CASE REPORT

ONLINE EXCLUSIVE

THE PATIENT
49-year-old woman

SIGNS & SYMPTOMS
– Headache and neck pain radiating to ears and eyes
– Severe hypertension

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THE CASE

A 49-year-old woman was hospitalized with a headache and neck pain that radiated to her ears and eyes in the context of severe hypertension (270/150 mm Hg). Her medical history was significant for heterozygous factor V Leiden mutation, longstanding untreated hypertension, and multiple severe episodes of HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome during pregnancy.

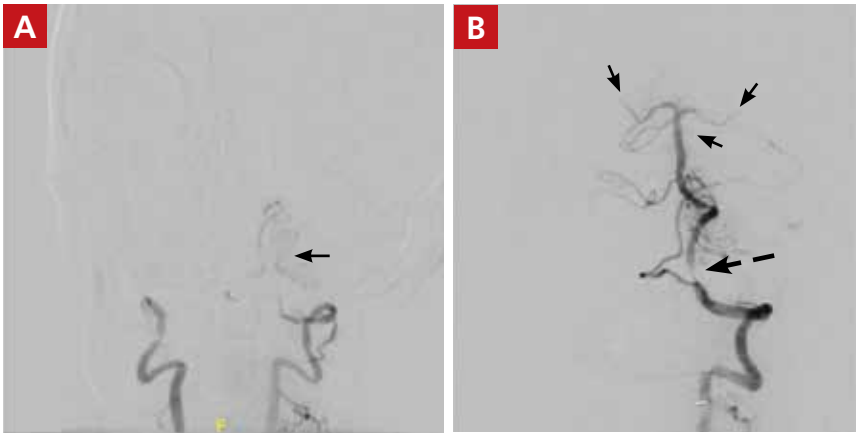
After receiving antihypertensive treatment at a community hospital, her blood pressure gradually improved to 160/100 mm Hg with the addition of a third medication. However, on Day 3 of her stay, her systolic blood pressure rose to more than 200 mm Hg and was accompanied by somnolence, emesis, and paleness. She was transferred to a tertiary care center.

THE DIAGNOSIS

On admission, the patient had left-side hemiparesis and facial droop with dysarthria, resulting in a National Institutes of Health Stroke Scale (NIHSS) score of 7 (out of 42) and a Glasgow Coma Scale (GCS) score of 13 (out of 15). Noncontrast computed tomography (CT) and CT angiography of the head and neck were ordered and showed occlusion of both intracranial vertebral arteries. There were also signs of multifocal infarction in her occipital lobes, thus systemic recombinant human-tissue plasminogen activator (tPA) could not be administered.

The patient was next taken to the angiography suite, where a digital subtraction angiography confirmed the presence of bilateral vertebral artery occlusions (**FIGURE 1A**). A thrombectomy was performed to open the left occluded segment, resulting in recanalization; however, a high-grade stenosis remained in the intracranial left vertebral artery (**FIGURE 1B**). The

FIGURE 1
Cerebral angiography was performed before and after the thrombectomy



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Simultaneous contrast injection of both vertebral arteries before thrombectomy (**A**) showed occlusion of the intracranial vertebral arteries bilaterally with contrast opacification of the left posterior inferior cerebellar artery only (arrow). Postinterventional recanalization of the left vertebral artery (**B**) showed residual high-grade stenosis (dashed arrow). The basilar artery, right superior cerebellar artery, and proximal posterior cerebral arteries were opacified with contrast; however, there were distal embolic occlusions of the posterior cerebral arteries bilaterally and the left superior cerebellar artery (small arrows).

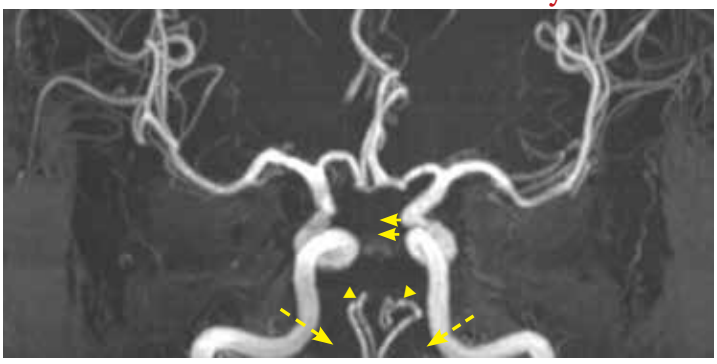
right vertebral artery had a severe extracranial origin stenosis, and balloon angioplasty was performed in order to reach the intracranial circulation; however, the occlusion of the intracranial right vertebral artery segment could not be catheterized. Subsequent magnetic resonance imaging (MRI) with a time-of-flight magnetic resonance angiography showed that the intracranial left vertebral artery with high-grade stenosis had closed down again; thus, there was occlusion of both intracranial vertebral arteries and absent flow signal in the basilar artery (**FIGURE 2**). There were scattered small acute strokes within the cerebellum, brainstem, and occipital lobes.

Unfortunately, within 48 hours, the patient's NIHSS score increased from 7 to 29. She developed tetraplegia, was significantly less responsive (GCS score, 3/15), and required intubation and mechanical ventilation. Reopening the stenosis and keeping it open with a stent would be an aggressive procedure with poor odds for success and would require antithrombotic medications with the associated risk for intracranial hemorrhage in the setting of demarcated strokes. Thus, no further intervention was pursued.

