

# Acute Bilateral Inferior Cerebellar Infarction in a Patient With Neurosyphilis

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**Background:** Bilateral simultaneous infarction in the territories of the posterior inferior cerebellar arteries (PICAs) is rare but was recently reported with increasing frequency, probably because of the wider availability of magnetic resonance imaging. The cause of these infarcts is believed to be atherosclerotic or embolic occlusion of a dominant PICA, which perfused the territories of the medial branches of both PICAs.

**Results:** We encountered a patient with simultaneous infarction in the territories of the medial branches of both PICAs. The clinical course, imaging results, and laboratory findings are presented. The patient was diagnosed with neurosyphilis based on a history of chancre, positive serum and cerebrospinal serologies, cerebrospinal pleocytosis, and increased intrathecal immunoglobulin

synthesis. We believe that meningovascular syphilis caused the bilateral cerebellar infarct via presumed thromboangiitis of a dominant PICA perfusing both cerebellar hemispheres. The patient was treated with intravenous high doses of penicillin.

**Conclusions:** This case reminds us that meningovascular syphilis should be considered in younger patients with stroke. Patients with bilateral cerebellar infarction may solely have symptoms of vertigo and ataxia but can develop life-threatening complications because of edema of the infarcted tissue with resultant hydrocephalus and pressure on the brainstem.

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**I**SCHEMIC STROKES AFFECTING THE cerebellum and brainstem were the subject of numerous reports in the past century. Even series published in the last 15 years impart the impression that such strokes are almost always unilateral.<sup>1-4</sup> Perhaps because of the recent widespread use of magnetic resonance imaging with diffusion-weighted sequences, several more recent reports have described bilateral simultaneous infarcts of the medial, posterior, and inferior regions of the cerebellum.<sup>5-10</sup> The underlying cause of these bilateral cerebellar strokes was believed to be atherosclerotic cerebrovascular disease or emboli. We now describe a relatively young patient with meningovascular syphilis who was initially seen with a bilateral cerebellar stroke.

## REPORT OF A CASE

A 43-year-old man was initially seen in the emergency department with new onset left occipital headache with vomiting and one episode of "passing out." Examination revealed ataxia. A computed tomographic

scan of the head was unremarkable except for an old left lacunar caudate infarct. Lumbar puncture was performed, and cerebrospinal fluid (CSF) examination results revealed 1 erythrocyte and 12 leukocytes (all mononuclear) per microliter; a glucose level of 68 mg/dL (3.8 mmol/L); and a protein level of 41 mg/dL. The CSF was also sent for a VDRL test. The patient was sent home. He returned 4 days later with worsening occipital headache and left-sided tingling and weakness, and a neurological consultation was sought. The consultant found nystagmus on bilateral gaze, more toward the left; bilateral dysmetria on finger-to-nose and heel-to-shin tests; dysdiadochokinesia; and truncal ataxia so severe that the patient could barely stand even when supported. The mental state was normal, and there were no facial sensory findings, dysarthria, ptosis, or anisocoria. Fundi were normal. Motor examination revealed normal strength and tone, and the stretch reflexes were normal. Babinski signs were not elicited. Sensory examination results were normal to all modalities. General physical examination findings were normal with a blood pressure of 140/85 mm Hg.

