

Persistent Hypoglossal Artery Dissection: A Rare Cause of Hypoglossal Nerve Palsy

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ABSTRACT

Background: Persistent primitive hypoglossal artery (PPHA) is a rare carotid–basilar anastomosis that may persist into adulthood and become clinically relevant when associated with vascular pathology or cranial neuropathy.

Methods: A middle-aged woman (51 years) presented with right hemilingual edema, dysphagia, and sialorrhea. Imaging demonstrated a persistent hypoglossal artery complicated by arterial dissection within the hypoglossal canal, along with secondary signs of hypoglossal nerve palsy. The patient was managed conservatively with antihypertensive therapy.

Results: The imaging findings correlated with the patient’s clinical symptoms, revealing that the PPHA was the site of both vascular injury and cranial neuropathy.

Conclusions: This case underscores that PPHA, although often incidental, can give rise to significant complications such as arterial dissection and hypoglossal nerve palsy. Recognition of this vascular variant is essential to avoid misdiagnosis and to guide safe management of cervico-cranial vascular disease.

Keywords: Persistent hypoglossal artery; Arterial dissection; Hypoglossal nerve palsy; Vascular anomaly; Case report.

Abbreviations: PPHA: Persistent Primitive Hypoglossal Artery; ICA: Internal Carotid Artery; CT: Computed Tomography; MRI: Magnetic Resonance Imaging; MRA: Magnetic Resonance Angiography; 3D TOF: Three-Dimensional Time-Of-Flight; 3D FSPGR: Three-Dimensional Fast Spoiled Gradient-Echo

INTRODUCTION

The persistent primitive hypoglossal artery (PPHA) represents a rare embryonic carotid–basilar anastomosis that typically regresses by the fifth week of gestation. It is the second most common persistent carotid–vertebrobasilar anastomosis after the trigeminal artery, with a reported incidence of 0.02–0.26% [1]. PPHA occurs more frequently in women, shows a predilection for the left side, and is bilateral in approximately 1.4% of cases [2].

Anatomically, the PPHA arises from the internal carotid artery (ICA) at the upper cervical segment (C1–C3), courses through the hypoglossal canal, and joins the basilar artery. The vertebral and posterior communicating arteries are often hypoplastic, making the PPHA the principal supply to the posterior circulation. First described

anatomically in 1889 by Batujeff [3] and angiographically in 1961 by Begg [4], the PPHA remains the persistent carotid–vertebrobasilar anastomosis most frequently associated with pathology and significant clinical implications. Its recognition is therefore crucial in patients presenting with atypical neurological symptoms and when planning vascular interventions.

PPHA has also been linked to aneurysm formation, glossopharyngeal neuralgia, and hypoglossal nerve palsy, and it may play a critical role in posterior circulation, particularly when the vertebral arteries are hypoplastic. In such cases, carotid procedures such as endarterectomy or stenting carry a considerable risk of ischemia, and emboli originating from the anterior circulation may lead to both anterior and posterior infarctions [5].

While aneurysmal complications are well documented, reports of PPHA associated with arterial dissection are rare. To date, the literature describes only cases of ICA dissection in the presence of a PPHA, without direct involvement of the anomalous artery itself [6]. To our knowledge, no previous report has documented a dissection of the PPHA, underscoring the importance of recognizing this vascular anomaly in patients with atypical neurological symptoms and when planning cervico-cranial vascular interventions.

