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*AJNR Am J Neuroradiol* 1987, 8 (6) 1134-1136

<http://www.ajnr.org/content/8/6/1134.citation>

This information is current as  
of April 19, 2024.

## Spontaneous Thrombosis of a Vein of Galen Aneurysm

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A case of spontaneous thrombosis of a vein of Galen aneurysm is presented. This is a rare event, with only six cases previously presented in the literature [1–5]. Our case is unique in that it represents the first in which serial documentation is provided by multiple imaging techniques. Evaluation of this patient included CT, cerebral angiography, and MR imaging.

### Case Report

A 6-month-old girl, while being evaluated by her family physician for otitis media, was found to have a head circumference of 47 cm (99th percentile), with a tense and full anterior fontanelle. Past medical history was unremarkable, with normal developmental milestones. A noncontrast CT scan showed enlarged lateral and third ventricles and a homogeneous, high-attenuation mass in the region of the posterior portion of the third ventricle (Fig. 1A). No definite enhancement was seen after the infusion of IV contrast material. Bilateral carotid and vertebral angiography revealed enlarged venous structures compatible with a vein of Galen aneurysm fed solely by the left posterior cerebral artery (Fig. 1B). These abnormal venous structures were smaller than the high-attenuation abnormality on CT, which led us to conclude that the lesion was partially thrombosed and readily amenable to surgical correction. The patient had a ventriculoperitoneal shunt, and 2 months later was admitted for craniotomy and clipping of the posterior cerebral artery. A preoperative scan done with and without IV contrast, however, demonstrated shunt malfunction with significant ventriculomegaly, necessitating shunt revision. The posterior third ventricular mass at this time was again identical on the noncontrast and contrast scans. It was now mixed in attenuation but unchanged in size from the earlier scan. The center of the malformation had areas that were hypodense, isodense, and hyperdense relative to brain. Peripherally, there was an area of calcification (Fig. 2A). Repeat cerebral angiography demonstrated slight mass effect in the posterior third ventricular region but no evidence of flow to the vascular malformation (Fig. 2B). Since further documentation of thrombosis of the aneurysm was demonstrated, the craniotomy was canceled.

The patient has done well since that time. A follow-up CT 6 weeks after shunt revision, without IV contrast administration, showed mild persistent ventriculomegaly. The mass had decreased in size slightly and was somewhat irregular in outline. The walls of the mass were more densely calcified than on the previous examination and the

center of the lesion was more homogeneous, being essentially isodense with brain (Fig. 3A). MR—using a spin-echo sequence, a 0.35-T magnet, a repetition time (TR) of 2.0 sec, and an echo time (TE) of 56 msec—revealed a lesion of high-intensity signal centrally, surrounded by absence of signal peripherally (Figs. 3B and 3C).

### Discussion

The clinical presentation and syndrome associated with vein of Galen aneurysm has been well described by Amacher and Shillito [6]. Six et al. [4] stated that while there is no well-described clinical syndrome associated with a thrombosing vein of Galen aneurysm, all their patients presented with progressive irritability and lethargy. The present case is unlike other reported cases of spontaneous thrombosis of a vein of Galen aneurysm for the following reasons: (1) our patient presented with an enlarged head only, (2) she is younger than any other patient described in the literature to spontaneously thrombose a vein of Galen aneurysm, and (3) she is the only patient for whom surgical correction was not attempted [4, 7, 8].

The CT and angiographic findings of a vein of Galen aneurysm are well documented [9, 10]. At presentation, the high-attenuation mass on CT was much larger than the enlarged veins seen on angiography (Figs. 1A and 1B), indicating the malformation was partially thrombosed [11–13]. Angiography 2 months later revealed no evidence of the abnormally dilated venous structures. On CT a central area of mixed hypodensity, isodensity, and hyperdensity was surrounded by a partially calcified rim of the malformation. The entire CT abnormality at this time was probably caused by thrombus, and the heterogeneous attenuation represented the variable maturation of the clot. The third CT, 4 months after presentation, revealed a decrease in size, further calcification of the rim, and a more homogeneous brainlike density of the central thrombus, indicating further maturation. A high-intensity signal on both T1- and T2-weighted images, compatible with clotted blood, was present on MR. The calcified periphery of the lesion lacked an MR signal, as would be expected. Although well demonstrated in axial and coronal MR images, the sag-

Received August 6, 1985; accepted after revision April 1, 1986.

