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CASE REPORT

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Delayed diagnosis of basilar artery occlusion in a 28-year-old postpartum woman

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Abstract

Background Basilar artery strokes, which are often challenging to diagnose due to their diverse symptoms and uncommon occurrence, demand timely identification to mitigate severe consequences.

Case presentation This case report discusses a 28-year-old woman who was admitted 7 months postpartum for suspected status epilepticus. Her initial presentation of convulsive activity and encephalopathy prompted an initial differential diagnosis including toxin-related seizures, encephalitis, and serotonin syndrome among others. A CT angiogram revealed nonocclusive basilar artery thrombosis and hypodensities in the basal ganglia and thalamus. MRI revealed additional strokes in the PCA and SCA territories.

Conclusions The patient's age and recent history of preeclampsia during pregnancy prompted pertinent discussions regarding pregnancy-related strokes. Most importantly, this case sheds light on the diverse presentations of basilar artery strokes and stresses the urgency of swift identification using imaging to avoid significant morbidity and mortality.

Keywords Basilar artery stroke, Hyperdense basilar artery sign, Stroke in the young, Pregnancy-associated stroke, Preeclampsia

Background

Basilar artery occlusions (BAOs) pose a diagnostic challenge due to their rarity and diverse clinical manifestations and often lack specific symptoms [1, 2]. Unlike other cerebral artery strokes, BAOs are predominantly identified through imaging rather than clinical presentation [1]. Clinical presentation, including convulsive activity, altered mental status, and dizziness contributes to uncertainty in timely identification, particularly when compared to anterior circulation strokes [3, 4]. Patients with ischemic stroke presenting with reduced levels of consciousness are at heightened risk of misdiagnosis or delayed identification [5]. Notably, BAOs can manifest as involuntary convulsive-like movements (ictus), easily mistaken for seizures or status epilepticus [6]. In our case report, we discuss the case of a young woman initially thought to be presenting with toxidrome or status epilepticus due to her reduced level of consciousness and reported single convulsion at the scene. This case emphasized the need for a high index of suspicion for BAOs despite nonspecific symptoms, while also underscoring the significance of considering further imaging when a patient's presentation lacks clarity.

Case presentation

A 28-year-old woman was brought to the Emergency Department (ED) of a comprehensive stroke center by Emergency Medical Services (EMS) after her family found her unresponsive on the ground with her baby beside her. Our patient underwent a cesarean section at

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35 weeks for preeclampsia 7 months prior to her presentation. Her mother reported that she was completely normal approximately 30 min prior to the event, and upon finding her on the floor, she observed convulsive movements. EMS treated her for seizures with midazolam and her neurologic exam was reported to have improved enroute to the ED; however, the patient remained comatose with bilateral clonus and dysconjugate gaze on arrival. Another family member, in retrospect, reported right facial drooping and noticed reduced movement on her right side compared to the left while the patient exhibited convulsive movements at home, although her mother and EMS did not observe these findings.

The patient had no prior history of seizures or thromboembolic events. She did have a known complex social history that included heavy alcohol use and domestic violence in the months prior to presentation, although her family reported that she had recently been stable regarding her mood and did not suspect an intentional overdose. The patient fell and briefly lost consciousness weeks to months prior to arrival during an altercation with her partner; an event with little further detail provided by family. Her home medications included clonidine, fluoxetine, and hydroxyzine, with recent dosage increases in clonidine and fluoxetine in the weeks preceding admission. Her urine drug screen on arrival was positive for benzodiazepines, cannabinoids, and ethanol (Quant < 10 mg/dL). Initially presenting with seizure-like activity and clonus, the differential diagnosis in the ED included seizures, serotonin syndrome, and intracranial hemorrhage. She was treated with midazolam, lorazepam and levetiracetam in the ED while preparing for urgent head CT. A head CT scan without contrast performed approximately 4.5 h after arrival was read as showing no acute intracranial abnormality.

