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Commentary

Venous Angioma, Cavernous Angioma, and Hemorrhage

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In this issue of *AJNR*, Wilms et al (1) add to the ongoing debate whether the venous angioma, more recently called *developmental venous anomaly* (2), can be symptomatic, whether it alone can bleed, or whether such hemorrhage occurs because of the concomitant occurrence of a cavernous angioma. This debate is not just semantic. It is essential to pathophysiologic understanding of a given lesion so that risks of sequelae can be determined. Therapeutic options can then be considered once those risk factors are known.

The venous angioma is a slow-flow venous anomaly consisting of a number of dilated medullary veins converging into a central hilum to produce the classic caput medusae. This venous complex then drains into an even more dilated transparenchymal vein that leads to a major venous outflow track such as a dural sinus. The venous angioma is frequently found incidentally on computed tomography (CT), magnetic resonance (MR), or cerebral angiography performed to evaluate a specific lesion such as a brain tumor, demyelinating disease, or carotid stenosis, but it also may be found in a patient having vague neurologic symptoms such as headache or dizziness. Since the introduction of MR imaging, and particularly the development of contrast-enhanced MR, the venous angioma is seen much more commonly than in the past (3–6). Moreover, venous angioma is frequently discovered in areas unrelated to the symptoms prompting the imaging procedure. This would suggest that the venous angioma is usually a normal variant and not productive of symptoms or hemorrhagic/ischemic sequelae. However, various series in the literature suggest that the venous angioma may produce the presenting symptoms in as many as 40% of patients (7, 8). We have seen an example of a fourth-ventricular hemorrhage secondary to bilateral cerebellar venous angioma in a young gymnast who probably raised her venous pressure during exercise (9); we have seen patients who have had a venous angioma and parenchymal hemorrhage and who have undergone surgery without the pathologic findings of a cavernoma or other vascular malformation other than the venous angioma. We have also seen cases of venous ischemia in the region of a venous angioma (10). Reports in the literature, albeit on the basis of a small number of cases, suggest that posterior fossa venous angiomas may produce symptoms and hemorrhagic sequelae more commonly than supratentorial lesions (11). These reports invariably refer to papers in the CT era at which time posterior fossa venous angioma was only detected when accompanied by hemorrhage. Even with this caveat, our observations and the literature suggest that the venous angioma may not be totally benign. Increased systemic venous pressure, or increased local venous pressure secondary to stenosis of the draining transparenchymal vein or other venous obstruction, might lead to hemorrhagic or ischemic complications (6, 9) (Dillon WP, Hieshima GB, Halbach VV, Dowd CF, A New Observation on the Association of Venous Angioma, Hemorrhage and 'Cryptic Vascular Malformation,' presented at American Society of Neuroradiology, Washington, DC, 1991).

With the increased use of MR, observers have identified lesions containing chronic blood products consisting of mixed low (hemosiderin), and high (methemoglobin) intensity on T2-weighted studies in the vicinity of venous angiomas, with or without evidence of more recent hemorrhage (12). The terminology for these lesions of mixed MR intensity can be confusing. Cavernomas, or cavernous angiomas, are slow-

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flow vascular malformations with large sinusoids that undergo recurrent hemorrhage and are characterized by combined low and high intensities. An arteriovenous malformation may undergo thrombosis or may be small enough ("cryptic") or have slow enough flow to not be seen angiographically, but to be seen with similar MR intensities. The term *angiographically occult vascular malformation* has been used to include both of these pathologic entities. However, old hemorrhage alone, without any type of vascular malformation, can be seen on T2-weighted MR as hyperintensity, hypointensity, or both. Slowly flowing blood likewise can be seen as hyperintensity. What then constitutes an appropriate set of criteria for the MR diagnosis of a cavernoma?

The MR analogue to the multiple sinusoids or "caverns" might be the presence of a multiseptated lesion, with the multiple compartments having different intensities suggesting the presence of hemorrhage of various ages and slowly flowing blood. Unfortunately, this concept of a multiseptated, multicompartmental lesion as characteristic of a cavernoma has not been adequately correlated with pathologic findings, especially with the aim of distinguishing it from old blood alone.

