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Case Report

Persistence of the hypoglossal artery: A rare case report *,**

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ABSTRACT

The persistence of the hypoglossal artery is a rare embryonic vascular variant in which a transient connection between the carotid and vertebrobasilar systems fails to regress. Typically asymptomatic and incidentally discovered on imaging, this condition can alter cerebral blood flow and increase the risk of vascular complications such as aneurysms, strokes, and arteriovenous malformations. We present the case of a 70-year-old female with no prior medical history who was admitted to the emergency department with right-sided hemiparesis. A cerebral CT angiography, performed to evaluate for acute ischemic stroke and potential thrombolysis, revealed a persistent hypoglossal artery arising from the internal carotid artery at the C1-C2 level and connecting to the basilar artery through the hypoglossal canal. This case highlights the importance of recognizing this rare anatomical variant on imaging to prevent misinterpretation and avoid complications during surgical or endovascular interventions at the skull base.

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Introduction

The persistent hypoglossal artery is one of the persistent carotid-vertebrobasilar anastomoses that would normally regress during embryonic development. It originates from the posterior aspect of the cervical internal carotid artery, typically between the vertebral levels C1 and C3. It follows the path of the hypoglossal nerve, passing through an enlarged

hypoglossal canal, and ultimately joins the basilar artery from below [1].

The PHA is the second most common type of persistent carotid-basilar anastomosis, following the persistent trigeminal artery. It is a rare anomaly, with an estimated prevalence of 0,02%-0,26% [1]. However, its recognition on imaging is important due to its potential to cause neurovascular complications [2].

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Case report

We report the case of a 70-year-old female with no known medical history who presented to the emergency department with sudden-onset left-sided hemiparesis evolving over 2 hours. She denied any history of trauma, headache, visual disturbances, seizures, or previous similar episodes. There was no family history of stroke, aneurysm, or other cerebrovascular diseases. The patient was not on any chronic medication and had no history of prior surgeries. She was active and independent, with no apparent cardiovascular risk factors.

On arrival, the patient was hemodynamically stable, with a blood pressure of 120/60 mmHg, heart rate of 88 bpm, respiratory rate of 18 breaths per minute, oxygen saturation of 98% on room air, and body temperature of 36.8°C. Neurological examination revealed motor weakness graded 3/5 in the left upper and lower limbs, while strength was preserved (5/5) on the right side. Superficial and deep sensitivity was intact, reflexes were symmetrical, and there was no facial asymmetry, speech impairment, or cerebellar dysfunction. Cardiovascular, pulmonary, and abdominal examinations were unremarkable.

Given the suspicion of an acute ischemic stroke, blood samples were drawn immediately to assess eligibility for thrombolysis, and a cerebral CT angiography was performed without delay. The noncontrast brain CT was unremarkable, ruling out hemorrhage. After contrast injection, imaging revealed a large vessel originating from the internal carotid artery at the C1-C2 level, connecting to the basilar artery through the hypoglossal canal (Fig. 2). 3D reconstructions (MIP and VR) facilitated visualization of the unusual anatomical course of this artery (Fig. 3). The left vertebral artery was found to be hypoplastic (Fig. 4), with no evidence of arterial occlusion. The findings were consistent with an anatomical variant, specifically a carotid-vertebrobasilar anastomosis of the persistent hypoglossal artery type.

While awaiting laboratory results, the patient remained clinically stable, and her symptoms began improving spontaneously. The blood tests returned within normal limits, with hemoglobin at 13.4 g/dL (reference: 12-16 g/dL), white blood cell count at 7,500/mm³ (reference: 4,000-11,000/mm³), platelet count at 235,000/mm³ (reference: 150,000-450,000/mm³), INR at 1.0 (reference: <1.2 for nonanticoagulated patients), and an aPTT of 30 seconds (reference: 25-35 seconds). Blood glucose was 98 mg/dL (reference: 70-140 mg/dL), and kidney function was normal, with a serum creatinine of 0.8 mg/dL (reference: 0.6-1.2 mg/dL). Electrolytes were within normal ranges, and troponin and ECG were unremarkable, ruling out a concurrent cardiac event.