Further standard stroke work-up (echocardiography, extracranial ultrasound of the cerebral circulation, and vasculitis screening) was unremarkable. In the intensive care unit, intravenous therapeutic heparin was initiated because of the potential prothrombotic effect of the factor V Leiden mutation but was subsequently switched to dual anti-aggregation therapy (aspirin 100 mg/d and clopidogrel 75 mg/d) as secondary stroke prevention given the final diagnosis of severe atherosclerosis. Nevertheless, the patient remained tetraplegic with a partial locked-in syndrome when she was discharged, after 2 weeks in the tertiary care center, to a rehabilitation center.

FIGURE 2

Subsequent imaging showed recurrent occlusion in the left vertebral artery



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The occlusion of the intracranial right vertebral artery segment could not be catheterized and the left vertebral artery with high-grade stenosis closed down again. Thus, normal flow-related signal was absent on magnetic resonance angiography in the intracranial vertebral artery bilaterally (dashed arrows) and the basilar artery (solid arrows). Only the distal loops of the posterior inferior cerebellar arteries were visualized (arrowheads).

DISCUSSION

Posterior circulation strokes account for 20% to 25% of all ischemic strokes[1,2] and are associated with infarction within the vertebrobasilar arterial system. Common etiologies of these infarctions include atherosclerosis (as seen in our patient), embolism, small-artery penetrating disease, and arterial dissection.[2] Although the estimated overall mortality of these strokes is low (3.6% to 11%),[2] basilar occlusion syndrome, in particular, is a life-threatening condition with a high mortality rate of 80% to 90%.[3]

Diagnosis can be particularly challenging due to the anatomic variations of posterior arterial circulation, as well as the fluctuating nonfocal or multifocal symptoms.[2] Specific symptoms include vertigo, ataxia, unilateral motor weakness, dysarthria, and oculomotor dysfunction. However, nonspecific symptoms such as headache, nausea, dizziness, hoarseness, falls, and Horner syndrome may be the only presenting signs of a posterior circulation stroke—as was the case with our patient.[2] Her radiating neck pain could have been interpreted as a pointer to vertebral artery dissection within the context of severe hypertension.[4] Unfortunately, the diagnosis was delayed and head imaging was obtained only after her mental status deteriorated.

Immediate neuroimaging is necessary to guide treatment in patients with suspected acute posterior circulation stroke,[1,5,6] although it is not always definitive. While CT is pivotal in stroke work-up and may reliably exclude intracranial hemorrhage, its ability to detect acute posterior circulation ischemic

strokes is limited given its poor visualization of the posterior fossa (as low as 16% sensitivity).[5] Fortunately, CT angiography has a high sensitivity (nearing 100%) for large-vessel occlusion and high predictive values for dissection (65%-100% positive predictive value and 70%-98% negative predictive value).[5,7] Diffusion-weighted MRI (when available in the emergency setting) has the highest sensitivity for detecting acute infarcts, although posterior circulation infarcts still can be missed (19% false-negative rate).[5,8] Thus, correlative vessel imaging with magnetic resonance or CT angiography is very important, along with a high index of suspicion. In some instances, repeat MRI may be necessary to detect small strokes.

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A patient-specific approach to management is key for individuals with suspected posterior circulation stroke.[5] Because specific data for the appropriate management of posterior circulation ischemic stroke are lacking, current American Heart Association/American Stroke Association (AHA/ASA) guidelines apply to anterior and posterior circulation strokes.[6] For eligible patients without multifocal disease, intravenous tPA is the first-line therapy and should be initiated according to guidelines within 4.5 hours of stroke onset[9]; it is important to note that these guidelines are based on studies that focused more on anterior circulation strokes than posterior circulation strokes.[6,9-13] This can be done in combination with endovascular therapy, which consists of mechanical thrombectomy, intra-arterial thrombolysis, or a combination of revascularization techniques.[3,5,6]

Mechanical thrombectomy specifically has high proven recanalization rates for all target vessels.[3-6] The latest AHA/ASA guidelines recommend mechanical thrombectomy be performed within 6 hours of stroke onset.[6] However, there is emerging evidence that suggests this timeframe should be extended—even beyond 24 hours—given the poor prognosis of posterior circulation strokes.[5,6,14] More data on the management of posterior circulation strokes are urgently needed to better understand which therapeutic approach is most efficient.

In patients such as ours, who have evidence of multifocal disease, treatment may be limited to endovascular therapy. Intracranial stenting of symptomatic lesions in particular has been controversial since the publication of the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis trial, which found that aggressive medical management was superior to stenting in patients who recently had a transient ischemic attack or stroke attributed to stenosis.[15] Although additional studies have been performed, there are no definitive data on the topic—and certainly no data in the emergency setting.[16] Further challenges are raised in patients with bilateral disease, as was the case with this patient.

When our patient was admitted to the rehabilitation clinic, she had a GCS score of 10 to 11/15. After 9 months of rehabilitation, she was discharged home with a GCS score of 15/15 and persistent left-side hemiparesis.

THE TAKEAWAY

Posterior circulation stroke is a life-threatening disease that may manifest with a variety of symptoms and be difficult to identify on emergent imaging. Thus, a high degree of clinical suspicion and additional follow-up are paramount to ensure prompt diagnosis and a patient-tailored treatment strategy. JFP

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