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Developmental Venous Anomaly Complicated by Cerebral Venous Infarction

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Developmental venous anomaly is a vascular malformation thought to be a benign embryologic variant. We describe a patient who presented with focal neurological deficits and parathesia due to an infarct associated with a developmental venous anomaly with a thrombosed draining vein

Introduction

Developmental venous anomaly (DVA), previously known as cerebral venous angioma, is a vascular malformation thought to be a benign embryologic variant. Its incidence has been reported as 2.5% at autopsy. At CT and MRI, the diagnosis of DVA is suggested with visualization of a draining vein. Although usually incidentally discovered on enhanced CT or MRI of the brain, a DVA can present with headache, focal neurological deficits or bleeding. However, a nonhemorrhagic brain infarction is a rare complication, with only about 10 previous cases reported in the literature [1]. We describe a patient who presented with focal neurological deficits and paresthesia due to an infarct associated with a developmental venous anomaly and a thrombosed draining vein.

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Abbreviations: CT, computed tomography; DVA, developmental venous anomaly; FLAIR, Fluid-Attenuated Inversion Recovery; Gd, gadolinium; MRI, magnetic resonance imaging

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Case Report

A 22-year-old right-handed Caucasian woman awoke the morning of admission with slurred speech, numbness and weakness of the left side of her face, left arm and leg. She had neither headache nor visual disturbance. She was in good health prior to this event and had no past medical history. Her family history was unremarkable. Her only medication was an oral contraceptive. The symptoms lasted for about two hours and then quickly resolved.

On examination, she was afebrile with normal vital signs. She had no signs of trauma and was normocephalic. She had equal and reactive pupils and a normal cranial nerve exam. Her speech was normal and there was no facial weakness at the time of examination. She had weakness of the left upper extremity with a weak left hand grasp and decreased biceps and triceps strength. Her left leg had slight decrease in strength, but she was able to keep it up against resistance. She was able to ambulate without a limp and could toe walk and heel walk.

Routine laboratory tests, including hematology and coagulation studies, were normal. Antithrombin III levels were normal. Lupus anticoagulant and cardiolipin antibodies were absent. She had no factor V Leiden mutation or prothrombin gene mutation.

A CT of the brain before and after contrast administration showed an area of decreased attenuation within the genu and posterior limb of the right internal capsule, and a prominent enhancing vessel within the affected region of the brain. These findings represent a DVA with venous thrombosis and infarction (Fig. 1A). A subsequent brain MRI showed an acute infarction with abnormal signal on the T2 (Fig. 1B), FLAIR, diffusion (Fig. 1C), and gadolinium enhanced T1 sequences within this same area. A curvilinear, prominent vessel coursed through the infarction, representing the thrombosed DVA (Fig. 1D). Intracranial MR angiography was unremarkable, showing no aneurysm or stenosis of the cerebral arteries or vertebrobasilar system. She was discharged to home after being admitted overnight for observation.

Discussion

Developmental venous anomalies, previously termed venous angiomas, represent the most common cerebral vascular anomaly. The term "developmental venous anomaly" was coined by Lasjaunias et al. [2] after suggesting that venous angiomas are actually embryologic variants of venous drainage instead of true vascular anomalies. These lesions are thought to represent an arrest of venous development after arterial development is nearly complete or a thrombosis of the developing venous drainage in a specific region. The process results in the retention of primitive, embryologic medullary veins that drain into a single, large draining vein, the DVA [3]. The region drained by a DVA has no other normal venous drainage. DVAs lack an arterial component and have unaffected intervening neural parenchyma, which distinguishes these lesions from arteriovenous malformations [4]. About one third of these lesions are located in the cerebellum and in the brainstem; the remaining are supratentorial.