Neurology consultation was prompted by concerns for seizures or serotonin syndrome, although toxicology consultation suggested that this was unlikely. The subsequent neurological exam conducted by neurology, while limited by the patient's recent intubation and level of sedation, revealed a comatose patient with movement limited to spontaneous right upper extremity, hyperreflexia throughout, and bilateral lower extremity clonus and triple flexion. A non-contract CT head and CT Angiogram (CTA) of the head and neck were recommended which identified a non-occlusive basilar artery thrombus and hypodensities in the basal ganglia and thalamus (Figs. 1 and 2). This CTA with delayed phase additionally showed patent dural venous sinuses and a patent deep cerebral venous system, ruling out venous sinus thrombosis.

An urgent MRI was performed which revealed diffusion restriction on DWI/ADC with FLAIR changes suggestive of completed infarcts in the bilateral thalami, basal ganglia, right cerebellum, and bilateral posterior cerebral artery (PCA) territories (Fig. 3).

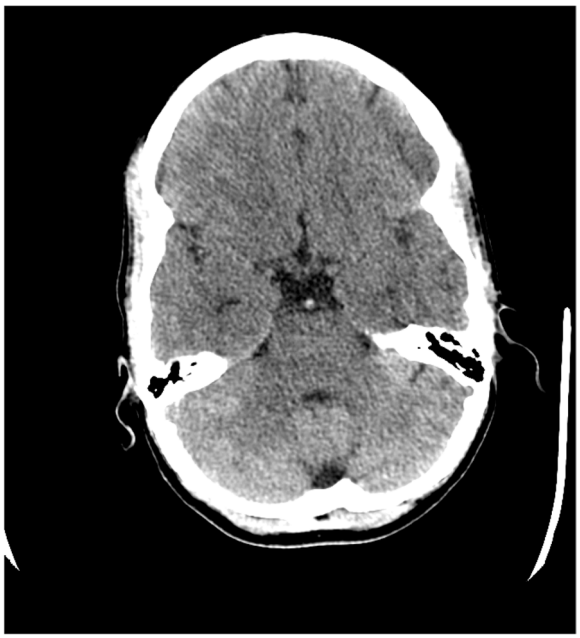

Fig. 1 Hyperdense basilar artery sign on CT head

Interventional Neuroradiology was consulted immediately after stroke diagnosis; however, they did not offer endovascular therapy as risks of the procedure would outweigh benefits in the setting of diffuse stroke burden and completed infarcts. She was started on a low intensity heparin drip upon identification of the thrombus. Despite aggressive medical management, the patient's neurological examination continued to decline with extensor posturing in the bilateral upper extremities and triple flexion in the bilateral lower extremities, prompting emergent posterior fossa decompression and external ventricular drain placement 2 days after admission.

To evaluate for etiology, a comprehensive stroke evaluation was completed including TTE with bubble study, pulmonary CTA, and continuous telemetry which ruled out patent foramen ovale, pulmonary arteriovenous fistula, and cryptogenic atrial fibrillation. Due to the lack of an obvious cause for this patient's stroke, further evaluation was conducted two weeks after admission. A liver ultrasound and CT scan of the abdomen and pelvis were performed, which ruled out a hepatic portal arteriovenous fistula. The initial hypercoagulable workup showed elevated levels of anticardiolipin IgM and Beta-2 glycoprotein IgM, two antibodies that are acute phase reactants; these tests normalized upon retesting two months after the initial insult. Tests for Factor V Leiden PCR and Prothrombin Gene 20,210 A returned negative. Three 12-hour continuous EEGs

