Dural Cavernous Angioma: MR Features

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Summary: The imaging features of a cavernous angioma, which originated from the meninges, are presented. The patient harbored a second cavernous angioma within the brain parenchyma. Cavernous angioma should be included in the differential diagnosis of dural lesions, especially when other cavernous angiomas are present.

Index terms: Angioma; Arteriovenous malformations, cerebral

Cavernous angioma, also known as cavernous hemangioma or cavernoma, was described by Luschka in 1853 as an incidental finding in a suicidal patient (1). Cavernous angiomas account for 5% to 13% of cerebral vascular malformations and are thought to be present at birth (2). Multiple lesions are not uncommon. Although cavernous angiomas may occur anywhere in the brain or spinal cord, only a few cases of primary dural cavernous angiomas have been reported (3–5). We present the magnetic resonance (MR) imaging features of a cavernous angioma arising in the dura mater.

Case Report

A 35-year-old man presented with generalized seizures, headache, and left visual blurring of 2 months' duration. Medical history and physical examination findings were unremarkable. An electroencephalogram showed low background activity in the right parietal occipital region. Computed tomography (CT) scan demonstrated an extraxial lesion in the right parietal region (Fig 1A). The mass had punctate calcifications and showed minimal contrast enhancement. MR imaging showed the lesion to be heterogenous with mainly intermediate signal intensity on precontrast T1-weighted images (Fig 1B). T2-weighted sequences showed the lesion to be of high signal intensity and surrounded by a halo of very low signal intensity (Fig 1C). The adjacent brain parenchyma showed abnormal high signal intensity. After gadolinium administration, the mass enhanced (Fig 1D). In addition, T2-weighted images showed a second lesion in the left midparietal region (Fig 1D). This lesion was of high signal intensity centrally and had an area of low signal intensity peripherally. Conventional catheter angiogram was negative. At surgery, the right parietal occipital lesion was found to be completely extraaxial and dura based, and was entirely resected. Microscopy showed numerous vessels of variable size with distorted collagenized thin walls that had significant intimal fibrosis (Fig 1F). Calcifications and peripheral hemosiderin also were present.

Discussion

Cavernous angiomas are commonly seen in the subcortical regions of the cerebral hemispheres, pons, or cerebellum, but may occur anywhere in the brain leptomeninges and spinal cord (6). The majority of cavernous angiomas remain asymptomatic in life and are incidental findings at autopsy. When they produce symptoms, these commonly include seizures, headache, hemorrhage, and focal or progressive neurologic deficits (7). Seizures are believed to occur secondary to a combination of mass effect and deposition of hemosiderin, which has epileptogenic properties (8). The risk of bleeding is small and usually not life threatening. The risk is increased in patients with a prior hemorrhage (9). Recurrent (mainly subclinical) bleeds cause encasement or pseudoencapsulation of the lesion by hemosiderin-stained gliotic tissues (10, 11).

Macroscopically discrete, cavernous angiomas are multiloculated, purple, mulberry-appearing masses filled with sinusoidal vascular channels separated by fibrous strands and no intervening neural tissues (2). They are thought to arise at the capillary level. Microscopically, the vessel walls lack muscle and elastic tissue, are lined by a single endothelial cell layer, and may be calcified or ossified (5). Close macrophages show extensive deposition of hemosiderin. Hemangioma calcificans, a variant of cavernous angioma, show extensive calcification (12). As many as half of patients with cav-
Cavernous angioma have a family history of similar lesions (5).

Cavernous angioma may be treated by surgical resection. The goals of surgery include control of progressive neurologic deficits, elimination of mass effect, treatment of medically refractory seizures, and prevention of hemorrhage (2, 11). Radiation therapy also may be useful in selected cases (13).

CT shows cavernous angiomas to be well-circumscribed, slightly hyperdense masses without adjacent edema or significant mass effect (14). Calcifications are seen in as many as 30% of lesions. After iodinated contrast infusion, mild enhancement may be seen. Occasionally, cavernous angiomas contain areas of low density that are thought to be related to old thrombosis and/or cystic degeneration. By MR imag-
ing, cavernous angiomas have a “salt and pepper” appearance (15). A peripheral rim of very low signal intensity is seen on all sequences and represents hemosiderin deposits within macrophages. After gadolinium administration, enhancement usually occurs but is variable. Catheter angiography is generally negative, although occasionally a faint vascular blush may be seen, especially after prolonged injection (16).

A recent article described similar MR findings in dural juvenile capillary hemangioma. Juvenile capillary hemangiomas are a subgroup of cavernous hemangioma (17).

In our patient, CT showed a hyperdense mass with internal calcifications (Fig 1A). MR confirmed the extraaxial location of the lesion and showed it to have heterogenous signal intensity surrounded by a rim of very low signal intensity (Fig 1B). The adjacent white matter also had abnormal high signal intensity on T2-weighted images that was most likely related to gliosis and/or edema (Fig 1C). The lesion enhanced markedly after gadolinium administration (Fig 1D). Presence of a second, smaller lesion similar in appearance to the larger one led us to postulate the possibility of vascular malformation; however, because we were not aware that cavernous angiomas may occur in the meninges, our main preoperative diagnosis was meningioma.

References