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Thrombus Simulating Flow Void: A Pitfall in Diagnosing Aqueductal Patency by High-Field MR Imaging

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Current medical literature suggests that absence of signal from the cerebral aqueduct on MR images is a reliable indicator of CSF flow, and therefore indicates aqueductal patency. We report a case in which absence of signal in the cerebral aqueduct simulating flow void was caused by an acute obstructive thrombus.

Case Report

A 21-year-old man with tuberous sclerosis presented with a 2-day history of progressive nausea, vomiting, ataxia, headache, and mild spasticity. The patient was known to have a large giant-cell astrocytoma occupying the third ventricle, and he had previously had ventriculoperitoneal shunts placed into both lateral ventricles.

A multiecho 2DFT MR scan was obtained on a 1.5-T unit* with TEs of 20 and 90 msec and a TR of 2000 msec (Fig. 1). In addition to showing distortion of structures adjacent to the third ventricle by the astrocytoma, this scan revealed enlargement of the fourth ventricle, which had not been present on prior studies. This finding, along with the posterior fossa symptomatology, raised the possibility of fourth-ventricle entrapment [1, 2] by aqueductal obstruction. However, the MR examination showed absence of signal from the cerebral aqueduct and superior aspect of the fourth ventricle, which was interpreted as indicative of aqueductal patency. Since this apparent patency did not correlate with the enlargement of the fourth ventricle or the patient's rapid clinical deterioration, a posterior fossa craniectomy was performed for decompression in spite of this MR finding.

After incision of the vermis and separation of the cerebellar hemispheres, fresh thrombus was discovered in the superior recess of the fourth ventricle suspended from the aperture of the cerebral aqueduct in stalactite fashion. A "tail" of this thrombus extended into the aqueduct and was removed. Microscopic examination of the pathologic specimen showed a typical blood clot with evidence of early lysis and organization, and no evidence of tumor cells.

Discussion

In this patient, hemorrhage from the third-ventricular giant-cell astrocytoma led to occlusion of the cerebral aqueduct by thrombus. This caused entrapment of the shunt-dependent fourth ventricle as well as the patient's clinical deterioration. The MR examination correctly demonstrated enlargement of the fourth ventricle, but was misinterpreted as indicating patency of the aqueduct because of absence of signal in this structure. Surgical exploration revealed the correct cause of the signal void in the aqueduct.

Current literature states that absence of signal from the cerebral aqueduct and other CSF pathways is due to pulsatile flow [3–6] and "confirms the patency of the area in which it is present" [7]. This has been termed the flow-void sign and has been well established as an important factor in the accurate interpretation of MR images [6, 8, 9]. It is commonly seen on T2-weighted images at points of narrowing of CSF pathways where flow velocity is greatest.

The MR appearance of the thrombus in the present case was identical to that caused by pulsatile flow of CSF (Fig. 2). This demonstrates that the MR finding of absence of signal from CSF pathways may be caused by fresh thrombus occupying these structures, and certainly does not establish their patency. Use of the term *flow void* to describe the MR finding of signal void in cases of this sort is unfounded.

Absence of signal from fresh thrombus on T2-weighted, high-field MR images has been attributed to the preferential T2 proton relaxation enhancement (PT2PRE) of intact erythrocytes containing deoxyhemoglobin [10]. Diffusion of water molecules through local field gradients caused by the different magnetic susceptibility of the intra- and extracellular environments leads to rapid dephasing of nuclear spins, and therefore to a shortened T2 relaxation time [11]. This PT2PRE is present only on high-field MR scanners, and will persist for several days, until erythrocyte membranes lyse.