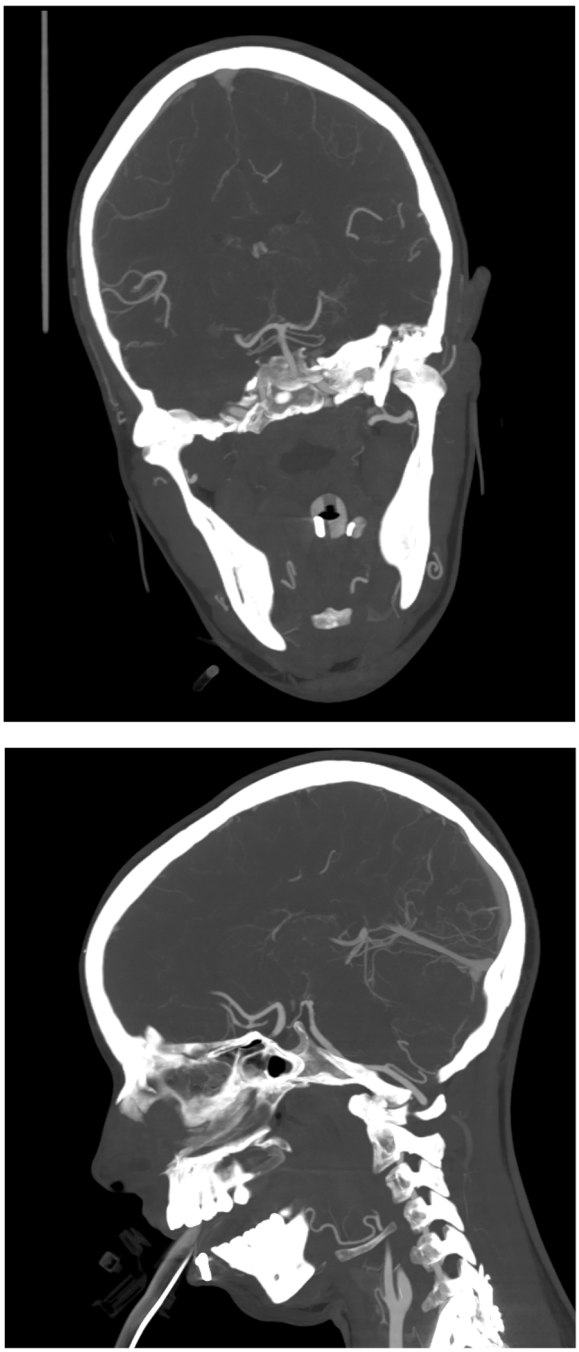

Fig. 2 Sagittal and Coronal images of the CTA head and neck

were negative for seizure activity within the first month of hospitalization. MRA with fat saturation was negative for basilar artery dissection which had been suspected based on CTA.

Her hospital course was notably complicated by elevated high sensitivity troponin to 15,213 with ST depressions in inferior and lateral leads within the first 6 h of arrival, consistent with NSTEMI (Fig. 4).

There was initial concern for pregnancy-related cardiomyopathy versus spontaneous coronary artery dissection given proximity to pregnancy, however a TTE showed left ventricular dysfunction with relative sparing of the apex most consistent with takotsubo variant stress cardiomyopathy (Fig. 5).

Within 8 days of admission, her ejection fraction and left ventricular function had normalized, consistent with reversible stress cardiomyopathy. A CTA of the coronary arteries was initially ordered to be completed once the patient stabilized; however, this study was canceled by cardiology given her LV function spontaneously normalized on repeat TTE.

Over 2 months of hospitalization, additional complications arose including ventilator-acquired pneumonia leading to sepsis and reactive critical thrombocytosis. Long-term management required tracheostomy and percutaneous endoscopic gastrostomy tube placement. She was discharged on apixaban with the mechanism "cryptogenic" but with high suspicion for a cardioembolic source or pregnancy related hypercoagulability. Prior to discharge there was improvement in her neurological status, with an NIHSS score decreasing from 29 at admission to 15, with her neurologic exam notable for being alert and following simple commands, though she remained mute with a right gaze preference and quadriparesis [7]. The patient was deemed able to participate in acute rehab despite severe motor deficits and was accepted to a specialized facility.

