Cerebral arteriovenous malformation. A case report

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Background: Cerebral arteriovenous malformations (AVMs) are rare and complex intracranial vascular lesions, with an estimated population prevalence ranging from 0.01% to 0.05%, and a detection rate of approximately 1–1.4 cases per 100,000 person-years. They are characterized by direct arteriovenous shunting through a nidus without an intervening capillary bed. Their imaging characterization requires assessment of the nidus architecture, arterial feeders, venous drainage pattern, and associated vascular or parenchymal abnormalities. Multimodality imaging, including computed tomography, magnetic resonance imaging, CT and MR angiography, and digital subtraction angiography, plays a central role in diagnosis and treatment planning. Specific vascular features, including deep venous drainage, venous stenosis, intranidal aneurysms, deep nidus location, and evidence of prior hemorrhage, have important implications for hemorrhagic risk and therapeutic management. Accurate recognition of these findings and differentiation from other intracranial vascular lesions are therefore essential for appropriate clinical decision-making.

Keywords: Cerebral arteriovenous malformation, cerebral angiography, deep venous drainage, Vascular nidus.

Case Report

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Brain arteriovenous malformations (bAVMs) are complex congenital vascular anomalies characterized by a direct connection between feeding arteries and draining veins through a vascular nidus, lacking an intervening capillary bed (1). Although their general prevalence is low, they constitute one of the primary causes of non-traumatic hemorrhagic stroke in young adults (2). The clinical presentation ranges from severe intracranial hemorrhage or seizures to completely incidental findings on routine neuroimaging studies (2,3).

Successful clinical management and risk stratification critically depend on precise angioarchitectural characterization (1,3). While digital subtraction angiography remains the reference standard, contemporary non-invasive techniques—such as computed tomography angiography (CTA) and time-resolved magnetic resonance angiography (MRA)—allow for the detailed evaluation of morphological biomarkers associated with hemorrhagic risk (1,2). Key imaging factors include the presence of intranidal aneurysms, venous stenosis, deep venous drainage, and deep location of the nidus (2,3).

Despite advanced diagnostic tools, therapeutic decision-making poses a critical dilemma where interventional risks must be carefully weighed against the natural history of the lesion (3). Therefore, the objective of this article is to report an unusual clinical case of bAVM, highlighting the crucial role of contemporary advanced neuroimaging in its accurate diagnosis and therapeutic planning.

Case report

A 19-year-old female university student, with no significant medical history, originally from and residing in Quetzaltenango, Guatemala, presented with acute-onset headache, after which she decided to go to sleep. When her mother attempted to awaken her, she was found unconscious and unresponsive to stimuli. She was subsequently transported to our institution by the Volunteer Fire Department.

On admission, her blood pressure was 90/60 mmHg, temperature 36.5 °C, heart rate 105 beats/min, and oxygen saturation 96%. She was unconscious with a Glasgow Coma Scale score of 11. Her pupils were isocoric and reactive to light. Cardiac auscultation revealed a regular rhythm without murmurs. The remainder of the physical examination was unremarkable. Initial laboratory studies showed a white blood cell count of 14.50 × 10^3/µL, neutrophils 78.8%, lymphocytes 13.6%, hemoglobin 16.0 g/dL, and platelet count 295 × 10^3/µL.

Given the acute neurological presentation, a non-contrast brain computed tomography (CT) was performed, demonstrating an acute intraparenchymal hemorrhage with extension into the ventricular system. Subsequently, cerebral CT angiography was performed, demonstrating a vascular nidus located in the left temporal and perimesencephalic regions, with arterial feeders arising from the left posterior cerebral artery (P2 segment) and the left internal carotid artery (cavernous segment, C4). Deep venous drainage was observed, with dilatation of the ipsilateral internal cerebral vein and vein of Galen. These findings were consistent with a cerebral arteriovenous malformation (AVM), Spetzler-Martin grade III. During

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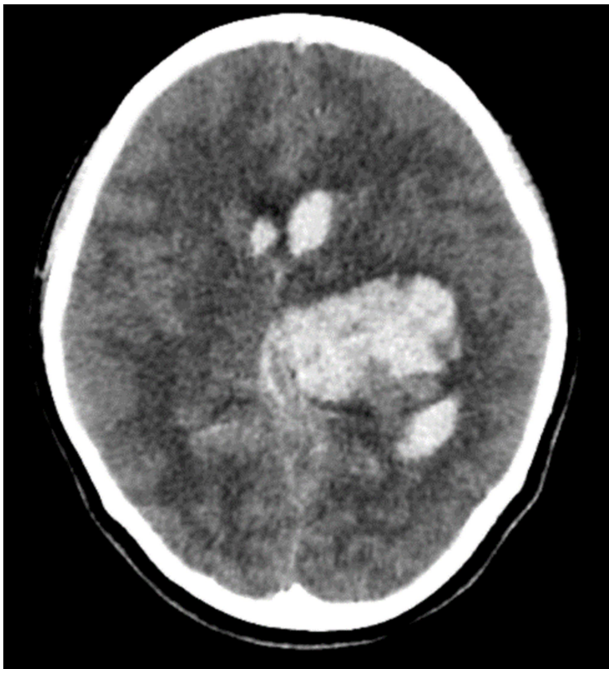

Figure 1. At the level of the left thalamus and basal ganglia, an amorphous, hyperdense image is observed, with attenuation coefficients up to +65 HU. In the axial plane, it measures 2.6 cm anteroposteriorly and 4.3 cm laterolaterally; in the coronal plane, it measures 3.4 cm craniocaudally, with a volume of 19.8 cc. These findings are consistent with an acute intraparenchymal hemorrhage. It exerts a mass effect, causing a 4.5 mm midline shift to the right."

