

Neuroimaging Case Review

A Drive Interrupted: Stroke of the Anterior Choroidal Artery – A Case Report

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Abstract

The anterior choroidal artery (AChA) is the most distal branch of the internal carotid artery (ICA). The AChA is significant because it supplies important structures in the brain, including the optic tract, anterior portion of cerebral peduncle, lateral geniculate body, uncus, globus pallidus, posterior and superficial areas of the thalamus, and the retrolenticular and posterior portions of the internal capsule on the same side as the artery. Isolated strokes involving the AChA are rare and can result in HHH Syndrome, consisting of contralateral hemiplegia, hemisensory loss, and homonymous hemianopia. Features which distinguish an AChA infarction from larger arterial pathology are lack of headache and lack of depressed level of consciousness in subacute infarction, and usually lack of aphasia acutely. The etiology remains controversial, with proposed mechanisms including cardioembolic, large-vessel atherosclerosis, dissection of the ICA, small-vessel occlusion, and cryptogenic causes. Herein, the authors report a case of an isolated AChA infarction resulting in a right-sided, pure motor hemiparesis with no sensory or vision loss, highly suggestive of cardioembolic origin, with the evaluation of the patient, and eventual treatment strategy.

Key words: Anterior Choroidal Artery, Rare Stroke Pathologies, Case Report, Cardioembolic, Small Vessel Disease, HHH Syndrome.

INTRODUCTION

The anterior choroidal artery (AChA) is the most distal branch of the internal carotid artery (ICA) after it enters the skull base. It arises most commonly after the posterior communicating artery.¹ The AChA is significant because it supplies important structures in the brain, including the optic tract, anterior portion of cerebral peduncle, lateral geniculate body, uncus, globus pallidus, posterior and superficial areas of the thalamus, and the retrolenticular and posterior portions of the internal capsule on

the same side as the artery.¹ Isolated strokes involving the AChA are rare and can result in AChA syndrome, which classically presents as a triad of contralateral hemiplegia, hemisensory loss, and homonymous hemianopia commonly referred to as HHH Syndrome.² Incomplete forms of the AChA syndrome are common, with motor deficits being the most frequently seen clinical symptom followed by hemisensory loss.^{3,4} Features that can distinguish an AChA infarction from a larger arterial pathology are lack of depressed level of consciousness and



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lack of headache with infarct evolution, and typically absence of aphasia in patients with right-sided motor and sensory deficits.⁵ The etiology of AChA infarcts remains controversial, with proposed mechanisms that include cardioembolism, large-vessel atherosclerosis, dissection of the ICA, small-vessel occlusion, and cryptogenic causes.^{4,6} We report a case of an isolated AChA infarction resulting in a right-sided, pure motor hemiparesis with no sensory or vision loss, highly suggestive of cardioembolic origin.

THE CASE

A 56-year-old, right-handed man with a history of untreated hypertension presented to the emergency department (ED) with right-sided face, arm, and leg weakness. He had been driving an hour before arrival and noted that he was becoming weak. He finished his drive home and called for the paramedics who appropriately brought him to a comprehensive stroke center. His National Institutes of Health Stroke Scale (NIHSS) score was 7 (item 4 = 2 points [right lower facial weakness]; item 5 = 2 points [right arm motor weakness]; item 6 = 2 points [right leg motor weakness]; item 10 = 1 point [dysarthria]). He was afebrile with stable vital signs. Neurologically, he was alert and oriented, with a Glasgow Coma Scale score of 15. He had no apparent visual field deficit or sensory disturbance. He denied any prior seizure, stroke, recent trauma, infection, and tobacco use. Initial computed tomography (CT) scan of the head without contrast was negative for subacute infarction or hemorrhage, CT angiography was negative for large vessel occlusion, and CT perfusion

was negative for any perfusion deficit. Following a discussion of the risks and benefits, the decision was made with the patient and family to treat with tenecteplase (TNK) which was administered without complications.

