

Variant carotid origin of left anterior inferior cerebellar artery mimicking infarct on angiography

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ABSTRACT

A case is presented of a rare anterior inferior cerebellar artery variant, originating from the internal carotid artery. This mimicked a cerebellar infarct on vertebral angiography of a patient who presented with dysmetria and gait imbalance. It is important for the neuroradiologist and neurointerventionist to be aware of common and unusual cerebellar arterial anatomic variants to avoid incorrect diagnoses, as well as understand possible implications for surgery and interventional therapies.

INTRODUCTION

The anterior inferior cerebellar artery (AICA) can vary in caliber, have variable site of origin from the basilar or distal vertebral arteries and may be asymmetric within a given patient.¹ Carotid origin of the AICA is a rare variant^{2–6} and has been attributed to persistent primitive arteries. The primitive cerebral circulation consists of paired internal carotid and longitudinal neural arteries, with the trigeminal, otic, hypoglossal and proatlantal arteries serving as connections between the anterior and posterior circulation. The primitive trigeminal artery, which approximates the origins of AICA and the posterior inferior cerebellar artery (PICA), usually involutes by 14 weeks' gestation as the longitudinal neural artery configuration changes to form the vertebrobasilar system.⁷ While these embryological remnants are usually incidental findings, they can present diagnostic and therapeutic dilemma to those who are not aware of their existence.

CASE REPORT

A 49 year old woman presented to the emergency room with a 6 h history of right arm and hand dysmetria as well as gait imbalance. Two months prior to her admission she was diagnosed with distal right vertebral artery dissection and pseudoaneurysm for which she underwent sacrifice of the distal right intracranial vertebral artery by coiling. Additionally, a 6 mm distal left vertebral dissection was noted. For the past 6 days the patient had abstained from any antiplatelet medications.

Angiography was performed to evaluate the coiled aneurysm and for possible thromboembolic etiology of current symptoms. Right vertebral angiography confirmed that the cervical portion of the right vertebral artery terminated in occipital branches and no antegrade flow approached or filled the coiled aneurysm (figure 1). Angiographic views of the left vertebral artery showed the distal left

vertebral artery dissection, now measuring 8 mm diameter. Additionally, there was absence of perfusion of the inferior left cerebellar hemisphere (figure 2). The left AICA was not identified; no abrupt cut-off was evident to suggest embolus as etiology. Normal caliber left PICA and left superior cerebellar arteries were identified. The left carotid artery was then selected, and angiographic run revealed a variant origin of the left AICA from the left internal carotid artery (figure 3).

Brain MR imaging was obtained after angiography and confirmed there were no areas of restricted diffusion. The patient had complete resolution of symptoms within 10 h of onset.

DISCUSSION

The AICA usually originate from the lateral aspects of the proximal basilar artery, just distal to the vertebrobasilar junction. They course lateral to the pontomedullary junction, providing branches to supply the lower pons and the medulla. AICA then extends into the cerebellopontine cistern to supply the labyrinthine/internal auditory branch which travels with the cranial nerve VIII into the internal acoustic meatus. AICA then branches; one branch extends inferiorly, to supply the anterior, inferior portion of the cerebellum and the restiform body in the inferior cerebellar peduncle, often anastomosing with PICA branches. The other branch extends laterally, looping around the acousticofacial bundle to anastomose with both superior cerebellar and PICA branches to supply the middle cerebral peduncle and adjacent pons.⁸

Accordingly, sequelae of AICA infarcts have included dysmetria, ataxia, ipsilateral facial palsy, ipsilateral trigeminal sensory loss, and contralateral loss of pain and temperature, corresponding to the anterolateral cerebellum and cranial nerve nuclei in the mid-lower pons. Vertigo and hearing loss can occur if the labyrinthine/internal auditory branch is involved.⁹ Clinically, AICA 'syndrome' can be difficult to differentiate from Wallenberg's syndrome (lateral medullary/PICA infarct).

When Scotti² described an AICA originating from the cavernous internal carotid artery in 1975, other anomalous cerebellar arteries with carotid supply had been reported but none supplied the AICA territory. A similar vessel was identified by Nutik and colleagues³ in a patient with an unusual carotid–anterior cerebral arterial anastomosis and anterior communicating artery aneurysm. Vertebral artery angiograms in both patients confirmed that the anomalous arteries had no supply from the posterior circulation. Ito and colleagues⁴ described two separate cases of unilateral AICA, arising from the cavernous and pre-cavernous internal carotid arteries.



Figure 1 Lateral angiographic view confirms that the cervical right vertebral artery terminates in occipital branches (arrows). No antegrade flow extends to the coiled distal right vertebral pseudoaneurysm (arrowheads).

Each case showed the aberrant artery extending to the biventral lobule or the great horizontal fissure, areas normally supplied by the AICA.