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Fig. 1.—Initial CT scan and cerebral arteriogram at age 6 months.

A, Noncontrast-enhanced axial CT scan. Large mass in posterior third ventricle region. High-attenuation center probably represents acute thrombus (arrow), while surrounding, slightly lower-attenuation region is consistent with older thrombus and flowing blood (arrow-head). Secondary obstruction of third and lateral ventricles.

B, Lateral projection of vertebral artery injection. Dilated internal cerebral vein and vein of Galen. Vessel superiorly is stretched around superior margin of thrombus (arrows).

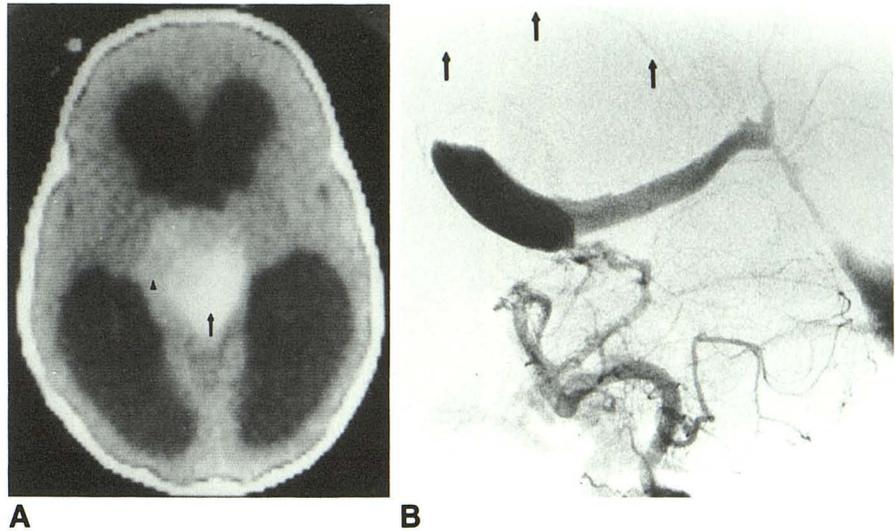
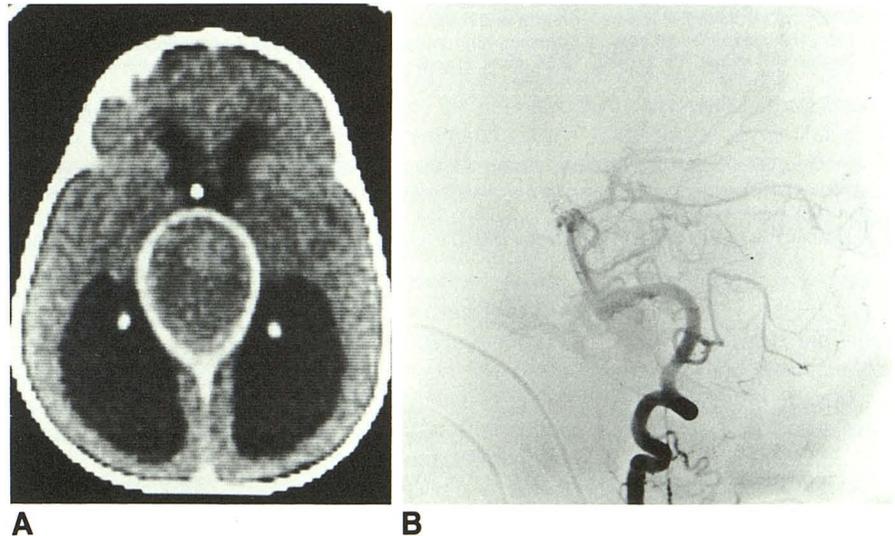


Fig. 2.—Follow-up CT scan and cerebral arteriogram at age 8 months.

