Endovascular Treatment of Intracranial Arteriovenous Malformation

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KEYWORDS

• Arteriovenous malformation • Endovascular treatment
• Intracranial hemorrhage • Embolization • NBCA • Onyx

Arteriovenous malformations (AVMs) of the brain are vascular lesions in which an abnormal tangle or nidus of vessels permits pathologic shunting of blood flow from the arterial to the venous tree without an intervening capillary bed. AVMs typically present in young adults (mean 35 years ±SD 18)[1] and have a variety of clinical manifestations including most commonly hemorrhage, but also seizures, headaches, and progressive neurologic deterioration.[2] Since the advent of contemporary brain imaging techniques, an increasing number of AVMs are detected before they hemorrhage. The number of AVMs identified before rupture is now twice those identified after rupture. This has led to new considerations and modifications of interdisciplinary AVM management strategies.[3]

The ultimate goal of AVM therapy is complete obliteration of the lesion because any residual AVM might result in hemorrhage and partial treatment may increase the chances of bleeding.[4–6] Complete obliteration is more commonly achieved by multimodal therapy rather than by embolization alone. The available options for treatment include endovascular embolization, microsurgical resection, radiosurgery, medical management, or a combination of these treatment modalities.

Neuroendovascular therapy is a critical component of this multidisciplinary and multimodal approach. In general, because the risk of rebleeding is high, and the main cause of disability in patients with AVMs is hemorrhage, early assessment and delineation of a stepwise treatment plan is recommended for those who have experienced an AVM-related intracranial hemorrhage (ICH). Newer embolization techniques and embolic agents will continue to be developed and introduced, affecting the treatments associated with embolization. Although better techniques allow a more aggressive embolization of the AVM nidus, it is unclear at this time if some portion of the complication risks previously carried by surgical resection may be transferred to the embolization procedure.

EPIDEMIOLOGY

The incidence and prevalence of intracranial AVMs has been mainly estimated from autopsy series and retrospective population-based studies. In the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage, symptomatic AVMs were found in 8.6% of all nontraumatic subarachnoid hemorrhages.[7]

During the era before noninvasive brain imaging, one autopsy series detected a prevalence of 4.3%.[8] Although not derived from a population-based study, these data are of interest because

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they represent a careful autopsy-based effort to determine the prevalence of AVMs.

In the Netherlands Antilles, between 1980 and 1990, an annual incidence of 1.1 symptomatic AVMs per 100,000 people was identified.[9] In this fairly isolated and homogenous population, however, an unusually high proportion of the patients had multiple brain AVMs (25%) and hereditary hemorrhagic teleangiectesia, or Rendu-Osler-Weber disease (35%), making it difficult to compare the findings with those described in other populations.

In a retrospective, population-based study conducted over 27 years in Olmsted County, Minnesota, the incidence of symptomatic ICH due to any type of intracranial vascular malformations was 0.8 per 100,000.[10,11] Over many years, the Olmsted County study has provided the most reliable data concerning the detection rate of brain AVMs. A limitation of the study is its relatively small and homogenous population base, and conclusions drawn for AVMs were based on only few lesions detected between 1965 and 1992.

The New York Islands Arteriovenous Malformation Study is the first ongoing prospective population-based survey determining the incidence of AVM hemorrhage and associated morbidity and mortality rates in a population of more than 9 million people located in New York. Initial results calculated an AVM detection rate of 1.34 per 100,000 person years and a first-ever acute AVM hemorrhage rate of 0.51 per 100,000 person-years.[1] These rates reflect increased use of MRI due to low threshold for imaging. As many as 62% of the AVMs in this study were diagnosed before hemorrhage.

PATHOGENESIS

Most brain AVMs occur sporadically; however, they also have been associated with several congenital and hereditary syndromes, including Rendu-Osler-Weber disease (hereditary hemorrhagic teleangiectesia), Wyburn-Mason syndrome, and Sturge-Weber disease.[12–14] Rare familial cases not associated with syndromes also have been described.[15]

Recent evidence suggests that not all brain AVMs are congenital in origin.[16] Although the large majority probably occur congenitally due to failure of capillary formation during early embryogenesis,[17] some AVMs seem to form in response to a postnatal stimulus of angiogenesis, particularly in younger patients. De novo development of AVMs in children and in adults has been reported.[18,19] Moreover, AVMs have reoccurred in children after complete surgical resection.[20]

CLINICAL PRESENTATION

ICH is the initial manifestation of AVMs in at least 50% of cases.[1,10,21,22] The next most common presentation is seizure, which occurs in approximately one third of cases, often alerting a physician to the presence of an AVM.[23–25] The available literature documents a remarkable variation in incidence of seizures associated with AVMs. Inconsistent data from reports preclude accurate determination of the relationship between seizures and subsequent risk of ICH. Several types of attacks labeled as seizures occur, and the type of seizure is often unreported in studies.

Headache is the presenting symptom in approximately 15% of AVM patients. Because headaches are a common complaint in the population at large, it is difficult to determine if the headaches associated with AVMs are unique to the condition. In contrast to early assumptions, the headaches in AVMs are of no distinctive type, frequency, persistence, or severity. Migraine headaches with and without aura have been documented in the literature.[26] Little evidence supports the claim that recurrent unilateral headaches should arouse suspicion of an ipsilateral AVM. The yield for AVMs in evaluation for headache is low; in one study, only 0.2% of patients with normal neurologic findings who underwent neuroimaging for headache were diagnosed with AVM.[27] The postoperative disappearance of migraine headaches is not unusual and may occur after any type of operation.

Focal neurologic signs without hemorrhage are distinctively rare. Slowly progressing neurologic deficits, once considered common, are part of the presentation in only few patients (4% to 8%).[28–32] Shunting through a low resistance AVM results in hypoperfusion of the surrounding normal brain tissue, a phenomenon known as ''vascular steal''; however, evidence for a causal link with ischemic symptoms is lacking.[24] Venous hypertension and mass effect of the nidus offer alternative explanations for progressing focal neurologic deficits.[33]

NATURAL HISTORY

The risks of invasive management should be evaluated against the background of the natural history of the disease. Ideally, physicians need to know whether or not there are large numbers of relatively asymptomatic patients who live normal lives; whether or not nonhemorrhagic but symptomatic patients can be maintained with conservative therapy only; and whether or not patients who have hemorrhaged in the past are prone to hemorrhage again.

Unfortunately, little unbiased natural history data are available, in part, because brain AVMs

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