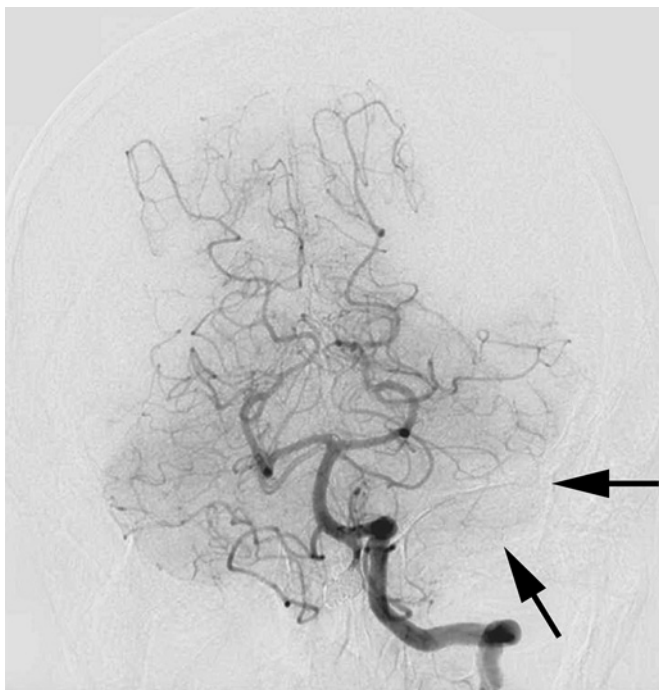


Figure 2 Frontal view of left vertebral angiogram shows absence of the left anterior inferior cerebellar artery and missing blush in the inferior left cerebellar hemisphere (arrows). Also noted is an 8 mm distal left vertebral dissection. Normal left posterior inferior cerebellar artery and superior cerebellar arteries are present.

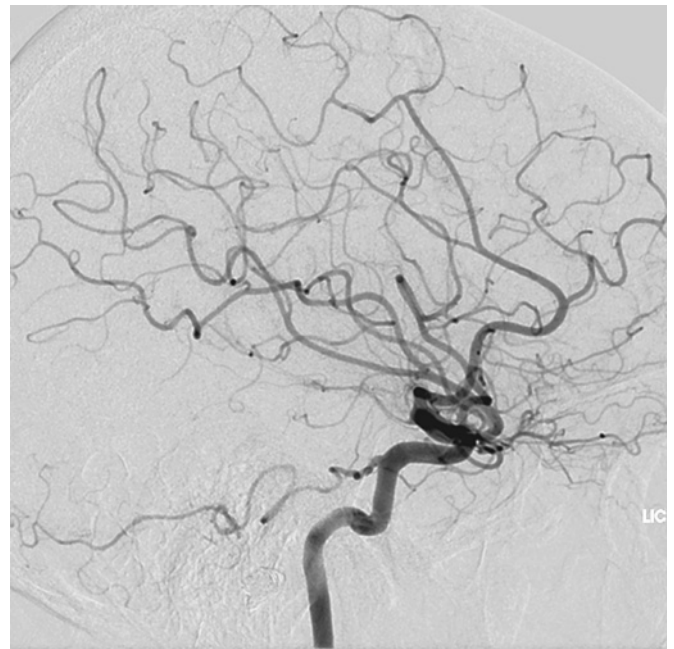


Figure 3 Lateral view of left carotid angiogram reveals anomalous origin of the left anterior inferior cerebellar artery from the cavernous left internal carotid artery.

Haughton and colleagues⁵ reported four incidental anomalous cerebellar arteries with cavernous internal carotid artery origins. They postulated that the variants were sequelae of persistent primitive trigeminal arteries, given the location and tortuosity of the most proximal portion. Vertebral angiography was not included in these cases, to evaluate any residual connection to the basilar artery. In the series of anomalous cerebellar arteries (three superior cerebellar, three AICA and three PICA) described by Siqueira and colleagues⁶, another variant supportive of the persistent trigeminal hypothesis was described. In one case, an artery was seen extending from the internal carotid to join the AICA, which also demonstrated retrograde filling of the basilar artery.

Anomalous carotid origin of the AICA has been an incidental finding in all described patients. In theory, these patients could present with posterior circulation ischemia due to carotid atherosclerosis or unusual thromboembolic distribution. As in this case, it presented a diagnostic dilemma on the vertebral angiogram as it resulted in a mimic of a left AICA infarct. Only on carotid injection was the true anatomy defined.

This anatomic variant does have implications for neuro-interventionalists and surgeons. In patients who undergo WADA testing, it is crucial to exclude any communications between the anterior and posterior circulation before injecting amytal, due to the risk of respiratory suppression. Neurointerventionalists need to be aware of an anomalous AICA before performing percutaneous gasserian ganglion blocks and surgeons should be informed of an anomalous AICA before any planned approach to the posterior fossa or cerebellopontine angle.

Competing interests None.

Patient consent Obtained.

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