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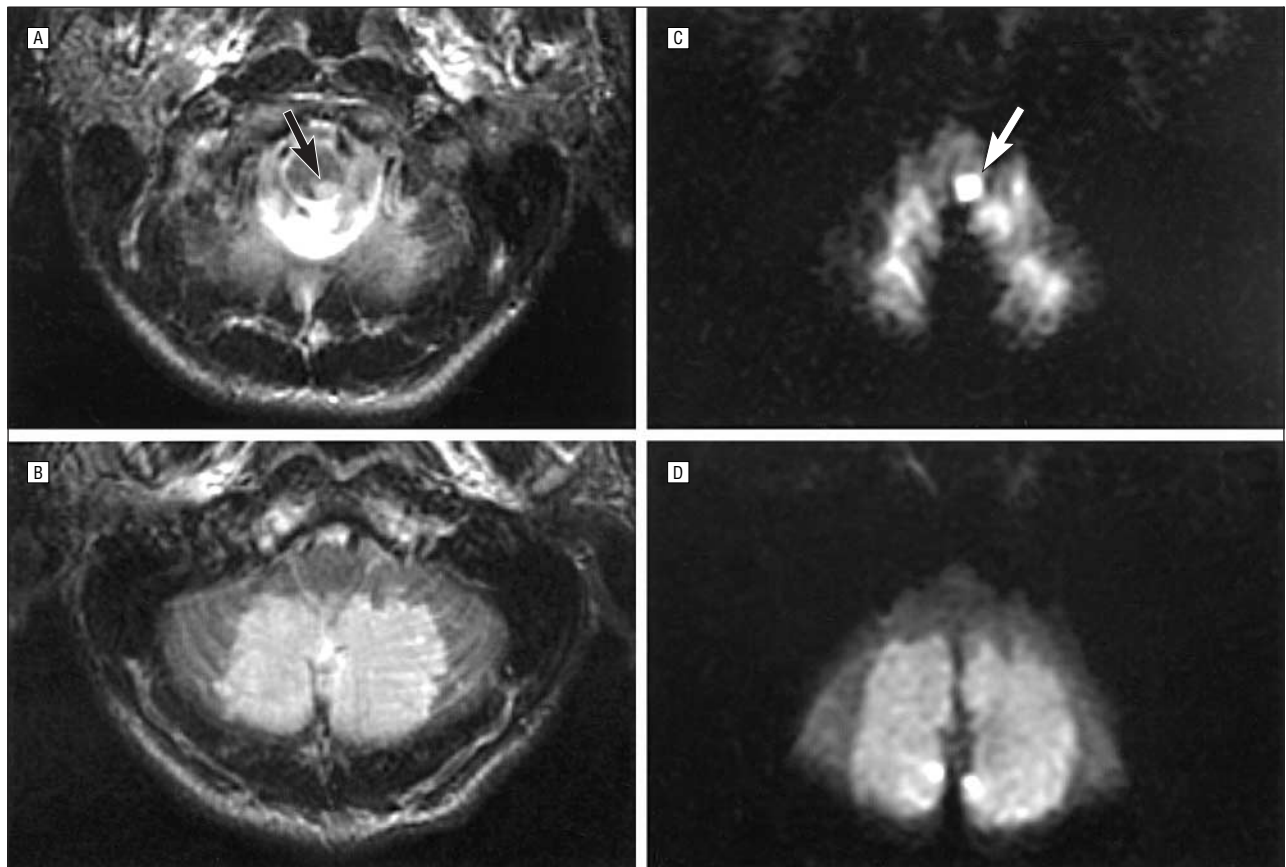


**Figure 1.** Noncontrast axial computed tomographic scan obtained 4 days after the start of symptoms, showing symmetric hypodensity in the medial posterior inferior cerebellar artery territories bilaterally (arrowheads).

