Acute Cerebral Infarction Presenting With Weakness in Both Legs and One Arm

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A 48-year-old white man who was healthy previously presented from an outside hospital with weakness of the bilateral lower extremities and right upper extremity. He awoke at 3 AM in morning on the day of initial presentation with unexplained urinary incontinence and diaphoresis. His symptoms resolved, and he went back to bed. At 7 AM, he dropped his daughter off at daycare; this was the last known normal time. At 7:15 AM, he was stopped by the police because of erratic driving. Although details of his condition at that time are sketchy, it seems, he was not following commands. Paramedics were called, and he was taken to a community hospital where he was described as shivering, staring, mute, and not following commands with left gaze deviation and plegia of both lower extremities and the right upper extremity; the left arm moved purposefully with full strength. He had an episode of vomiting and difficulty managing his secretions. Computed tomography scan of the head was unremarkable, and the diagnosis remained uncertain. Because of his shivering, he was loaded with phenytoin for possible seizures and transferred to a tertiary care hospital for further evaluation.

On arrival, he was not following any commands and found to have copious oral secretions that prompted emergent intubation for airway protection. As in the outside hospital, both lower extremities and the right arm were plegic. The left hand moved purposefully. He had extensor posturing of both lower limbs and the right upper limb with noxious stimuli. There was sustained clonus and bilateral plantar extension reflexes present in both lower limbs. Emergent magnetic resonance imaging of the brain showed an acute to subacute infarction in the bilateral frontal lobes, bilateral basal ganglia, and left frontal operculum. Magnetic resonance angiography showed that the right A1 segment of the anterior cerebral artery (ACA) was absent and the right ACA territory was supplied both ACA territories. The ACA supplies the medial and lateral convexities of the frontal and parietal lobes, as well as the anterior limb of internal capsule, inferior head of the caudate, part of inferior putamen, globus pallidus, part of hypothalamus, anterior column of fornix, and corpus callosum are also supplied by the ACA.2 Although each hemisphere typically is supplied by an ipsilateral ACA (Figure 2A), several anatomic variants of the ACA imaging the next day showed a completed left middle cerebral artery and bilateral ACA infarctions (Figure 1B). Because of his large infarct volume, he required hemicraniectomy for malignant cerebral edema. The patient required a tracheostomy and percutaneous gastrostomy tube for feeding. After the first few days, he lost movement in the left upper extremity because of worsening edema with mass effect. An embolic mechanism was suspected although work-up did not reveal an embolic source.

The patient was eventually discharged to a long-term care facility after placement of a tracheostomy tube and a percutaneous gastrostomy tube. At the time of discharge, he had spontaneous eye opening, did not follow commands, and had bilateral upper extremity extension with bilateral lower extremity triple flexion to noxious stimuli.

Discussion

This patient appeared to suffer an embolus within the left carotid that occluded the branches of the left middle cerebral artery and both ACAs. In this case, the right A1 segment of the ACA was absent and the right ACA territory was supplied from the left via the left anterior communicating artery (Figure 1). Bilateral ACA stroke is unusual, but there are some cases in the literature. Bogousslavsky et al1 reported only 2 cases of bilateral ACA stroke of 1490 stroke cases. Borgreeve et al2 reported a case in 1994 in which the patient presented with aphasia, right hemiplegia, plegia of the left leg, and paresis of the left arm. Postmortem analysis revealed bilateral ACA infarction caused by thrombosis of a distal left ACA that supplied both ACA territories.

Anatomy of the ACA and Normal Variants

The ACA supplies the medial and lateral convexities of the frontal and parietal lobes, as well as the anterior limb of internal capsule, inferior head of the caudate, part of inferior putamen, globus pallidus, part of hypothalamus, anterior column of fornix, and corpus callosum are also supplied by the ACA.2

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have been reported. The most common variants include aplastic (Figure 2B) or hypoplastic (Figure 2C and 2D) A1 segments often accompanied by dilatation of the contralateral A1 segment and the anterior communicating artery. Hypoplasia is found in 10% and aplasia in 1% to 2% of postmortem examinations, whereas magnetic resonance angiography demonstrated hypoplasia of the A1 segment in 3% and of the A2 segment in 2% of cases.3 These configurations result in 1 ACA supplying both ACA territories.

