Editorial

Dural arteriovenous fistulas

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The authors of this study present a remarkable series of 251 patients with 261 intracranial dural arteriovenous fistulas (DAVFs) collected between 1944 and 2006 at two university hospitals, Helsinki and Kuopio, in Finland. From the practical point of view, these cases were collected between 1970 and 2006 because there were only 11 patients diagnosed before 1970 during a period when only carotid artery angiography, performed by direct cervical puncture, was available for diagnosis. Nevertheless, this is a remarkable consecutive series, and the fact that the authors were able to study every patient with this diagnosis during this long period and that all but 15 cases had at least 3 months of follow-up is a tribute to the remarkable medical recordkeeping system in Finland. Additionally, the well-known high stability of the population in that country, as well as the centralization of care at one university hospital in any specific region, contributed to making this review possible. I will comment briefly on some of the information in this paper that I found interesting.

In order to give a relatively accurate assessment of the incidence of DAVFs, the authors analyzed the subgroup of patients diagnosed between 2001 and 2005 when full modern diagnostic modalities were available. They came up with an incidence of 0.51 per 100,000 individuals per year. Although similar incidence has been quoted in other studies, I believe that this is the most accurate estimate because of the relative stability of the population in the catchment area of the two centers involved and because of the fact that in each of these two areas the patients were treated only at one hospital. For comparison, the authors tell us that during the same period in these two regions, the yearly incidence of brain arteriovenous malformations was 1.09 per 100,000 individuals, which is essentially twice as high as the incidence of DAVFs.

The authors appropriately use the Borden classification for DAVFs outside of the cavernous sinus. As they imply, this classification, which is based on the pattern of venous drainage, is extremely useful to predict the natural history of these fistulas in terms of risk of hemorrhage or serious neurological deficit based on whether the lesions drain only into a major venous sinus (Borden Type I), into a sinus but also with retrograde drainage into pial veins (Borden Type II), or exclusively in a retrograde fashion into pial veins (Borden Type III). The classification of these fistulas in this manner has a very direct impact on the aggressiveness with which they should be treated, as is well known. When it comes to carotid-cavernous fistulas (CCFs), however, the Borden classification is not relevant since these fistulas can threaten vision and lead to blindness, which is of course a major neurological deficit, regardless of the absence or presence of retrograde pial venous drainage. That is why, again appropriately, the authors use the well-known Barrow classification for CCFs. It should be noted that the Barrow classification is essentially descriptive and was not intended to denote prognosis and therefore guide treatment decisions. The latter should be guided mostly by ocular symptomatology and objective measurements of intraocular pressure to determine the risk to the eye and vision. An occasional pitfall is illustrated in Fig. 1. This patient presented simply with a subjective bruit in the eye, which was actually quite tolerable to him. On this basis alone, one would be inclined to treat the patient conservatively since his eye, his vision, and the intraocular pressure were normal. However, as indicated in Fig. 1, the reason that this patient had no significant ocular symptomatology is that the drainage of his CCF was not into ophthalmic veins, as in the usual CCF, but rather in a retrograde fashion into the basal vein of Rosenthal through a crural vein that connected the cavernous sinus with the basal vein. Given the presence of retrograde pial venous drainage, I felt that the patient was at high risk of hemorrhage and/or serious neurological complications, and therefore we treated the fistula by undertaking direct surgical interruption of the pial venous drainage, which resulted in complete occlusion of the CCF.

I found it interesting that the authors had 3 patients with Borden Type I fistulas (no retrograde pial drainage) that presented with hemorrhage. This challenges our belief, amply supported by the literature, that these fistulas do not bleed. It would be interesting if the authors can give more details on these 3 patients in a response to this editorial since it would be important to note that, though rare, hemorrhage can still occur with fistulas in which the drainage is entirely confined to a major venous sinus.

One of the most interesting aspects of this histori-
The current treatment paradigm used by these authors is very reasonable and by and large similar to ours except for two areas of minor disagreement. Although the authors treat many Borden Type I fistulas conservatively, they believe that when the patient suffers intolerable symptoms such as bruit or exophthalmos, the fistula should be treated with the intention of complete occlusion since, in their experience, incomplete obliteration may lead to gradual revascularization of the fistula, at times converting it into a more complex and more difficult to treat fistula. Although the latter is true, it is also well known that the risk of a Type I fistula converting into the more aggressive Type II or III fistula is very small, probably less than 2%,8 and therefore we more frequently than not treat these Borden Type I fistulas relatively conservatively with palliative embolization; the aim is to reduce or eliminate the bothersome symptoms, knowing that in the future the fistula may need to be re-treated and that there is a very small risk of conversion into a more threatening fistula. In other words, we feel that taking serious risks, as those incurred when performing endovascular complete occlusion of the fistula or craniotomy with excision of the fistula, is not unjustified given the relatively benign natural history of the fistula.8

The second area of disagreement is that in Borden Type II and III fistulas (fistulas with retrograde pial drainage), the authors feel that simple disconnection of the pial venous drainage is insufficient treatment and they prefer a goal of total occlusion of the fistula by embolization and, if that is not sufficient, with craniotomy and excision of the fistula. It is true that in many of these fistulas, particularly the complex ones in the area of the transverse-sigmoid sinus, simple venous disconnection is bound to fail. However, our experience, as well as considerable support from the literature, has led us to believe that many if not the majority of fistulas that have only pial venous drainage (Borden Type III) can be treated by simple microsurgical occlusion of the pial venous drainage (disconnection), which is frequently through a single vein. In these cases, the fistula has nowhere else to drain and generally thromboses. This is particularly the case with fistulas located in the anteromedial base of the anterior cranial fossa (“ethmoidal” fistulas) and in the tentorium, in the region of the superior petrosal sinus, which is usually thrombosed in these cases and the fistula drains exclusively into the petrosal vein.1,6 We have also had several examples of fistulas in other locations, such as the torcula, the tentorial incisure, the superior sagittal sinus, and the lateral wall of the cavernous sinus, that have been treated successfully with simple disconnection of the pial venous drainage.

We are very thankful to the authors for presenting us with this unique series of considerable historical significance.

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Disclosure

The author reports no conflict of interest.

References