DVAs have a characteristic appearance on angiography, CT and MRI. In the late venous phase of angiography,

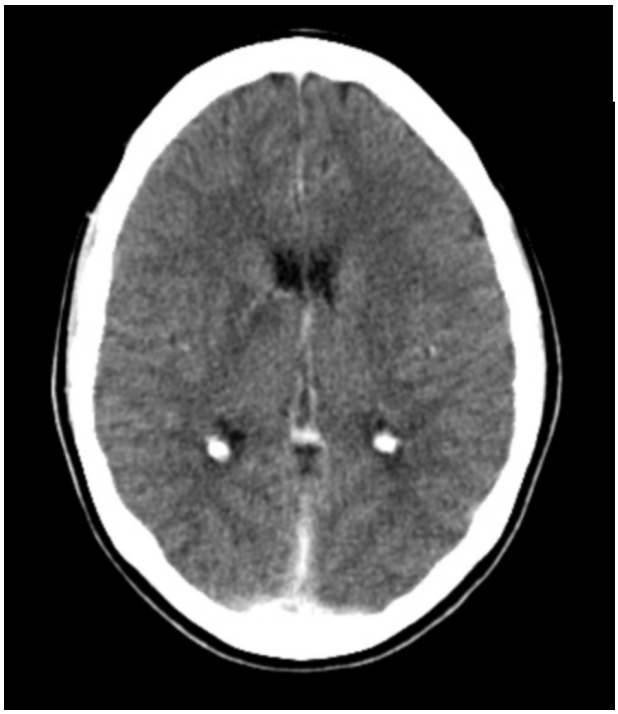

Figure 1A. 22-year-old woman with developmental venous anomaly. Axial contrast-enhanced CT reveals an area of decreased attenuation within the genu and posterior limb of the right internal capsule, with a prominent enhancing vessel within the central portion of the lesion, consistent with a nonhemorrhagic infarction from a developmental venous anomaly.

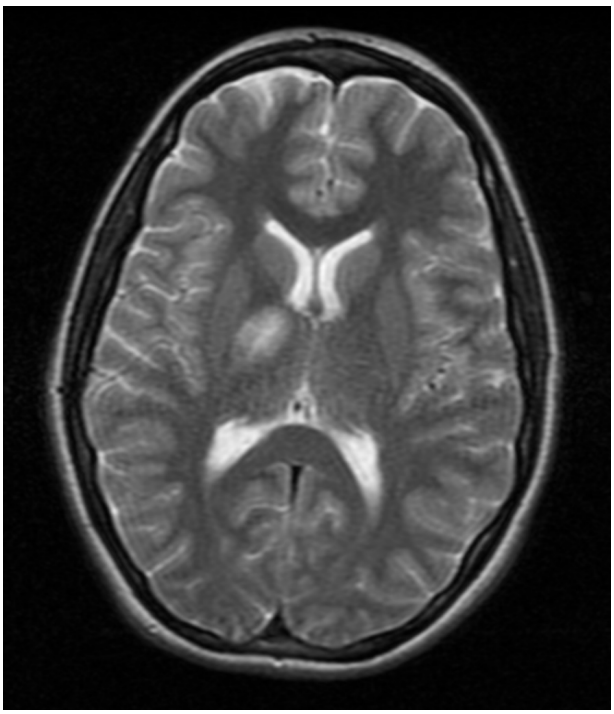

Figure 1B. Axial fast spin echo T2 weighted MRI image shows the abnormal increased signal within the infarction.

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several radially arranged dilated veins converge in a central large caliber central vein to form a caput medusae image [1]. On contrast-enhanced CT, the draining vein of a venous angioma appears as an enhancing linear or curvilinear structure, typically coursing from the deep white matter to a cortical vein, a deep vein, or to a dural sinus [3]. DVAs demonstrate low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Following gadolinium administration there is marked enhancement of the main collecting vein and peripheral radial veins [1].

Venous angiomas are often asymptomatic and therefore are often found incidentally. The most frequent signs include headache, seizure, focal neurological deficits and dizziness. Thrombosis of the draining vein of the malformation is an extremely rare complication leading to nonhemorrhagic venous infarction. To our knowledge, only ten previous cases of a nonhemorrhagic brain infarction due to a DVA have been reported in the literature [1].

