascular practice, only very rarely. In this respect, it is remarkable that Nussbaum and colleagues have collected 3 such cases in an 18-month period. The purpose of this editorial is to emphasize the potential problems with this approach and to give the authors the opportunity to clarify in their response to this editorial a couple of points that I will raise.

Clearly, the authors were imaginative and courageous in using this technique to deal with these aneurysms. As they readily admit, the concept is not new and there have been reports, which they quote and discuss briefly, of using this approach of distal outflow occlusion to treat complex aneurysms of the proximal segment of the middle cerebral artery (MCA). The rationale for treating unclippable aneurysms of the proximal MCA by distal outflow occlusion is similar to the rationale used by Nussbaum and colleagues in treating PICA aneurysms. Both the M1 segment of the MCA and the proximal segments of the PICA have important perforators going to the basal ganglia and to the brainstem, respectively. The hope with distal occlusion is that it will allow continuing perfusion of those perforators and yet reduce the pressure within the aneurysm sufficiently to promote its thrombosis. The authors’ cases prove that in fact this can happen. I have encountered a somewhat similar situation in several patients with giant unclippable MCA aneurysms; in these cases I performed a distal bypass and placed a clip on the MCA proximal to the aneurysm but did not trap the aneurysm segment because it contained important perforators.

My hope was that there would be enough retrograde flow from the bypass into the aneurysm segment to keep open those perforators but yet that the pressure in that segment would be low enough to promote aneurysm thrombosis; I have been able to document angiographically in those cases that indeed this has occurred.

As Dr. Nussbaum and his colleagues discuss, there has been at least one publication that has provided an elegant theoretical scientific explanation, based on the Bernoulli equation, of the expected decrease in pressure in the proximal arterial segment that should follow from distal occlusion. There are also related clinical situations that offer some support for this rationale. For example, we routinely treat some cases of dural arteriovenous fistulas by occluding the venous outflow, which predictably results in thrombosis of the fistula. Dr. Mullan has treated cerebral arteriovenous malformations (AVMs) by occluding the venous outflow after a significant but incomplete reduction in the arterial inflow to the lesion, resulting in complete AVM thrombosis. We have ample evidence in the literature and from personal cases involving AVMs in which ligation of a feeding artery to the AVM distal to an aneurysm has resulted in size diminution and/or complete thrombosis of the proximal aneurysm. Less commonly, however, such a maneuver has resulted in rupture of the aneurysm.

In spite of the solid rationale for assuming, as these authors did, that outflow occlusion would result in thrombosis rather than rupture of the aneurysm, it is obvious that the latter situation needs to be feared. I think it would be dangerous to trust, unreservedly, mathematical data obtained from in vitro models to predict that the aneurysm will always thrombose rather than rupture after distal outflow occlusion. I feel certain that the flow/presence relationships within a complex biological entity such as an artery bearing an aneurysm are complicated and different in each clinical case. I do not know of any examples in which this treatment of distal outflow occlusion has been employed and the aneurysm has ruptured, either intraoperatively or shortly thereafter; however, this treatment has probably been used infrequently enough that it is no surprise that these cases have not come to our attention, if they indeed have occurred. Even if this had never occurred, I would still be very apprehensive about using this maneuver, reserving it only if there is absolutely no other way of treating the aneurysm. Dr.
Nussbaum is a very experienced vascular surgeon, and I truly believe that if he believed that there were no other options, it is indeed very likely that there were not. I personally wonder, however, whether under similar conditions, I would not choose to occlude the PICA at its origin and risk a probable lateral medullary infarct rather than risk a potentially fatal rehemorrhage from the aneurysm, which fortunately for the authors’ 3 patients did not occur, but could possibly occur in the next patient. Undertaking this type of treatment of a newly ruptured dissecting aneurysm, which, as we know, has a tremendous tendency to rebleed, is very different than applying it to an unruptured giant MCA aneurysm, which is frequently calcified and at least partially thrombosed and in which I would presume the risk of hemorrhage is considerably less.

In addition to aneurysmal rupture, the other obvious risk of distal occlusion, which was mentioned by the authors, is that of retrograde thrombosis of the occluded artery. We know that this has occurred occasionally after occluding a feeder to an AVM even though there may have been smaller branches coming off proximally to the occlusion. Again, the authors’ patients fortunately did not have this problem, but it is an ever-present danger with this therapeutic approach.

It would be helpful if the authors would clarify exactly where they placed their clip. They talk about the “tonsillar loop” and the “tonsillar point” and it would be helpful if they clarified this, referring to the well-established nomenclature of the different segments of the PICA. I presume that by “tonsillar loop” they mean the tonsillomedullary segment. It should be kept in mind that although there are no important perforating branches to the brainstem distal to the tonsillomedullary segment, important perforators to the basal ganglia of the cerebellum originate distally, from the telovelotonsillar segment, and their occlusion can lead to a significant cerebellar deficit from an infarct such as the one demonstrated in the authors’ Fig. 1E.

It would also be important to know whether these aneurysms have indeed been “cured” at a longer-term follow-up. In the first case, it appears that a postoperative angiogram was obtained only 8 hours after the procedure; was there a subsequent follow-up angiogram? In the second case an angiogram acquired 1 month postoperatively showed the aneurysm to be completely thrombosed; was there a subsequent angiogram? In the third case, the authors state that there was “delayed cerebral angiography.” How late was this angiography performed?

To conclude, we are grateful to Dr. Nussbaum and colleagues for raising our level of awareness of this therapeutic maneuver, which is certainly one that we must keep in mind in desperate cases in which there appears to be no other reasonable option. My cautionary comments in this editorial are not meant as criticism but rather they are meant to emphasize what the authors state clearly: this form of therapy should only be considered where there are no other more conventional therapeutic approaches available.

References


Response

ERIC S. NUSSBAUM, M.D.
National Brain Aneurysm Center, Twin Cities, Minnesota

We would like to thank Dr. Heros for his everthoughtful comments and for the opportunity to address the important points that he raises. In a previous report in the Journal of Neurosurgery, we outlined our preferred management algorithm for treating dissecting PICA aneurysms. Although we have argued strongly for either attempted parent artery preservation or for distal revascularization in the setting of vascular sacrifice, there have been rare situations such as those detailed in the current report when these options did not seem feasible. Dissecting aneurysms often have a malignant tendency to bleed repeatedly; nevertheless, our previous work has underscored the fascinating potential for delayed vascular remodeling and healing of these lesions. Thus, we suspected that if we could stabilize the situation, preventing early rebleeding and diminishing flow across the aneurysm segment, the lesions might in fact regress over time. The use of distal occlusion remote from the aneurysm itself seemed particularly well suited to this problem, creating significantly decreased flow through the aneurysm while, at least in theory, maintaining perfusion of the critical brainstem perforators interposed between the aneurysm and the occluding clip.

Despite the fact that the rationale for the use of distal occlusion in treating an intracranial aneurysm is well grounded in theory, we too shared Dr. Heros’ concern for the possibility of a dangerous intraoperative rupture precipitated by application of a clip distal to such a friable, recently ruptured lesion. As mentioned in our report, the notion that a ruptured aneurysm could be treated safely with distal occlusion had been suggested to us previously by Dr. Robert Spetzler. Still, several years elapsed before we found ourselves in a situation in which this seemed the

References


