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Neuroimaging Highlight

Developmental Venous Anomaly Thrombosis Presenting with Intracerebral Hemorrhage

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We present the case of a 63-year-old female with no significant medical history who experienced sudden-onset incoherent speech and vision changes at home, following a 2-day history of headaches and emesis. Upon arrival at a Regional Stroke Center, she was normotensive (127/85 mmHg), with aphasia and a right homonymous hemianopsia. Initial bloodwork demonstrated a platelet count of 234×10^9/L, an International Normalized Ratio (INR) of 1.0 and a prothrombin time (PTT) of 18.

A non-contrast CT scan demonstrated an intraparenchymal hemorrhage within the left temporal lobe, with serpiginous hyperdense veins extending from the hematoma to the cortex and atrial trigone distribution with mild peripheral enhancement (Figure 1A,B). The differential, based on the CT findings, included partially thrombosed veins with associated hemorrhagic infarct and/or dural venous anomaly with associated hemorrhage. There was a localized mass effect with effacement of the left ambient cistern and temporal horn, without transtentorial herniation or midline shift. Follow-up magnetic resonance venography (MRV) revealed a thrombosed developmental venous anomaly (DVA) with "caput medusae" (Figure 1C,D) located in the depth of the left temporal lobe, draining via two large collecting veins into an atypical left vein of Labbe and terminating in the left transverse sinus. There was intracerebral hemorrhage with vasogenic edema, focal mass effect and cortical gyriform subacute ischemia. No associated cerebral cavernous malformation (CCM) was identified. A prothrombotic workup was negative.

DVAs, the most common vascular malformation in the brain, are congenital formations consisting of radially oriented medullary veins converging into a central draining vein, resulting in the characteristic "caput medusae" pattern.[1] The overwhelming majority are asymptomatic and incidentally discovered, but rare symptomatic presentations may arise from hemorrhagic or ischemic events.[2,3] In DVA thrombosis, a hyperdense collecting vein on non-contrast CT can be an early radiological indicator, prompting further evaluation.[1,4] On MRI, thrombosis results in alterations in flow voids and phase-shift artifacts affecting the collecting vein and its larger venous radicles.[5] The thrombus may demonstrate high signal intensity on T1-weighted sequences, while susceptibility-weighted imaging enhances the detection of microhemorrhages due to venous congestion. MRV further delineates the affected venous architecture, typically revealing absent or diminished flow within the thrombosed segment.[1] In cases where no thrombus or CCM is identified as the source of hemorrhage, conventional angiography should be considered to rule out an underlying arteriovenous shunting lesion.[6] Moreover, as symptomatic DVAs frequently co-occur alongside other vascular or structural abnormalities,[7] a multimodal neuroimaging approach is essential for morphological characterization, risk stratification and differentiation of DVAs from other vascular pathologies.

The primary management of DVA thrombosis is anticoagulation to prevent thrombus progression and promote recanalization of the collecting vein.[8,9] In the event of hemorrhage requiring surgery, including those with another associated vascular malformation, the preferred approach involves hematoma evacuation with preservation of the DVA, as its removal may lead to catastrophic venous infarct.[8,9] In this case, the patient was anticoagulated with heparin and then eventually bridged to warfarin. After a brief hospitalization and stroke rehabilitation, a follow-up MRI at 4 weeks demonstrated recanalization of the DVA (Figure 2), with marked neurological improvement.