Hammoud et al. [5] believe that the same predisposing factors for dural sinus thrombosis in the central nervous system apply for the thrombotic event associated with a DVA, including oral contraceptive use and hereditary hypercoaguable states such as protein C or S deficiency, antithrombin deficiency, or factor V Leiden mutation. Our patient had a negative hypercoagulability screen, but used oral contraceptives, which may have contributed to her thrombotic event involving the DVA.

Developmental venous anomalies are usually thought of as incidental findings on brain imaging. However, infarctions are a known complication of developmental venous anomalies. Non-hemorrhagic infarction is a potential complication of a DVA that should be considered in the differential diagnosis of a bland infarct, where a thrombosed draining vein may be identified.

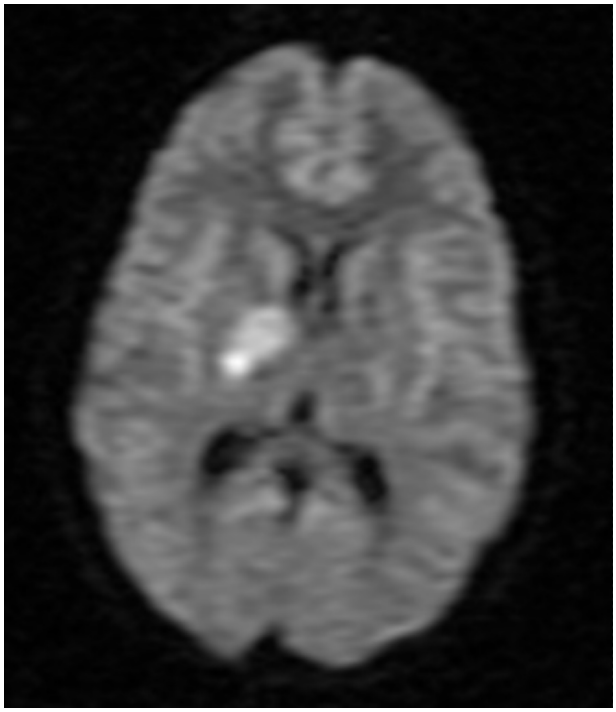

Figure 1C. Axial MRI diffusion weighted sequence shows the abnormal increased signal within the infarction.

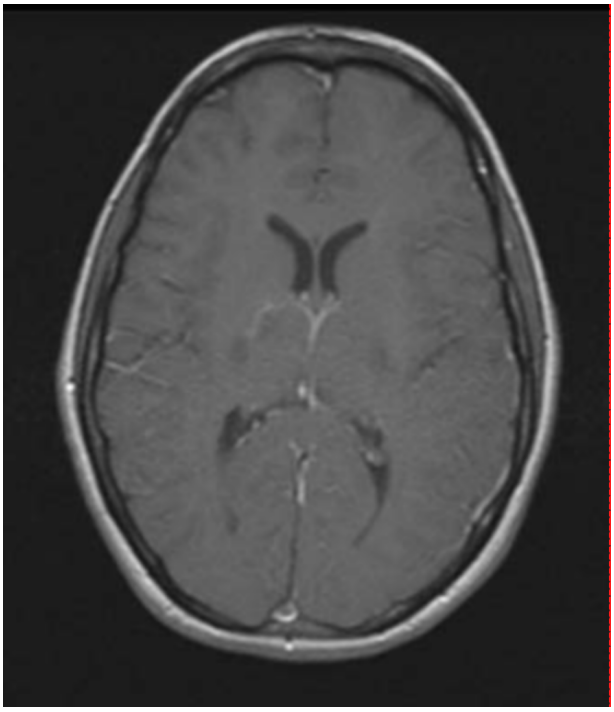

Figure 1D. Axial Gd-enhanced T1 weighted MRI sequence shows abnormal increased signal intensity within the same affected region as the CT scan, with a curvilinear, prominent vessel coursing through the infarction, consistent with a nonhemorrhagic infarction.

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