A Case of Unilateral Thalamic Hemorrhagic Infarction as a Result of the Vein of Galen and Straight Sinus Thrombosis

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Straight sinus vein thrombosis represents 15% of all diagnosed sinus vein thrombosis. Thrombotic occlusion of the deep cerebral venous system, the straight sinus, and the vein of Galen causes centrally located and usually bilateral thalamic infarcts. Unilateral thalamic venous infarction is extremely rare. The clinical and radiologic findings can be nonspecific and diagnostically challenging. We report a patient with this unusual condition and review the available literature. **Key Words:** Sinus—vein—thrombosis—unilateral—thalamic—infarction.

Straight sinus vein thrombosis normally causes symmetric deep venous infarction of both thalami with possible extension into the basal ganglia and white matter bilaterally.[2-5] This reflects the venous drainage of the thalami into the straight sinus via the vein of Galen. The prognosis is poor if both deep cerebral veins are affected.[3] Deep cerebral venous thrombosis causing unilateral thalamic venous infarction has a much better prognosis but is considerably rarer.[6-9] There are a number of risk factors including pregnancy and the postpartum period, infection, oral contraception, and hypercoagulable states. Risk factors present in this case include oral contraception and heterozygous factor V Leiden status.

Clinical Findings

A 14-year-old girl with learning disability and using oral contraception presented with a 3-day history of headache, vomiting, and progressive drowsiness. On the day of her admission she became drowsy, and developed a right-sided hemiparesis and slurred speech. On admission the patient was somnolent and had a severe headache. Neurologic examination demonstrated a Glasgow Coma Scale score of 13/15, unequal pupils with reduced light reflex on the left, right-sided facial weakness, reduced tone and power of the right upper (1/5) and lower (2/5) limbs, and an up going (positive Babinski) plantar reflex on the right. The patient was transferred to the local neurologic center for further treatment. Her electrocardiogram, urea, electrolytes, and full blood cell count were all within normal limits. Thalamic biopsy specimen revealed reactive gliosis only. On the second day of her admission she continued to be drowsy and developed receptive and expressive dysphasia. During the following days she improved clinically. A diagnosis of unilateral internal cerebral vein (ICV) infarction was made. The patient was subsequently found to be heterozygous for factor V Leiden.

She continued to recover, and after 3 months there was mild residual muscle weakness and some short-term memory impairment.

Discussion

Cerebral venous thrombosis is a life-threatening condition, especially if the ICVs are occluded.[3] Early diagnosis and therapy can be life saving.[11] Anticoagulation using intravenous heparin is routinely performed[1,17]; local thrombolysis remains controversial.[16,18,32]

However, even using the modern imaging tools,[8] the condition may be difficult to diagnose, because along with being rare, there are a number of other unilateral

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thalamic abnormalities that may mimic the imaging appearances. Knowledge of the imaging features is, therefore, important. Magnetic resonance (MR) imaging (MRI) with MR venography is the most sensitive imaging technique for the detection of cerebral sinus venous thrombosis.[13,14] The degree of venous congestion depends on the extent of thrombosis in the deep veins, the territory of the involved vessels, the establishment of venous collaterals, and the duration of the thrombotic occlusion. When collaterals are insufficient, cerebral edema with venous infarction may develop; this applies to deep cerebral vein thrombosis (DCVT) and to dural sinus thrombosis. Whether hemorrhagic infarction develops depends on the intensity of venous congestion.[41,42]

The clinical presentation of deep ICV thrombosis is variable and nonspecific. It can range from headache, nausea, vomiting, severe focal neurologic deficits, and seizures to coma and death.[2,3,6-10] The outcome of bilateral deep cerebral vein thrombosis can be poor and lead to death or significant long-term sequel. The outcome in unilateral deep cerebral vein thrombosis is much more favorable.[6,7,9]

In most cases of deep ICV, the thalamus is affected bilaterally.[2-4] Additional cerebral structures adjacent to the thalami such as the basal ganglia and the mesencephalon are affected in one third of patients. Computed tomography (CT) findings in deep venous thrombosis are bilateral hypoattenuation of the thalami (and, in some cases, the basal ganglia and adjacent white matter). The venous structures, such as the thalamostriate vein, ICV, vein of Rosenthal, vein of Galen, straight sinus, and confluens sinuum are hyperattenuating.[8,11,12] Hydrocephalus may occur as a consequence of edema and swelling of both thalami.[3,15] Bilateral edema of the thalami and internal capsule in the appropriate clinical context has been reported as being highly suggestive, for cerebral vein thrombosis (CVT), although it is a relatively late finding.[2,17] In cases of hemorrhagic transformation, scattered areas of hyperattenuation (usually unilateral) may be seen.