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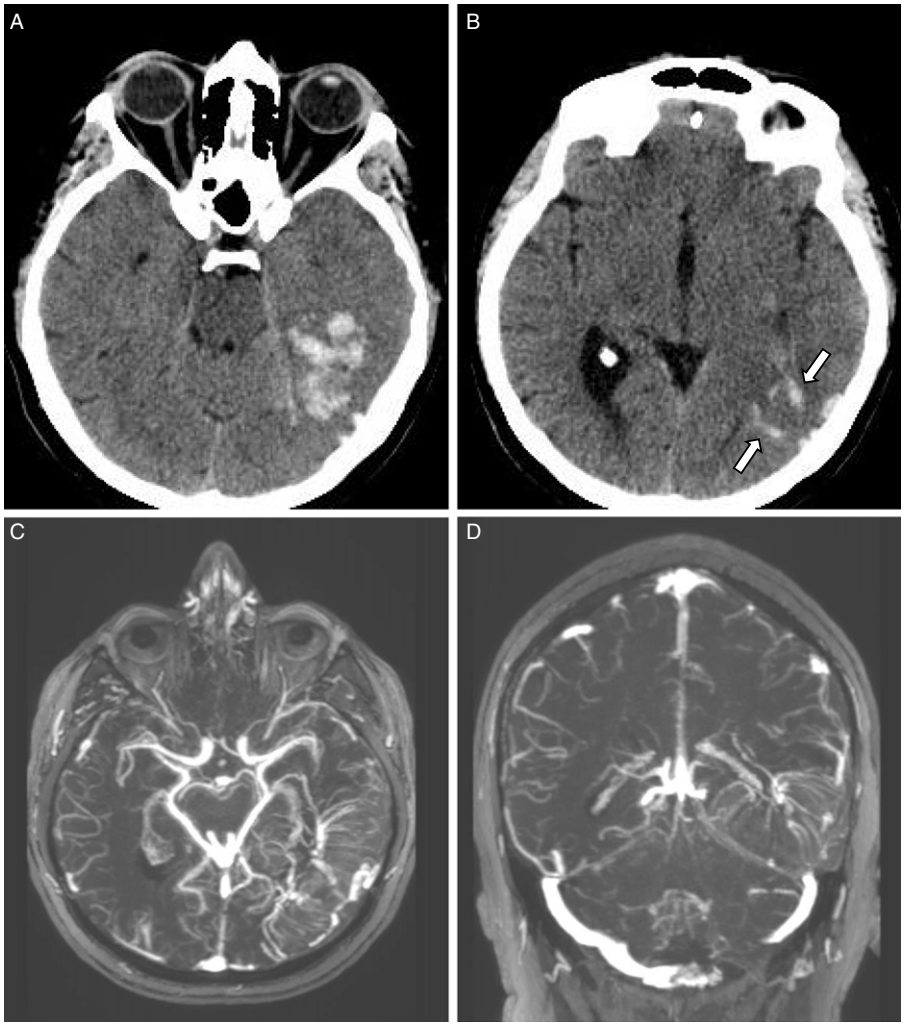


Figure 1. Non-contrast CT head identifying (A) a left temporal hematoma and (B) a linear hyperdense thrombus in the two transcerebral veins forming the developmental venous anomaly (DVA) (arrows). Axial (C) and coronal (D) contrast-enhanced magnetic resonance venography images demonstrating the caput medusae of the DVA and the lack of opacification of the transcerebral thrombosed veins.

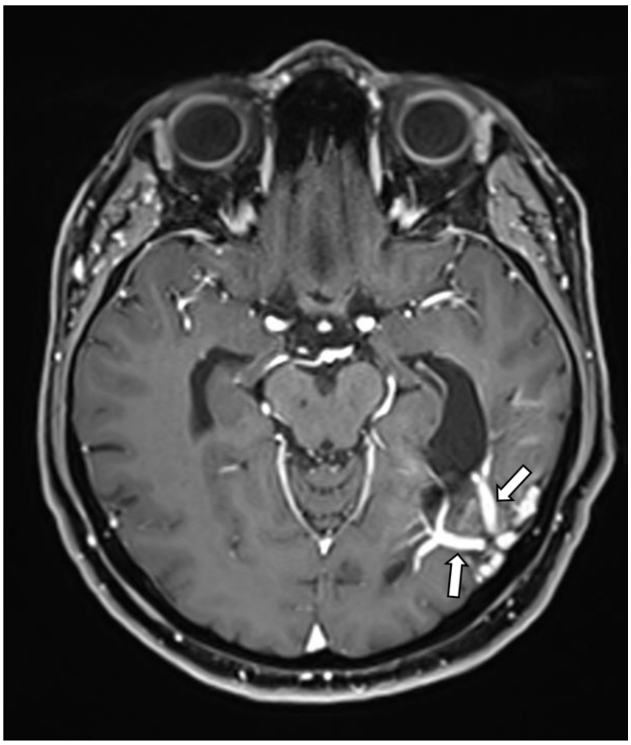


Figure 2. Follow-up contrast-enhanced 3DT1 MRI at 4 weeks demonstrating complete recanalization of the previously thrombosed transcerebral veins.

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