Discussion and conclusions

Our case highlights a significant diagnostic challenge in a young postpartum woman with delayed recognition of basilar artery occlusion (BAO). While previous reports note BAO presentations involving convulsive activity, these predominantly involve individuals aged 40 or older, with a mean age exceeding 50 [8, 9]. To our knowledge, there are no case reports discussing delayed diagnosis of cardioembolic basilar artery occlusion in a young postpartum woman who initially presented with presumed status epilepticus.

The cause of this patient's acute cerebral infarction has two possible explanations. The first is the presence of embolization to multiple vessels, including the basilar artery. The second explanation is the thrombus was first occlusive and by the time CTA head was completed, the thrombus had become nonocclusive. While the bilateral thalamic infarcts could have been from embolization to an artery of Percheron, such an anatomic variant would not explain her PCA territory infarcts. In addition to considering the path of the intracranial thrombus, multiple

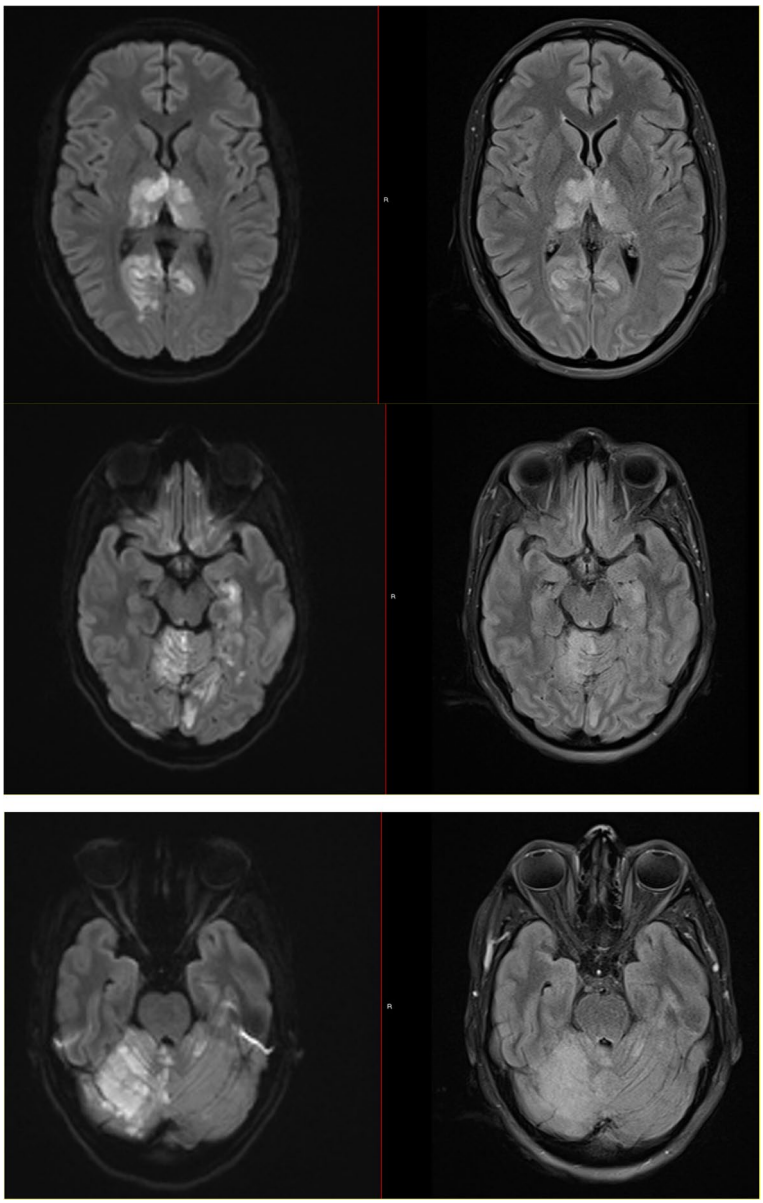

Fig. 3 Fast Stroke MRI with DWI (left) and FLAIR (right) demonstrating extensive damage present in the posterior circulation territories, including the bilateral thalami

underlying etiologies to explain this patient's stroke were considered.