CASE PRESENTATION

A 51-year-old woman with a history of arterial hypertension presented with right-sided hemilingual edema, dysphagia, and sialorrhea. Clinical examination excluded lesions of the tongue body. Computed tomography (CT) was first performed as part of the diagnostic work-up, using contrast-enhanced phase acquisition with multiplanar reconstructions and volume-rendered images, demonstrating an anomalous vascular structure arising from the ICA and directed toward the hypoglossal canal, consistent with a PPHA (Figure 1a). A dissection flap was identified within the ICA extending into the PPHA, whose false lumen appeared thrombosed within the hypoglossal canal (Figure 1b and c). Subsequent Magnetic Resonance Imaging (MRI) was then carried out on a 3T scanner. The protocol included T1- and T2-weighted sequences, Magnetic Resonance Angiography (MRA) with three-dimensional time-of-flight (3D TOF) technique and three-dimensional fast spoiled gradient-echo (3D FSPGR) sequences acquired in axial and coronal planes (slice thickness: 6 mm) after contrast medium administration. MRI confirmed the anomalous bifurcation and dissection with thrombosis of the false lumen in both 3D TOF and 3D FSPGR sequences (Figure 2 and 3). Associated signs of right hypoglossal nerve palsy were also observed (Figure 4). The patient was managed conservatively with antihypertensive therapy and remains clinically stable on follow-up.

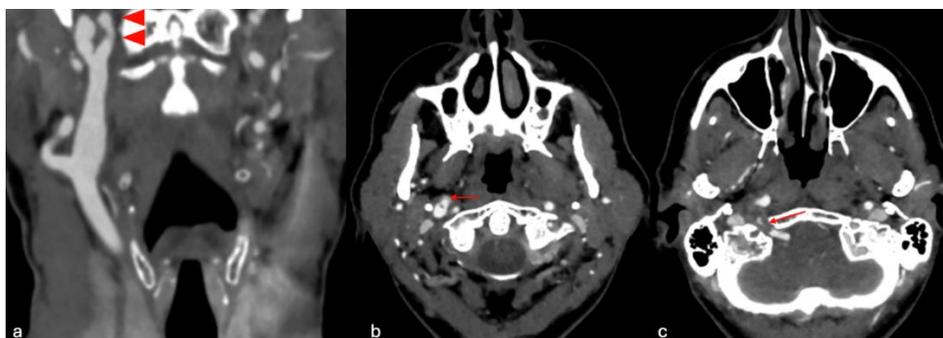


Figure 1: Coronal post-contrast CT showing abnormal origin of a vascular structure from the extracranial ICA (a, arrowhead). Axial post-contrast image: dissection flap within the ICA (b, arrow) extending to the PPHA, whose false lumen appears thrombosed within the hypoglossal canal (c, arrow).

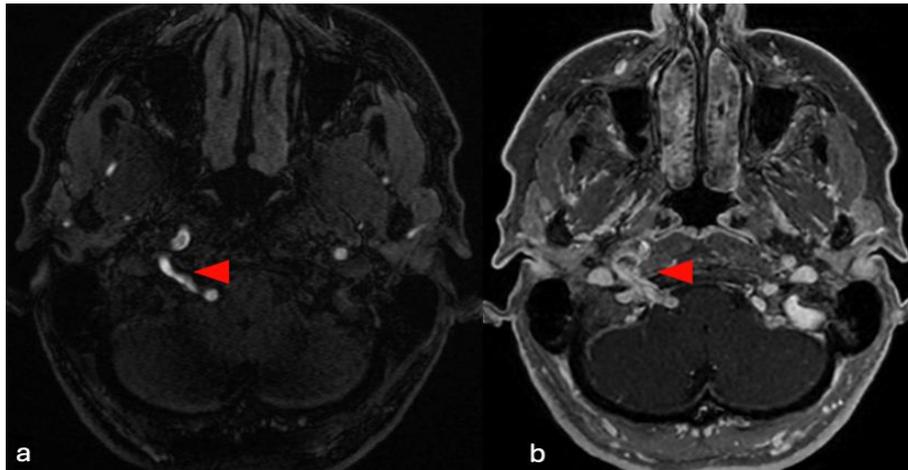


Figure 2: Axial 3D TOF (a) and T1 3D-FSPGR (b) images showing the anomalous vascular structure within the hypoglossal canal.