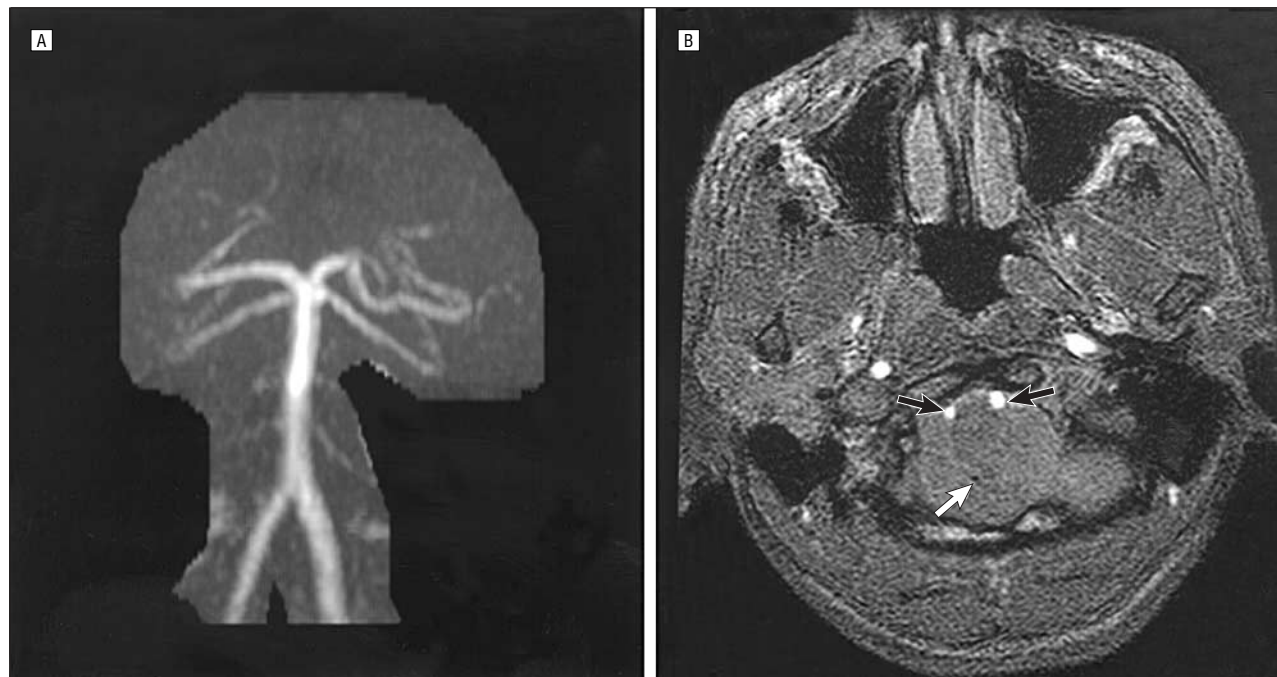
Repeated computed tomographic scans of the head revealed hypodensity in the territories of the medial branches of the posterior inferior cerebellar arteries (PICAs) bilaterally (**Figure 1**). The CSF sample that was sent earlier for a VDRL test was now reported positive. The patient admitted to having a penile chancre 8 years previously for which he received “shots and pills.” The patient was admitted to the hospital with the presumptive diagnosis of meningovascular syphilis and bilateral cerebellar infarction.

In the hospital, magnetic resonance images of the head confirmed the acute infarction in the territories of the medial branches of the PICAs in addition to an acute, small infarct in the left dorsal and caudal medulla (**Figure 2**). Contrast administration showed no enhancement. Results of magnetic resonance angiography of the neck were normal. Magnetic resonance angiography of the head using axial 3-dimensional time-of-flight technique revealed normal carotid systems. The basilar and both vertebral arteries (left dominant) were patent. The posterior cerebral, superior cerebellar, and the anterior inferior cerebellar arteries were patent bilaterally. However, neither the PICAs nor their branches were visualized (**Figure 3A**). On the axial source images, a single vessel devoid of flow-related signal was seen between the right cerebellar tonsil and medulla in the expected location of a PICA branch (**Figure 3B**).

Laboratory studies revealed normal blood counts, metabolic panel findings, and liver function test results,



**Figure 2.** Fluid-attenuated inversion recovery axial- (A and B) and diffusion-weighted (C and D) magnetic resonance images from the same day as the computed tomographic scan (**Figure 1**) confirming acute infarction in the medial posterior inferior cerebellar artery territories bilaterally (B and D). In addition, there was infarction of the left dorsal medulla (arrows).



**Figure 3.** A, Maximal intensity projection of the 3-dimensional time-of-flight magnetic resonance angiogram showing patency of both vertebrals, basilar, both posterior cerebrals, both superior cerebellar, and both anterior inferior cerebellar arteries. B, Axial source image from the 3-dimensional time-of-flight magnetic resonance angiogram showing absence of flow-related signal in a branch of the posterior inferior cerebellar artery located between the medulla and the right cerebellar tonsil (white arrow). Normal flow signals were noted in the vertebral arteries (black arrows).

and the Westergren erythrocyte sedimentation rate was 4 mm/h. The serum fluorescent treponemal antibody absorption test result was positive, but the human immunodeficiency virus serology was negative. Electrocardiography and transthoracic echocardiography results were normal. Repeated CSF examination on hospital admission showed 95 erythrocytes and 51 leukocytes (all mononuclear) per microliter; the glucose level was 55 mg/dL (3.1 mmol/L), and the protein level was 77 mg/dL. The CSF VDRL test result was negative.