As there was no evidence of large vessel occlusion, no established ischemic infarct, and the patient's neurological function was rapidly improving, intravenous thrombolysis was not administered. She was admitted to the stroke unit for neurological monitoring and was started on aspirin 100 mg daily, atorvastatin 40 mg daily, and blood pressure management as preventive measures. A carotid Doppler ultrasound was performed, showing no significant atherosclerosis.

The patient recovered fully within 1 hour, and the diagnosis of a transient ischemic attack (TIA) was confirmed. She was discharged on secondary stroke prevention ther-

apy and scheduled for follow-up in neurology and vascular medicine.

This case highlights the importance of recognizing persistent hypoglossal artery as a rare but clinically relevant vascular anomaly. While often asymptomatic, this variant can alter cerebral hemodynamics and predispose patients to vascular complications such as aneurysms and ischemic events. Identifying it on imaging is crucial to prevent misdiagnosis and avoid complications during surgical or endovascular interventions at the skull base.

Discussion

During early embryonic development, posterior cerebral vascularization relies on the neural longitudinal arteries, which are the precursors of the vertebral arteries and the basilar artery. These primordial vessels emerge very early and run along the developing brainstem. At this stage, the vertebral arteries have not yet formed, and the posterior brain depends on anastomoses between the anterior carotid system and these neural longitudinal arteries. These include several important transient arteries, such as the embryonic trigeminal artery, the embryonic otic artery, the embryonic hypoglossal artery, and the embryonic proatlantal artery [3].

Each of these anastomoses temporarily allows the internal carotid artery to supply the posterior cerebral structures, including the brainstem, until the vertebral and basilar arteries are fully developed. These connections persist during the early weeks of embryonic development, typically up to the sixth week of gestation, when the vertebral arteries form from the cervical intersegmental arteries. The right and left vertebral arteries then ascend along the cervical spine to merge at the base of the brain, forming the basilar artery [3].

At this point, the posterior circulation becomes fully established, and the embryonic anastomoses progressively regress and disappear [3].

If this regression does not occur correctly, these primitive anastomoses persist, forming persistent carotid-vertebrobasilar anastomoses. This means that the vertebrobasilar system remains partially or entirely dependent on blood flow from the internal carotid artery through these anastomoses. These abnormal connections give rise to vascular variants, [4].(Fig. 1):

- √ Persistent trigeminal artery: This is the most common variant, with a prevalence of 0.1%-0.2%. It connects the ICA at the cavernous level to the basilar artery.
- √ Persistent hypoglossal artery: With a prevalence of 0.03%0.09%, it emerges from the cervical ICA, usually at the C1C2 level, and traverses the hypoglossal canal to join the
 basilar artery.
- √ Persistent otic artery: It connects the ICA to the basilar artery via the path of the vestibulocochlear nerve.
- √ Persistent proatlantal intersegmental artery: This variant links the ICA or external carotid artery (ECA) to the vertebral artery. It has 2 subtypes:

Type I: Originates from the ICA and connects to the vertebral artery at the C1-C3 levels.

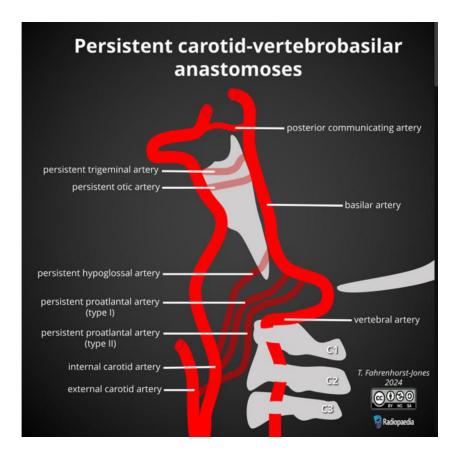


Fig. 1 – Diagrams of persistent carotid-vertebrobasilar anastomoses from cephalic to caudal; persistent trigeminal artery, persistent otic artery, persistent hypoglossal artery, and persistent proatlantal artery (type I and II). (Radiopaedia.org) [9].



Fig. 2 – Brain CT angiography ([A] Axial view, [B] Coronal view) showing the internal carotid artery (green arrow) giving rise to a large vessel passing through the hypoglossal canal at the C1-C2 level, identified as the "persistent hypoglossal artery" (red arrow), which joins the basilar artery (blue arrow).