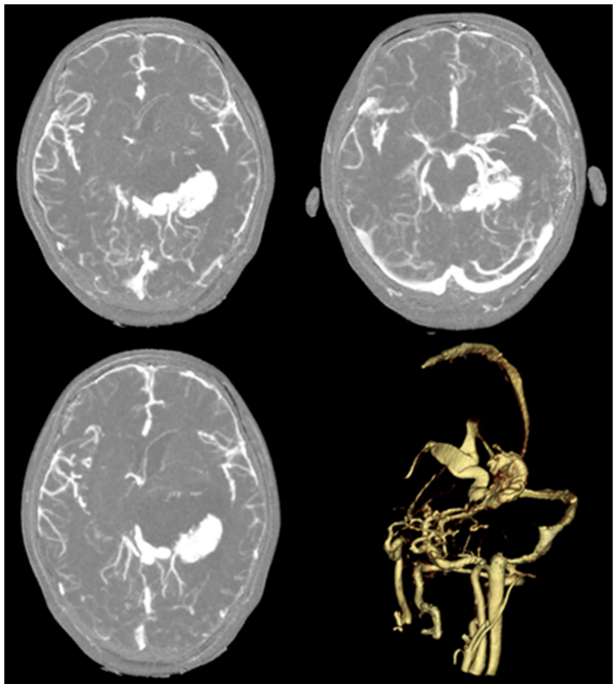

Figure 2. In the left temporal and perimesencephalic region, an amorphous, nidus-like image is observed, which appears slightly hyperdense on the non-contrast phase and shows homogeneous enhancement following intravenous contrast administration. This nidus is supplied by feeding arteries originating from the P2 segment of the left posterior cerebral artery and the cavernous segment (C4) of the left internal carotid artery, causing dilation of the ipsilateral internal cerebral vein and the vein of Galen. The area occupied by the nidus measures approximately 3.0 x 1.4 cm (axial plane) and 3.2 cm (coronal plane). These findings are highly suggestive of a Spetzler-Martin Grade III arteriovenous malformation (medium size: 2 points; eloquent location: 1 point; deep venous drainage: 1 point).

hospitalization, the patient developed cardiopulmonary arrest on the fourth day, with unsuccessful resuscitation, and subsequently died

Discussion

Cerebral arteriovenous malformations (bAVMs) represent a critical neurovascular challenge, with acute intracranial hemorrhage being their most lethal clinical presentation. As documented in the literature, the catastrophic clinical course observed in young populations—characterized by acute severe headache followed by sudden loss of consciousness—correlates directly with the massive mass effect and elevated intracranial pressure exerted by spontaneous intraparenchymal bleeding (2). When the hemorrhage extends into the ventricular system, it warrants a high-grade Fisher classification, which carries a severely guarded prognosis due to the imminent risk of acute hydrocephalus and intracranial hypertension. A meticulous angioarchitectural assessment through emergency computed tomography angiography (CTA) is fundamental to establishing the definitive vascular etiology behind these spontaneous hematomas (1). identifying specific morphological features is paramount to understanding lesion vulnerability and predicting high-risk rupture dynamics (2). High-risk anatomical biomarkers, such as a deep-seated nidus, feeding pedicles arising from major cerebral arteries (such as the posterior cerebral or internal carotid segments), and exclusive deep venous drainage pathways (involving the internal cerebral vein or the vein of Galen), significantly compound the hemodynamic stress on anomalous vessels (2).

The implementation of emergency CTA in acute neurovascular settings remains a cornerstone of the diagnostic workup, providing comprehensive data within a short timeframe (1,3).Multi-detector CTA offers excellent spatial resolution capable of performing a rapid three-dimensional mapping of the vascular tangle, confirming the direct communication between feeding arteries and draining veins without an intervening capillary bed (1). Ultimately, despite advanced neuroimaging and aggressive intensive care monitoring, the clinical outcome of ruptured deep-seated bAVMs can be rapidly fatal due to the severity of the primary hemorrhagic insult and subsequent secondary brain injury. This tragic scenario highlights that while contemporary non-invasive imaging techniques excel at defining complex vascular anatomy, therapeutic decision-making remains a dilemma once an acute rupture occurs (3).

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Documenting these presentations is essential to further understand the morphologic variables of high-risk nidi—particularly those involving deep feeding branches—and to stress the need for early detection before catastrophic clinical deterioration takes place.

Conclusion

Cerebral arteriovenous malformations are rare vascular lesions that may present with life-threatening intracranial hemorrhage in young patients. Multimodality imaging is essential for accurately characterizing the nidus, arterial feeders, and venous drainage, as these findings are critical for diagnosis, Spetzler-Martin grading, and treatment planning. In this case, the combination of intraparenchymal and intraventricular hemorrhage with a Spetzler-Martin grade III AVM and deep venous drainage highlights the importance of prompt radiological diagnosis and characterization.

Conflicts of interests

The authors declare that there are no financial, personal, or institutional conflicts of interest that could have influence the work reported in this manuscript.

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