Since hemorrhage into the ventricular system is a relatively common neuropathologic entity, it is important to address the issue of how to distinguish thrombus from pulsatile flow of CSF as a cause of signal void on MR images. Extension of signal void from the aqueduct into the superior recess of the fourth ventricle is commonly seen with CSF flow [3], but may be simulated by extension of fresh thrombus from the aqueduct into this area as it was in the case presented here. It is

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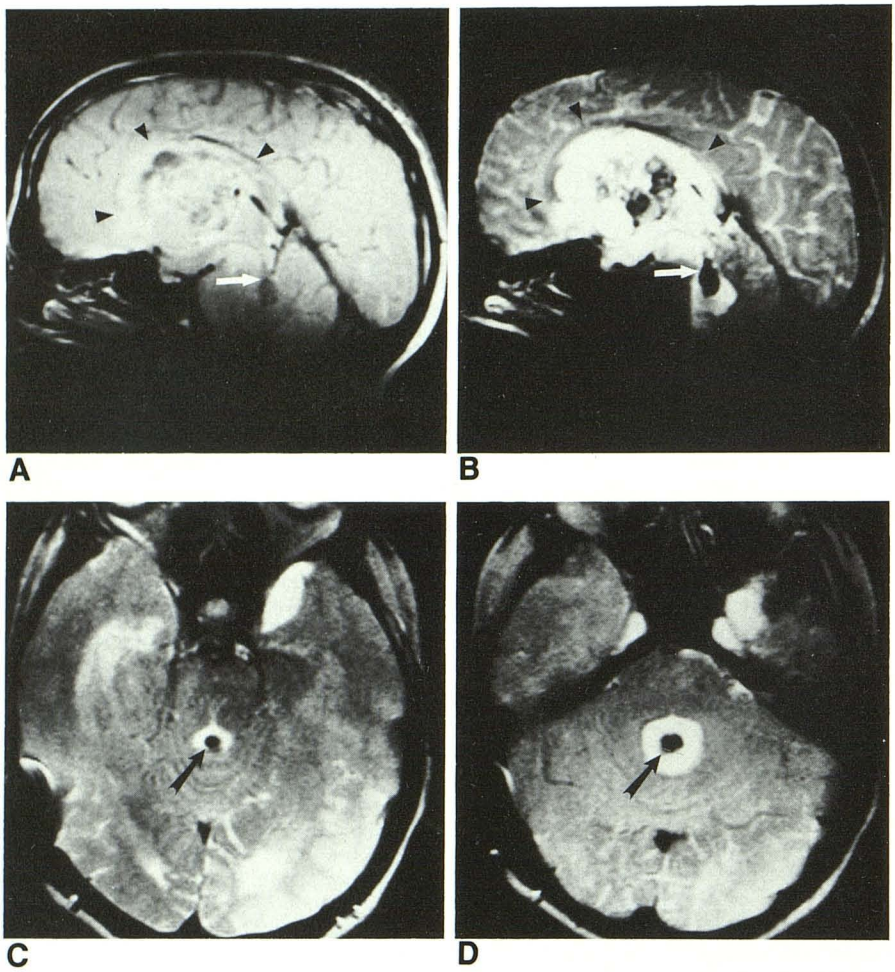

Fig. 1.—Patient with thrombus causing entrapment of fourth ventricle. Large giant-cell astrocytoma (*arrowheads*) occupies third ventricle. Decreased signal on spin-density-weighted image (*A*) and signal void on T2-weighted images (*B–D*) in cerebral aqueduct and superior recess of enlarged fourth ventricle are due to obstructive thrombus (*arrows*).

A, Sagittal image. TE 20, TR 2000 msec; 5-mm thick.

B, Sagittal image. TE 90, TR 2000 msec; 5-mm thick.

C and *D*, Transverse images. TE 90, TR 2000 msec; 10-mm thick.