The azygous ACA is a rare variant in which both ACA territories are supplied by 1 midline A2 trunk. The prevalence of an azygous ACA is 0.3% to 2% based on postmortem studies and angiography.7 There are 3 types of azygous variants: type I is an unpaired ACA that supplies both ACA territories (Figure 3A), type II is a single bihemispheric ACA that gives branches to both ACA territories and is supplied either entirely or predominantly from 1 Al (Figure 3B), and type III has an additional vessel that originates from the anterior communicating artery along with 2 hypoplastic ACAs that originate from the A2 segments. Additional variants include an accessory or triplicate ACA in which a third vessel originates from the anterior communicating artery (Figure 3C).4 Large postmortem and surgical studies find some degree of ACA fenestration in ≈40% of subjects. Fenestrations are usually small and difficult to visualize in imaging studies.4

**Differential Diagnosis of Bilateral Lower Extremity Weakness**

The differential diagnosis of bilateral lower extremity weakness is broad. The majority of cases are because of a spinal cord lesion, such as demyelinating disease, infarction, or compression caused by a herniated disc, abscess, hematoma, or tumor. Brain-stem stroke can cause bilateral leg weakness, but symptoms are unlikely to be confined to the lower extremities. Importantly, lesions within the frontal cortex that cause mass effect on the contralateral frontal lobe may result in bilateral lower extremity weakness. Although the classic example of this is a meningioma of the falx cerebri that exerts pressure on both frontal lobes, tumors within either hemisphere can have the same effect if there is enough mass effect. Traumatic brain injury affecting both frontal lobes may also present as bilateral leg weakness.

Vascular lesions affecting the bilateral ACA territories can also cause sudden bilateral lower extremity weakness. There are several congenital variations of the ACA vasculature that may cause bilateral frontal lobe ischemia with a single lesion. The absence or hypoplasia of 1 A1 segment results in 1 ACA supplying all or most of both ACA territories (Figure 2B–2D). Occlusion of an azygous ACA or an accessory vessel arising from the anterior communicating artery can result in ischemia of both ACA territories with an embolus via either ACA (Figure 3). Alternatively, vasospasm after subarachnoid hemorrhage can simultaneously affect both ACAs as they lie adjacent to each other. In our patient, bilateral ACA stroke occurred in...
the setting of an absent right A1 segment similar to that illustrated in Figure 2B.

**Causes and Clinical Features of ACA Infarcts**

ACA stroke is rare and accounts for only 3% of all strokes with bilateral ACA strokes being significantly rarer. ACA stroke can be caused by atherosclerosis, thrombosis, artery to artery embolism, or cardioembolism. Bogousslavsky et al reported that 63% of all ACA infarcts are because of either cardioembolism or artery to artery embolism, whereas a Japanese study showed that intracranial atherosclerosis is the most common cause in an Asian population. Unilateral internal carotid artery occlusion with distal extension of thrombosis and cerebral vasculitis are also potential causes of ACA infarction.

Clinical syndromes associated with ACA infarction are varied. Large ACA infarcts cause hemiparesis and hemisensory loss with the leg more involved than the arm, urinary incontinence, mutism, apathy, or euphoria and less commonly a callosal disconnection syndrome. The classical picture of a bilateral ACA infarct includes akinetic mutism, paraplegia, incontinence, and amnesia with apathy. Aphasia has also been described in left sided lesions. It has been postulated that this results from disconnection of Broca area from supplementary motor inputs that may act as a supramotor center for voluntary speech. Motor symptoms are associated with infarction of the precentral area, whereas sensory symptoms are associated with infarcts of the postcentral region. Mutism was noted in more than one third of patients with ACA infarcts and may be because of acute anterior callosal involvement resulting in a disconnection syndrome. Sphincter incontinence, hemineglect, prolonged mutism, and severe apathy, hypokinesia, and abulia were associated with the larger infarcts but had no specific anatomic correlate. Strokes involving bilateral ACA distributions may have additional symptoms referable to the basal ganglia, including Parkinsonism, tremor, and dystonia, as well as primitive reflexes, such as a grasp reflex.

**TAKE-HOME POINTS**

- Normal variants of vascular anatomy can cause unusual presentations of ischemic stroke.
- Bilateral lower extremity weakness can occur with normal variants of the anterior cerebral artery.
- Infarction of the bilateral anterior cerebral artery territories can result in additional symptoms referable to the basal ganglia, including Parkinsonism, tremor, and dystonia.

**Disclosures**

None.

**References**


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