Outside of the placement of a NuvaRing 5 months prior to admission, a contraceptive device that has at least the same amount of prothrombotic potential as combined oral contraceptives, our patient has no other obvious risk factors for hypercoagulability [10]. Multiple etiologies of her stroke were considered including pregnancy related hypercoagulability, in-situ thrombus formation, vasculopathy involving progression to the PCAs, and less likely traumatic thrombotic dissection based on negative MRA findings. In this case, the stroke etiology seems to point towards an embolus from a proximal source, likely cardiac in origin. We believe the recent increase in her clonidine and fluoxetine dosages prompts consideration that the patient's perceived anxiety might have been misinterpreted tachycardia from an underlying peripartum versus stress cardiomyopathy. Preeclampsia patients commonly exhibit asymptomatic left ventricular (LV) systolic dysfunction, diastolic dysfunction, and LV hypertrophy persisting for a minimum of one year after childbirth, with the possibility of progressing to congestive heart failure

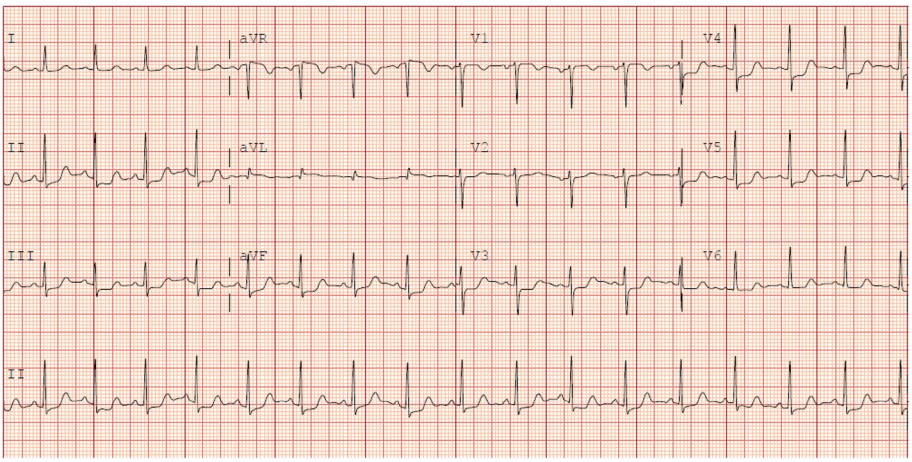

Fig. 4 EKG

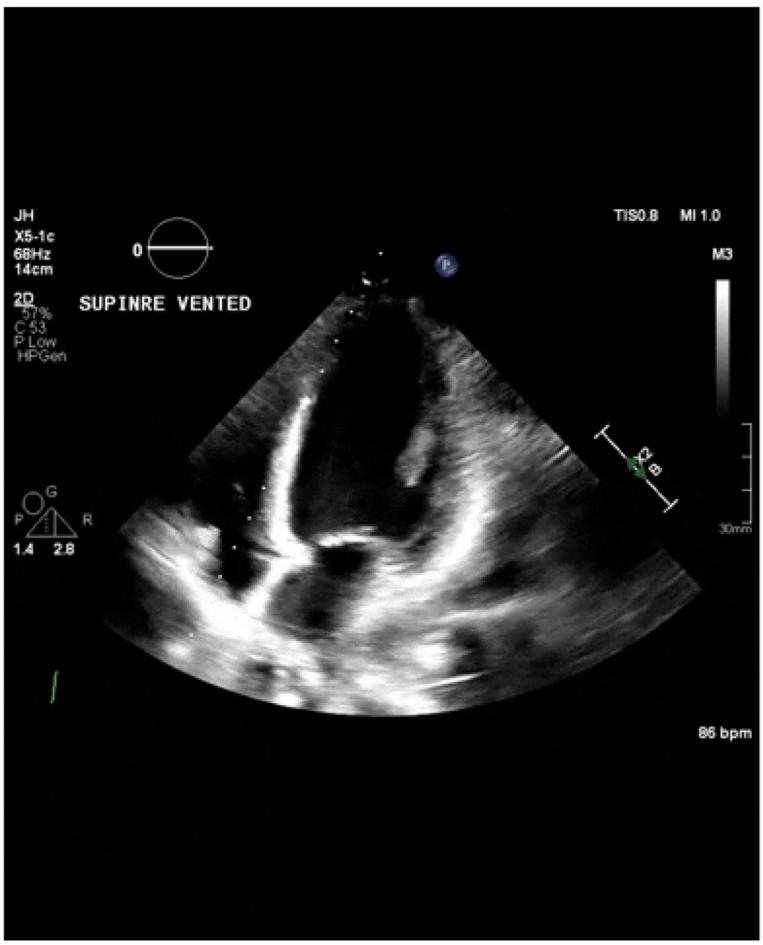

Fig. 5 TTE CINE

[11]. This trend is particularly notable in cases of preterm preeclampsia, as observed in our patient [11, 12]. In addition to the possibility of peripartum cardiomyopathy, our patient was shown to have takotsubo variant stress cardiomyopathy, which is a known risk factor for stroke as well [13]. Finally, spontaneous coronary artery dissection (SCAD), especially pregnancy-related, is a possible cause of this patient's NSTEMI and stroke, although not suspected given spontaneous resolution of symptoms [14, 15].