A, Contrast-enhanced axial CT scan. Central area of heterogeneous attenuation. Periphery is partially calcified, and appearance is consistent with a maturing thrombus.

B, Lateral projection of vertebral artery injection. Nonvisualization of previously demonstrated dilated venous structures.



ittal projection was especially informative in evaluating this midline malformation (Fig. 3C).

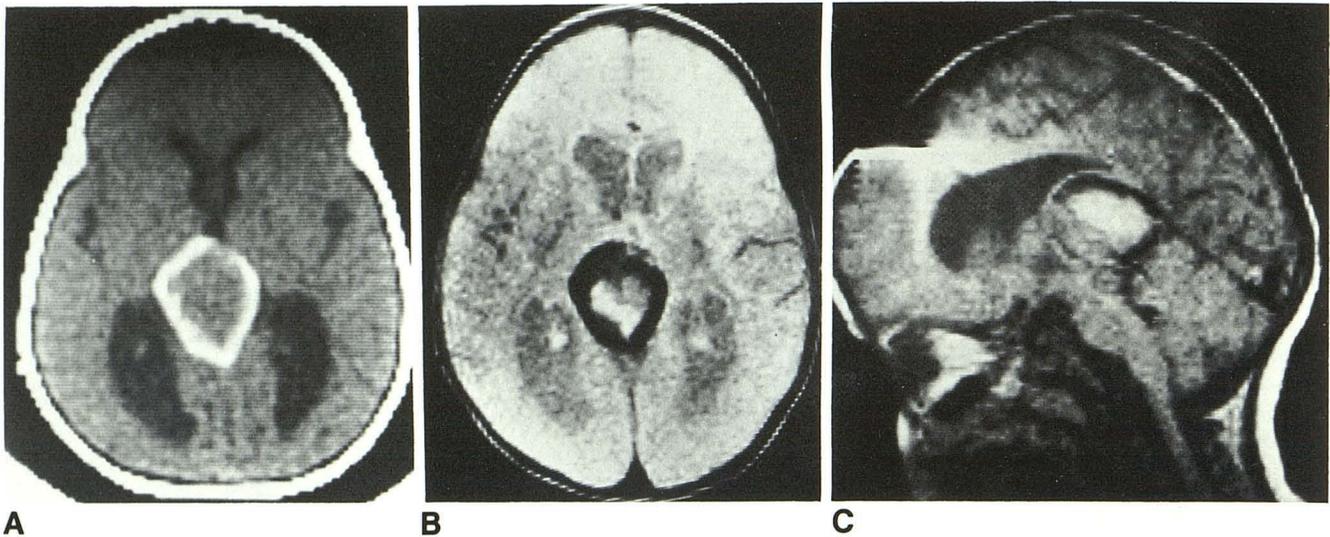
In the current case, CT and angiography documented the transformation from a partially thrombosed but still high-flow malformation to a totally thrombosed lesion. MR of the thrombosed malformation confirmed the diagnosis and, because of the lack of ionizing radiation and the ease of obtaining sagittal images, it is the optimal method for following any future regression in the size of the thrombosis.

#### ACKNOWLEDGMENT

We thank Angela Troyer and Karen Good for assistance in manuscript preparation.

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**Fig. 3.**—Follow-up CT scan and MR image at age 10 months.  
**A,** Axial nonenhanced CT scan. Interval decrease in size of lesion. Central area of more homogeneous attenuation, essentially isodense with brain. Calcification of periphery of lesion is greater and more irregular than in previous scans.  
**B,** Axial MR image, TR 2.0 sec, TE 56 msec. High-intensity central signal surrounded by rim of absent signal.  
**C,** Sagittal MR image, TR 2.0 sec, TE 56 msec. Signal characteristics same as axial MR image (Fig. 3B).

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