We have also seen numerous examples within and outside the literature of lesions with pronounced hypointensity, with or without a central core of hyperintensity, which have been diagnosed by others as cavernomas. However, these lesions could simply be old hemorrhage without an underlying vascular malformation. Such lesions are increasingly being detected in patients who have undergone radiation therapy for brain tumor. In fact, we have recently encountered yet another such case, but with an interesting twist. The patient has a small cerebellopontine venous angioma. In this patient, the preoperative MR shows the tumor and the venous angioma; the postoperative MR (at 7 years) shows a 1-cm focus of T2 shortening adjacent to the venous angioma. Clearly, this hypointense abnormality is an acquired lesion; it may be radiation induced. Moreover, it is probably a venous hemorrhage related to the venous angioma. Does this not suggest that the bleeding is caused not by a cavernoma, but by a small radicle of the venous angioma?

Recently, an intriguing hypothesis has been suggested that unifies the venous angioma, cavernoma, and hemorrhage (Dillion et al, A New Observation, 1991). In a series of 16 patients with angiographically defined venous angioma and MR findings of old blood, angiographically occult vascular malformation, or acute hemorrhage in close association with the venous angioma, 13 of these 16 patients had a significant stenosis of the transcerebral vein draining the venous angioma on the angiogram. These authors believe that obstructed venous outflow of the venous angioma may lead to hemorrhage, which then secondarily develops into a "cavernoma."

The paper by Wilms et al in this issue reports a review of 65 cases of venous angioma, with 15 (23%) also having a "cavernoma" (1). Nine patients had acute symptoms. All patients had a CT scan at the time of presentation. Ten of the 15 patients also had an MR at presentation, 5 had an MR some time later, and 2 patients with early MR had repeat MR at a future time. The authors used the presence of a complex internal structure as the imaging criterion for a diagnosis of cavernoma. The lesion had to have multiple septations and multiple areas of varying MR intensity producing a round, multilobular mass. The authors say they did not accept a lesion with just a black rim and white center as evidence of a cavernoma. In one patient, there were multiple cavernomas. The CT criteria included the classic findings of increased density without contrast, frank calcification (which was not always present), and a minimal to mild degree of contrast enhancement.

Four of the five illustrated cases in this paper have the complex MR intensities used to make the diagnosis of cavernoma, and we understand that not all cases can be illustrated. Unfortunately, there is no pathologic or surgical proof in any of these cases to validate that a cavernoma was actually present. The authors state that MR was far more effective than CT in both the acute and chronic state for the demonstration and diagnosis of a cavernoma; CT missed the diagnosis of a cavernoma in 9 patients, usually because the acute hemorrhage masked the high density lesion. The authors indicate that the hematoma was always in the vicinity of the cavernoma but that the venous angioma was usually at some distance from the hemorrhage. This was said to be demonstrated on two early and five late MR images. However, only one case in the article (their Figure 4, case 11) illustrates this finding convincingly. In our opinion, it is difficult in their Figure 3 to know

whether the hemorrhage has come from the venous angioma or the cavernoma.

Wilms et al are certainly on a better track than other investigators in attempting to make a diagnosis of cavernoma. They required a more complex MR pattern for this diagnosis than simply a hyperintense center and hypointense rim, findings typical of many evolving hemorrhages. Unfortunately, none of their cases is pathologically proved. In addition, there is no statement regarding symptoms of the other 50 patients with venous angioma but without cavernoma. Although this paper addresses yet another piece of the puzzle, it still does not answer the underlying question of whether the venous angioma itself can produce symptoms.

It is essential that we all define our terms, agree on diagnostic criteria to make a given diagnosis, expend significantly more effort to correlate imaging and pathologic data, make more effort to understand the pathophysiologic character of a given lesion, and attempt to define the risks of deficit-producing sequelae for a given lesion. If the multicompartmental concept for the MR diagnosis of a cavernoma is to be proved correct, there is need to correlate that imaging pattern more closely with pathologic material. However, MR may demonstrate intensity patterns that are too nonspecific to differentiate consistently a cavernous angioma from chronic hemorrhage alone. The venous angioma may be of very low risk in most cases. However, to say that it is totally benign may not be correct; it may be relatively benign compared with other vascular entities. If a venous angioma is found, with or without previous hemorrhage, should the patient be advised to avoid situations that produce increased intracranial venous pressure? Does the presence of a venous angioma and a "cavernoma", whether a true cavernoma or an evolving hemorrhage, mean that there is a higher risk for further bleeding than if the findings were that of a venous angioma only or a cavernoma without a venous angioma? Does a patient with a posterior fossa venous angioma have a greater risk of hemorrhage and/or "cavernoma" than does a patient with a supratentorial venous angioma? Or, do only those patients with a venous angioma that drains through the brain stem (to a petrosal sinus) have a greater risk? These are unanswered questions at this time. We urge all investigators to keep an open mind regarding labels. Avoiding dogma is a necessity if we are to make headway in understanding and treating neurologic disease.

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