The potential impact of our patient's cesarean delivery seven months before her presentation on her ischemic stroke remains uncertain. Pregnancy-associated stroke independent of preeclampsia typically manifests during the postpartum period, primarily within 10 days of hospital discharge, with rare occurrences beyond 60 days post-discharge [16]. Notably, the incidence of pregnancy-associated stroke appears to be rising, likely correlated with an increased prevalence of hypertensive disorders during pregnancy [16]. Our patient had a history of gestational hypertension which progressed to preeclampsia necessitating early delivery, establishing a pertinent risk factor for pregnancy-associated stroke. While a presentation of pregnancy-associated stroke seven months post-delivery is sometimes considered to be atypical, it is known that the risk for major adverse cardiovascular events (including stroke) in women with preeclampsia is significantly increased for ≥ 3 years after delivery [11]. As evidence grows, it is becoming more apparent that a history of preterm preeclampsia identifies women at high cardiovascular risk even in the absence of other concomitant risk factors [12]. As such, it is imperative to consider cardiovascular dysfunction secondary to pregnancy and preeclampsia as the etiology of this patient's otherwise cryptogenic stroke.

Despite being rare, accounting for only about 1% of ischemic strokes, BAOs typically yield poor prognosis [8, 17, 18]. Studies show grim outcomes in BAO cases with a mortality rate ranging from 31.1 to 51.7%, marking it among the highest for large vessel ischemic strokes [19]. Moreover, over 65% of BAO survivors face severe residual deficits, underscoring the substantial morbidity associated with this stroke type [18]. Given their catastrophic nature, swift diagnosis remains paramount as prompt treatment significantly improves outcomes [20, 21]. However, misdiagnosis of strokes occurs frequently, particularly with posterior circulation strokes like BAOs which are misidentified nearly three times as often as anterior strokes [3, 4].