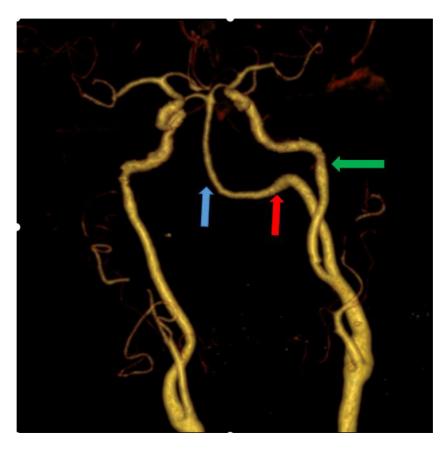


Fig. 3 – Brain CT angiography, a 3D volume-rendering technique (VRT) showing the internal carotid artery (green arrow) giving rise to a large vessel passing through the hypoglossal canal at the C1-C2 level, identified as the "persistent hypoglossal artery" (red arrow), which joins the basilar artery (blue arrow).



Fig. 4 – Brain CT angiography (Coronal view) showing hypoplasia of the left vertebral artery (blue arrow).

Type II: Originates from the ECA and joins the vertebral artery at the same levels.

The precise mechanisms behind this failure of regression are not yet fully understood. However, several hypotheses exist, including genetic mutations affecting vascular development, which may prevent the regression of primitive anastomoses, as well as environmental and epigenetic factors.

The clinical presentation of a persistent hypoglossal artery depends on the presence or absence of associated complications. On its own, it is often asymptomatic and is discovered incidentally during imaging studies performed for other reasons, such as CT angiography or MR angiography. However, when symptoms appear, they are generally due to complications such as ischemic strokes, which may present with symptoms such as vertigo, ataxia, diplopia, and dysarthria, or intracranial aneurysms, which are often asymptomatic but can sometimes present with headaches, neck stiffness, or loss of consciousness [5].

In imaging, the most useful techniques for detecting PHA are magnetic resonance angiography or computed tomography angiography. These modalities provide detailed visualization of vascular structures, enabling the identification of this rare anatomical variant. PHA is characterized by the presence of a large vessel originating from the posterior aspect of the internal carotid artery, typically at the cervical level (C1-C2).

It follows a specific path along the hypoglossal nerve (cranial nerve XII), then enters the hypoglossal canal, running posteromedially toward the base of the skull to join the basilar artery just above it. The hypoglossal canal may appear enlarged due to the passage of this artery [6].

It is common to observe hypoplasia or even absence of the vertebral artery, as well as the posterior communicating artery. This results in a redirection of blood flow to the posterior circulation, which is maintained by the persistent hypoglossal artery originating from the ICA [6].

The persistence of the hypoglossal artery can lead to significant complications, including increased risks during neurosurgical procedures, intracranial aneurysms, particularly at the junction between the hypoglossal artery and the basilar artery, and strokes. If a pathology such as atherosclerosis, dissection, or embolism affects the internal carotid artery, the blood flow in the vertebrobasilar system may be compromised, increasing the risk of ischemia in the posterior brain. In rare cases, it may also cause compression of the hypoglossal nerve. Early recognition of this variant through imaging is crucial to prevent these complications and to adapt therapeutic management accordingly [7].

The treatment of a persistent hypoglossal artery primarily depends on the symptoms, associated complications, and the risk of surgical complications. In the absence of complications, a persistent hypoglossal artery is usually asymptomatic and does not require routine monitoring. However, if associated with an aneurysm or other vascular abnormalities, periodic imaging surveillance may be warranted [8].

Conclusion

The persistence of the hypoglossal artery is a rare but clinically significant vascular anomaly, particularly due to its potential neurovascular complications, such as intracranial aneurysms and strokes. Early recognition through imaging is essential to prevent these complications and to tailor therapeutic approaches, especially during neurosurgical procedures. Treatment varies depending on the presence or absence of symptoms, ranging from regular monitoring to endovascular interventions in cases of complications.

Patient consent

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

Ethical approval

No ethical approval is required for de-identified single case reports based on our institutional policies.

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