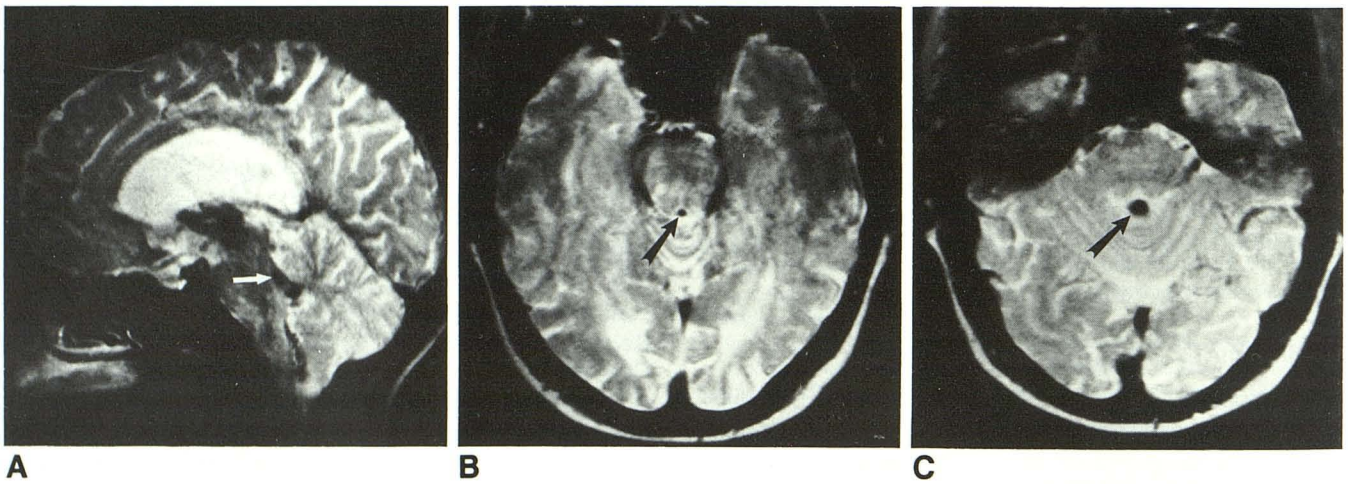

Fig. 2.—2DFT MR images of patients with patent aqueducts. TE 90, TR 2000 msec. Signal void in cerebral aqueduct and superior recess of fourth ventricle (*arrows*) in these cases results from CSF flow, but has an appearance virtually identical to that in Fig. 1.

A, Sagittal image, 10-mm thick, of patient with multiple sclerosis.

B and *C*, Transverse images, 10-mm thick, of patient with white-matter disease of uncertain origin.

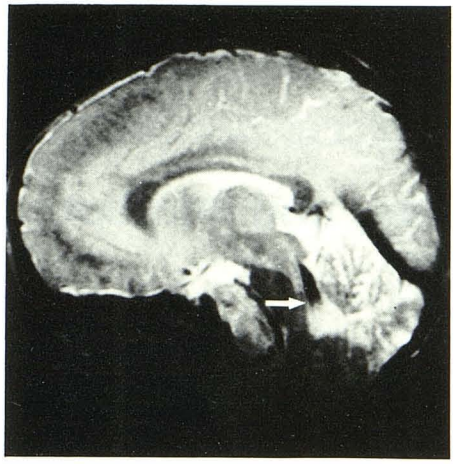

Fig. 3.—Sagittal 2DFT MR image of patient with tumor compressing the anterior pons. TE 90, TR 2000 msec; 5-mm thick. Signal void in cerebral aqueduct and superior aspect of fourth ventricle is due to CSF flow. Gradual disappearance of inferior margin of signal void in fourth ventricle (*arrow*) contrasts to sharp inferior margin of signal voids in Figs. 1 and 2.

tempting to speculate that the sharp inferior margin of the thrombus seen in the present case (Fig. 1A) may be distinguished from the more gradual disappearance of signal void (Fig. 3) often seen with CSF flow, and may therefore be a useful differentiating feature. Partial volume averaging would be expected to limit the usefulness of this sign, however; and absence of signal caused by CSF flow may also have a sharp inferior margin (Fig. 2A). T1-weighted images were not obtained in the present case but would be useful in distinguishing CSF flow from thrombus if enough time has elapsed for cell membrane lysis and methemoglobin formation to occur. A thrombus would then appear bright on the T1-weighted image [10, 12], unlike CSF. In an acute setting, however, one could not afford to wait for this process to occur, and flow-specific MR pulse sequences [13–16] or high-resolution X-ray CT may be required to distinguish fresh blood clot from CSF flow.

In conclusion, we wish to alert the radiology community of the pitfall of interpreting signal void in CSF pathways as indicative of patency. Just as absence of the flow-void sign does not necessarily indicate aqueductal obstruction [3, 4], the presence of an apparent flow void does not always indicate patency. Fresh thrombus should be included in the differential diagnosis of absence of signal from a CSF pathway on a T2-weighted, high-field MR image.

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