A thorough neurologic examination, including assessment for pathological signs such as the Babinski sign, is essential for identifying manifestations suggestive of intracranial pathology. In this patient's case, the initial neurologic examination in the emergency department did not report on Babinski's sign findings. However, a positive Babinski sign likely would not have altered the differential diagnosis, as both serotonin syndrome and seizures can present with this sign. Additionally, sedation could obscure its interpretation. Beyond the physical examination, non-contrast head CT scans remain the primary for the initial assessment of neurological symptoms. However, early imaging often lacks sensitivity in identifying ischemic changes, a problem that is amplified in posterior circulation strokes [2]. While a hyperdense basilar artery sign on non-contrast CT may indicate a basilar thrombus, its subtle nature and low sensitivity due to proximity to osseous structures can lead to oversight [2]. Therefore, complementing initial imaging with additional modalities like CTA or MRI becomes crucial for accurate detection of basilar artery occlusion (BAO).

Our patient's presentation of seizure-like activity aligns with reported manifestations of BAO [3, 6, 8, 9]. However, the literature describes a diverse array of BAO presentations including dizziness, vertigo, reduced consciousness, quadriplegia, hemiparesis, pupillary abnormalities, and pseudobulbar symptoms [21–23]. These nonspecific and non-localizing symptoms often lead to diagnostic delays and contribute to biases during initial assessments. In addition to having nonspecific symptoms, this patient's youth and known social history likely contributed significantly to the delay in conducting a CTA and MRI, as the primary focus was on investigating potential seizure causes rather than considering stroke as the underlying reason for her symptoms. Given stroke was initially considered lower on the differential diagnosis than other more common causes of ictus in a 28-year-old patient, and as CT head was read without acute intracranial abnormality, there were reasonable delays in this diagnosis. The initial management did not include a D-dimer test or urgent vessel imaging while other etiologies were ruled out, namely toxidrome and seizure. The delay in diagnosing the stroke may have contributed to heightened risk of intravascular intervention, as the stroke had more time to cause diffuse damage and completed infarctions, though interventions on basilar artery occlusions are extremely risky and would still have been considerably deferred by further diagnostic testing such as MRI. While the benefits of an earlier diagnosis remain uncertain, we hope that this case will benefit future patient care teams as they consider basilar artery occlusion rule out in cases without obvious etiology of acute global deficits in the young.

Our intent in presenting this case is to emphasize the necessity of considering basilar artery occlusion as a diagnosis of inclusion in young patients presenting with new acute coma and convulsive activity while emphasizing the

link between hypertensive disorders of pregnancy and major adverse cardiovascular events. Maintaining a high index of suspicion for BAOs is imperative due to the non-specific symptomatology and heightened susceptibility to misdiagnosis. It is crucial to consider further imaging beyond a non-contrast head CT when a patient presents with nonspecific neurologic deficits, even in young patients without specific thromboembolic risk factors. Additionally, extending consideration of pregnancy-associated stroke beyond the immediate postpartum time frame is important to prevent overlooking a critical diagnosis. Addressing such diagnostic challenges early on is critical to preventing morbidity and mortality associated with delayed recognition and treatment of BAOs.

Abbreviations

BAO	Basilar artery occlusion
PCA	Posterior cerebral artery
CTA	CT Angiogram
DOACs	Direct oral anticoagulants
ED	Emergency department
EMS	Emergency medical services
LV	Left ventricular
SCAD	Spontaneous coronary artery dissection

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Authors' contributions
AD completed the electronic medical record review, literature review, and wrote the main case report text. DF edited the report, contributed to writing the text, and obtained images from the electronic medical record. JD edited the report, obtained consent and case report information from family, and obtained images from the electronic medical record. MT edited the report, contributed to the literature review, and helped to obtain images from the electronic medical record. RS edited the report and contributed to designing goals of the case report. All authors read and approved the final case report.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
The patient does not have the capacity to consent for their personal or clinical details and identifying images to be published. However, the patient's power of attorney did provide written informed consent for the patient's personal and clinical details as well as identifying images to be published in this study.

Competing interests
The authors declare no competing interests.

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