The following day, fluid attenuated inversion recovery magnetic resonance imaging of the head showed a left anterior choroidal stroke which affected the insula and some structures of the basal ganglia, including the posterior lentiform nucleus extending to the caudate body (Figure 1). Mild chronic small vessel ischemic disease was also noted. Because of the vessel affected, the team discussed potential causes of the large vessel disease, and the patient underwent cardiac workup to determine the etiology of the stroke. Transesophageal echocardiogram (TEE) revealed a small atrial septal aneurysm (ASA) and a small patent foramen ovale (PFO) with late appearing bubbles and no evidence of intracardiac masses or thrombi. Discussion with the cardiology team revealed that based on the small size of the shunt, they did not believe that it was large enough to warrant closing. Discussion was had with our outpatient ultrasound technologist to reevaluate the small shunt using transcranial Doppler in the outpatient clinic. Cardiac monitoring during his inpatient stay did not reveal evidence of paroxysmal atrial fibrillation (AF). An implantable loop recorder was placed to monitor for possible paroxysmal AF.

During the patient's stay, evaluation for secondary causes of stroke were identified and treated. His urine drug screen was



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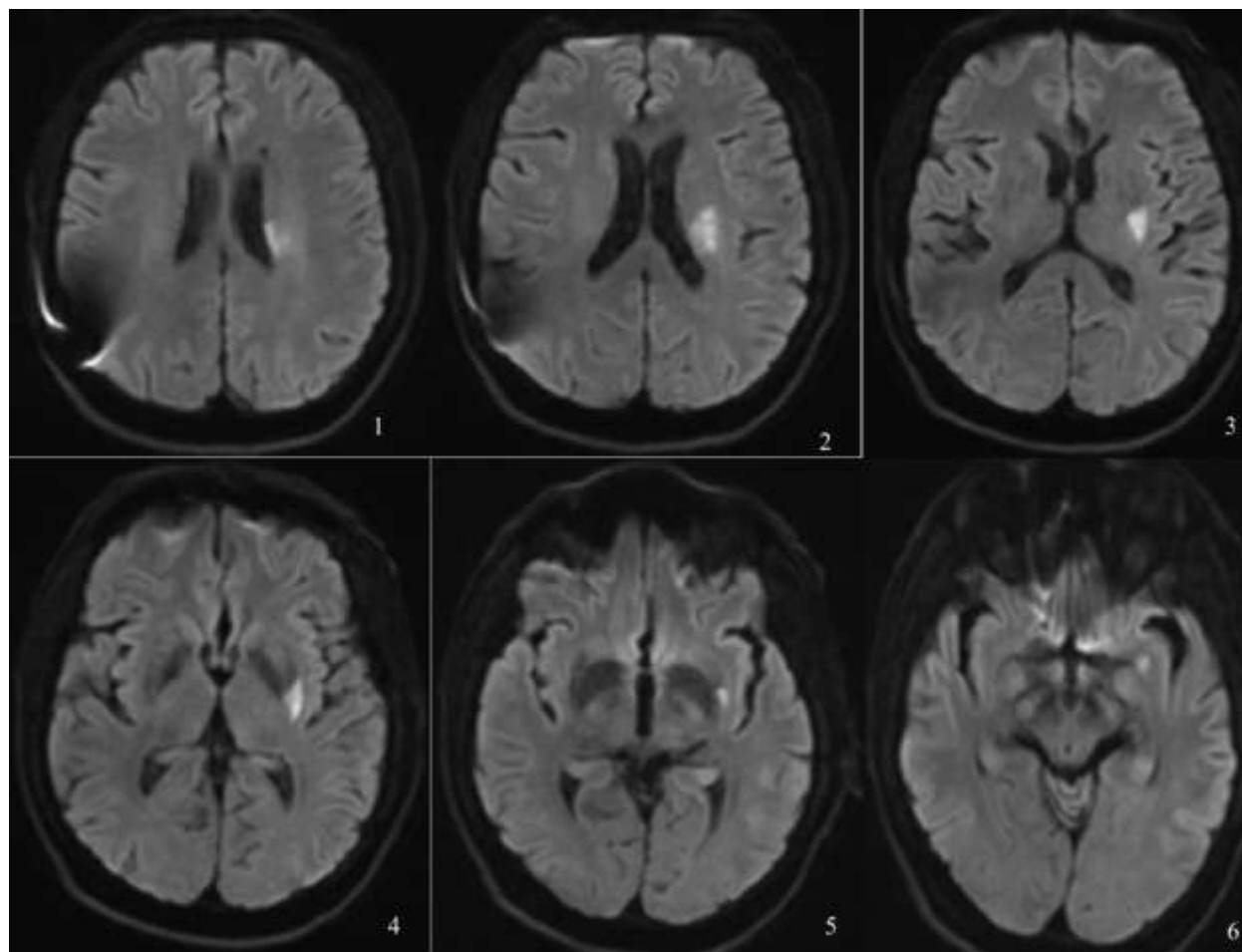