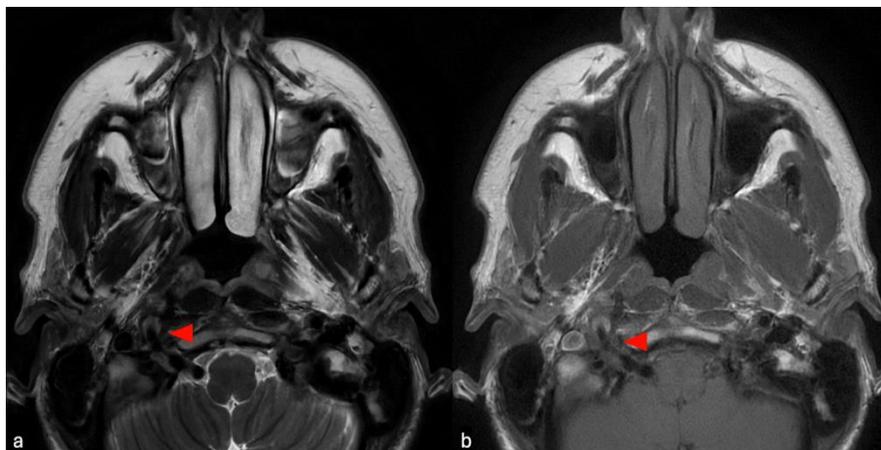


Figure 3: T2 (a) - and T1 (b)-weighted images showing a double-lumen appearance, consistent with arterial dissection complicated by thrombosis of the false lumen involving the PPHA in the hypoglossal canal.

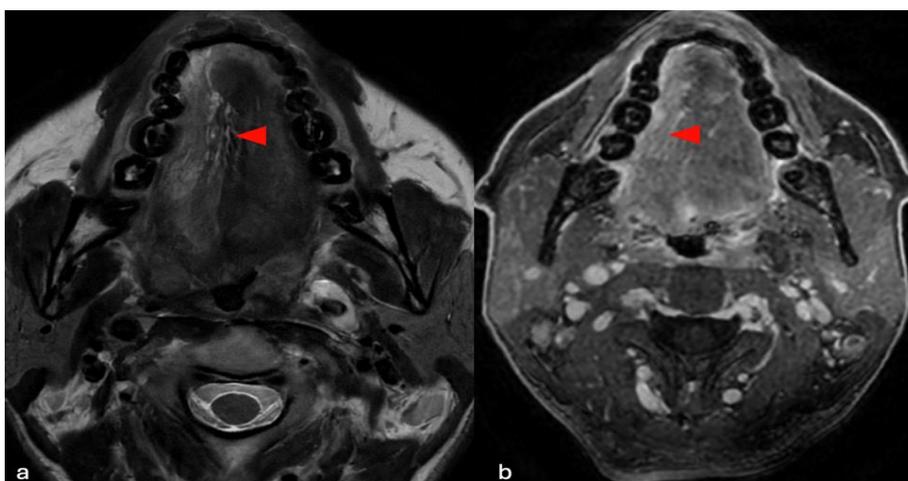


Figure 4: Axial T2 (a) and T1 3D-FSPGR (b) sequences showing edema of the right tongue, with its base prolapsing into the ipsilateral glossoepiglottic vallecula.

DISCUSSION

PPHA is a rare vascular variant that may play a critical role in the posterior circulation, especially when vertebral arteries are hypoplastic or absent. Arterial dissection directly affecting the PPHA itself is exceptionally uncommon; previous reports have described ICA dissection occurring in patients with a PPHA, while the anomalous vessel itself was not directly involved [6]. In our case, imaging demonstrated direct dissection of the PPHA itself, expanding the spectrum of reported complications. The clinical presentation correlated with imaging findings of hypoglossal nerve palsy, likely due to mechanical or ischemic compromise within the hypoglossal canal. MRI and MRA sequences were both crucial in delineating the vascular anomaly as well as associated hypoglossal nerve pathology. Conservative management was appropriate in the absence of ischemic complications, but awareness of PPHA is crucial when planning surgical or endovascular interventions in the carotid region.

This case underscores the importance of recognizing PPHA as more than an incidental finding. It may be a potential site of dissection and cranial neuropathy. Early recognition of such variants is essential for accurate diagnosis and safe management of cervico-cranial vascular disease.

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