Ischemia and vasogenic edema caused by thrombosis of the deep veins appear hyperintense on T2-weighted images and hypointense on T1-weighted images. Focal hyperintense areas on T1-weighted images correspond to hemorrhage. The thrombus in a deep vein appears as an area of hyperintensity on T1-weighted images, which fails to enhance after administration of Gadolinium-Diethylenetriamine Penta-acetic acid (Gd-DTPA).

Phase-contrast MR angiography shows deep venous thrombosis as absent flow signal intensity in the deep venous system.[2,3,7] Time-of-flight MR angiography may be more difficult to interpret, because the high signal intensity of the thrombus can be mistaken for turbulent flow, although there is usually a difference in the degree of hyperintensity between flow signal intensity and thrombus.

When straight sinus venous thrombosis only causes unilateral infarction the signs are much less specific. These include hypodensity and swelling of the thalamus as a result of edema and focal areas of hemorrhage. The differential diagnoses of unilateral thalamic lesions include vascular lesions, mainly arterial thalamic infarctions.[4] Arterial infarctions typically also presents with hyperintensity on T2-weighted images and hypointensity on T1-weighted images, similar to venous edema or venous infarction. Diffusion-weighted imaging may be helpful in differentiating the two.[21] Other differential diagnoses of unilateral thalamic lesions include tumors such as germinomas, gliomas, neurocytomas,[22-25] and cavernous hemangiomas.[26,27] Particular imaging techniques and attention to specific features may be helpful in establishing the correct diagnosis. Cavernomas are usually easily identified by a typical hypointense ring on T2- and T2-weighted images.[26] Tumors can present as masses with compression of adjacent structures. Imaging changes found on MR and CT, however, are not specific. Neurocytomas may contain areas of calcifications.[24] Germinomas usually enhance[28] and may be associated with elevated levels of human chorionic

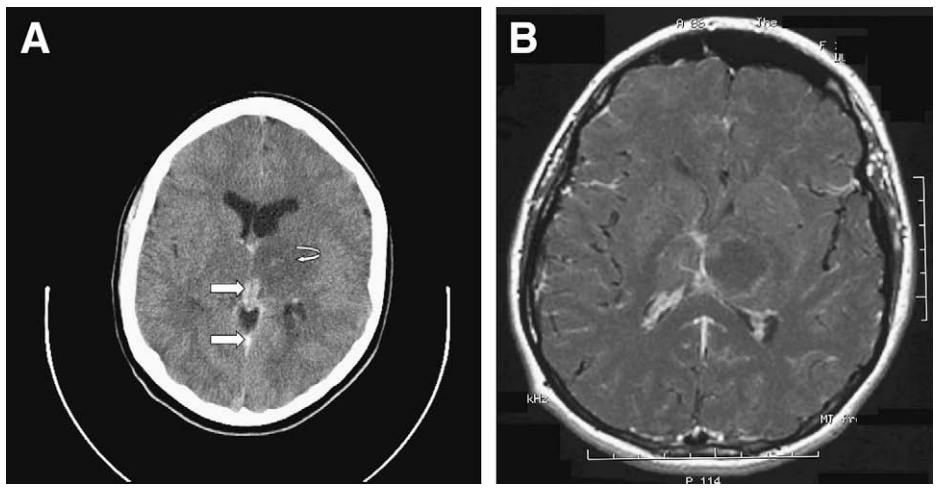

Figure 1. (A) *Noncontrast-enhanced axial CT examinations showed abnormally dense-appearing vein of Galen and straight sinus (straight arrows) mimicking contrast enhancement. Fairly well-circumscribed area of low attenuation with flecks of hemorrhage was seen in left thalamic region (curved arrow).* (B) *T1-weighted axial MRI postgadolinium shows ring of enhancement around left thalamus.*