Aqueduct compression from venous angioma: MR findings.

C C Blackmore and A C Mamourian

AJNR Am J Neuroradiol 1996, 17 (3) 458-460
http://www.ajnr.org/content/17/3/458

This information is current as of August 23, 2024.
Aqueduct Compression from Venous Angioma: MR Findings

C. Craig Blackmore and Alexander C. Mamourian

Summary: Vascular compression as the cause of aqueductal stenosis is rare. In a 16-year-old girl with hydrocephalus, MR imaging provided evidence of aqueductal stenosis caused by a venous angioma in the tectum and midbrain. This indicates the usefulness of MR imaging for the evaluation of obstructive hydrocephalus.

Index terms: Angioma; Aqueduct of Sylvius; Hydrocephalus

Venous angiomas are relatively common findings on brain magnetic resonance (MR) images. These lesions are usually incidental findings but may be symptomatic (1, 2). Clinical presentations include headache, seizures, and focal neurologic deficits (1). In this unusual case, venous angioma caused compression of the aqueduct and obstructive hydrocephalus, which was shown by contrast-enhanced MR imaging.

Case Report

A 16-year-old girl had a 2-month history of intermittent throbbing occipital headache, associated with photophobia and motion sickness. She also had pain and fullness in the left ear. Her medical history was significant for chronic sinusitis and otitis, and she had undergone a rhinoplasty and septoplasty several years earlier. She also had a history of depression, irregular menses, and an eating disorder. Findings on physical examination at presentation were entirely normal, with no papilledema or focal neurologic deficit. Her laboratory workup included complete blood count, electrolytes, liver function test, and thyroid function tests. The results of all these tests were within normal limits.

Because of the persistent headaches, MR imaging was performed. The images showed hydrocephalus in a pattern consistent with aqueductal stenosis (Fig 1). At the level of the midbrain, a vascular abnormality was noted, characteristic of a venous angioma (Figs 2 and 3). On the basis of these findings, a cardiac-gated, cine MR study of the aqueduct was performed. This examination revealed an absence of the expected flow of cerebrospinal fluid in the aqueduct.

Given the mild and nonspecific nature of the patient’s symptoms, as well as the absence of papilledema, shunting was not considered a reasonable option. The patient will be closely followed clinically.

Discussion

Venous angiomas represent the most common intracranial vascular malformations, composing 63% of such lesions in two recent autopsy series (1, 2). They consist of a network of dilated medullary veins, surrounding and draining into a large central vein. The intervening parenchyma is normal, suggesting that venous angiomas may represent developmental venous anomalies (3). Contrast-enhanced MR imaging can show these slowly flowing vascular channels. The characteristic radially arranged veins converging on a large central draining vein are described as the caput medusa. These generally have low signal intensity on T1-weighted images and show bright enhancement with administration of gadopentetate dimeglumine. On T2-weighted images, the draining veins may have high or low signal intensity depending on the flow velocity, orientation, and specifics of the pulse sequence (4, 5).

Commonly, the medullary veins will drain into the subependymal venous system, and the central vein will lie in the ventricular wall. In our patient, the central draining vein coursed along the floor of the third ventricle and compromised the aqueduct. The MR images in this case showed the typical pattern of hydrocephalus caused by aqueduct obstruction: enlargement of the lateral and third ventricles with a normal fourth ventricle. The cardiac-gated, cine MR study showed no evidence of flow within the
cerebral aqueduct, which confirmed the stenosis.

In most cases, venous angiomas are not thought to cause symptoms. However, seizures, headache, and focal neurologic deficits have been described and attributed to venous angiomas (1, 2, 6). In addition, hemorrhage has been reported in association with venous angiomas. It is not clear, however, if the venous angioma or a coexistent cavernous angioma is the source of the bleeding (1, 7, 8). Our patient presented with headache and with psychiatric symptoms of depression and eating disorder. Significantly, aqueduct stenosis in the adult has been associated with psychiatric symptoms, particularly depressive episodes (9, 10).

Venous angioma associated with ventricular outflow obstruction is extremely rare. Rosenheck reported a venous angioma with obstruction of the cerebral aqueduct in 1937 in a female patient with symptoms similar to Alzheimer disease (11). Since then, four other cases have been reported, in which all the patients presented with headache (12–14). In addition, a case of a venous angioma causing unilateral hydrocephalus from obstruction at the foramen of Monro has been reported (15).

The most important differential diagnosis in an adult patient with aqueduct stenosis is congenital stenosis and midbrain tumor. Although the subtype of congenital stenosis (forking, gliosis, aqueductal membrane) (16) may not be evident, MR imaging can help exclude an underlying neoplasm. In some instances, central nervous system ventriculitis with gliosis can cause aqueductal stenosis, which may also be shown by MR imaging. Vascular compression has been described as a cause of aqueduct stenosis, particularly from dolichectasia of the basilar artery (17, 18) or from an aneurysm of
the vein of Galen (19). The MR appearance of basilar artery dolichoectasia has recently been described (20, 21).

This report provides an additional case in which cerebrospinal fluid outflow is obstructed because of a venous angioma. The presentation in this case provides further support for the concepts that venous angiomas may be symptomatic and that obstruction of the cerebral aqueduct may be associated with psychiatric symptoms.

References