The patient was treated with intravenous penicillin, 4 million units every 4 hours for 14 days. In the first few days of the hospital stay, he was lethargic and complained of headache, nausea, and vertigo and had frequent episodes of vomiting. He underwent 2 additional lumbar punctures during his hospital course. The first puncture yielded a negative VDRL test result, but the last yielded a positive CSF VDRL test result (titer 1:2) and an elevated CSF IgG index with oligoclonal bands detected by immunoelectrophoresis. Clinically, he slowly improved but continued to have marked truncal ataxia on hospital discharge to a rehabilitation facility. He was seen thereafter on several occasions as an outpatient and showed continued improvement.

#### COMMENT

The first report of a single patient with bilateral simultaneous cerebellar infarction in the medial PICA territories by Tada et al<sup>5</sup> in 1994 suggested several putative underlying mechanisms. The mechanism that they favored was that both medial branches of the PICAs arose from a single dominant PICA that was occluded either because of arteriosclerotic cerebrovascular disease or from

an embolus.<sup>5</sup> Several similar reports followed. In 1996, Brusa et al<sup>6</sup> described 1 patient with a similar infarct whose only risk factor was long-standing hypertension; in 1997, Sorenson et al<sup>7</sup> described 3 such patients. In 2000, Kang et al<sup>8</sup> described 12 patients with acute bilateral cerebellar PICA infarcts, at least 6 of whom were similar to the case that we now report. These patients were identified among 40 consecutive patients with acute cerebellar infarcts in the PICA territory, which represents a considerably high fraction that may be related to an underlying anatomical variation more frequent in Korea.<sup>8</sup> Another 2 reports, each describing 1 patient with a similar syndrome, were recently published.<sup>9,10</sup> In all of these reports,<sup>5-10</sup> the presumed causes of stroke were cerebrovascular atherosclerotic disease and embolism. In the patient that we describe, the underlying cause was most likely meningovascular syphilis, evidenced by abnormal CSF findings including intermittently positive VDRL test results, pleocytosis, and increased intrathecal immunoglobulin synthesis.

The cause of simultaneous bilateral cerebellar infarct is probably related to an anatomical variant of the PICAs. It is worth emphasizing that of all the major arteries of the human brain, the PICAs have the most varied anatomy.<sup>11</sup> In cases of bilateral simultaneous cerebellar infarcts, the variation is likely either a single PICA perfusing territories ordinarily perfused by both PICAs or a dominant PICA that perfuses the medial territories of both PICAs. We favor the latter hypothesis because of lack of evidence of ischemia in the lateral cerebellar regions in our patient. It should be noted that our patient also had an acute infarct in the left dorsal medulla (Figure 2), a region typically perfused by a branch of the left PICA. However, our patient did not have a Wallen-



berg-type infarct involving the lateral medulla. That, in addition to the left occipital headache that the patient initially had, made us suspect that the main problem was in the left PICA. The findings in our case are most consistent with the type II anatomical variant (a dominant PICA on one side perfusing the inferior and medial PICA territories bilaterally) of PICA as described by Lazorthes.<sup>11</sup> We did not deem it necessary to perform cerebral angiography to further define the vertebrobasilar vasculature in this patient because of the associated risks of this procedure. The patient's "old" lacunar infarct in the head of the left caudate is also consistent with the diagnosis of meningovascular syphilis.

Physicians in general and neurologists in particular should be aware of the possibility of simultaneous bilateral medial cerebellar infarcts. Such infarcts, which may initially cause only vertigo and ataxia, may rapidly evolve because of edema of the infarct, and the resultant pressure on the brainstem and obstruction of CSF flow, both of which can cause an altered mental state that may progress to coma. These patients should be admitted to the hospital for close observation in case they need neurosurgical intervention. The eventual clinical prognosis of these patients is good, and the improvement that we noted in our patient was not different from that observed before.<sup>5-10</sup> Meningovascular syphilis should be included in the differential diagnosis of strokes, particularly when the patient is relatively young and in the absence of risk factors for vascular disease. The fact that only 2 of 4 CSF samples obtained during this patient's hospitalization were VDRL positive serves to remind us that a positive CSF VDRL test is specific but not sensitive for neurosyphilis.<sup>12</sup> Thus, neurosyphilis should be seriously considered in the differential diagnosis of suspected patients even when a CSF VDRL test is nonreactive.

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