Figure 1: T2 FLAIR images presented from superior (slice 1) to inferior (slice 6) with signal located in the classically described tract of the anterior choroidal artery; note some patchy sparing of the most distal portions of the arterial tract likely from the administration of Tenecteplase.

negative for any illicit substances. The patient initially presented with a hypertensive emergency requiring nicardipine and intensive care unit admission, and required continued treatment for hypertension throughout his hospital stay with losartan 100 mg, carvedilol 6.25 mg, and hydrochlorothiazide 25 mg. His hemoglobin-A1c was 5.6% and low-density lipoprotein was 140 mg/dL. He was placed on atorvastatin 40 mg and aspirin 81 mg daily for secondary stroke prevention. The patient was discharged after 4 days with no further

complications, except continued right-sided face, arm, and leg weakness exhibiting no change or improvement. He was referred for occupational and physical therapy.

DISCUSSION

The origin of isolated AChA infarcts has been a subject of debate in the literature for many years. The area supplied by the AChA is frequently involved in larger infarcts of the internal carotid artery, further complicating research on the topic. Earlier studies suggested that isolated AChA infarcts were



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most commonly caused by lipohyalinosis of the small and deep branches of the AChA, or small vessel disease (SVD).⁶ More recent research has indicated that this is not always true, with many cases of AChA strokes involving cardioembolic and large vessel atherosclerosis etiology.⁴ This suggests that cardiac workup may be needed to determine the cause of the stroke in order to manage and treat patients appropriately.

The patient arrived to the ED with pure motor hemiparesis, which are most classically associated with lacunar infarcts of the internal capsule, corona radiata, or the corticospinal tracts in the brain stem (most commonly in the pons). Lacunar infarcts generally develop in patients with hypertension, diabetes, hyperlipidemia, or a mixture of all three.^{7,8} Our patient's history of hypertension fit the clinical description for a classic lacunar infarct, suggesting that a cardioembolic source was unlikely. Upon further imaging, it was noted that the patient's infarct was in the anterior choroidal artery territory, a vessel which at one time was generally thought to be damaged secondarily to SVD. However, based on the newest findings in the literature, it was appropriate to continue evaluation for cardiac causes of the stroke.⁴ At hospital discharge, both imaging and advanced work up failed to provide a clear cause of the patient's stroke.

Nevertheless, the patient received a cardiac work up with associated TEE study, revealing an ASA and small PFO. Both ASA and PFO have been associated with a higher risk of cardiac arrhythmias, including AF, and cryptogenic stroke;⁹⁻¹¹ although not observed during cardiac monitoring at the hospital, it is possible the patient has paroxysmal AF aggravated by the ASA and PFO. The patient will receive transcranial Doppler bubble testing outpatient to further assess the functionality of the right-to-left shunt.

CONCLUSIONS

This case report highlights the importance of investigating the etiology of isolated AChA strokes, as they may not all be caused by SVD. Cardiac workup may in some cases reveal a cardioembolic source, which would drastically change the management and treatment of these patients. In the evaluation of patients with rare pathologies, it is appropriate to widen the differential and perform further testing to prevent future strokes. This is especially important in young patients, recognizing that in such cases rare etiologies may not in and of themselves be rare, but instead undiagnosed.¹² It is the hope of these authors that this case helps to improve clinical gestalt in the determination of pathogenic mechanism for these patients.

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