



Multi-Territorial Stroke in a Patient with Persistent Hypoglossal Artery

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Introduction

Persistent Hypoglossal Artery (PHA) is a rare anatomical variant characterized by an abnormal anastomosis between the Internal Carotid Artery (ICA) and the Vertebral Artery (VA) [1]. PHA passes through the hypoglossal canal, connects the ICA and VA, and serves as the primary blood supply route to the posterior fossa [1]. In this report, we presented a case of acute stroke, where the presence of PHA resulted in an intriguing distribution of infarction lesions.

Case Presentation

A 70-year-old male patient presented with a sudden onset of vertigo and difficulty in speech, accompanied by numbness in his right-sided limbs for the past three days. He had a 40-year history of arterial hypertension and a 50-year history of smoking. Upon neurological examination, the patient exhibited impaired memory, anomia, and right hemianopia. Additionally, he demonstrated reduced sensation of pain and ataxia in his right arm and leg.

All routine biochemical tests conducted on the patient were within normal limits. Magnetic Resonance Imaging (MRI) of his head revealed multiple acute infarcts distributed across his right frontal and parietal lobes, left temporal and occipital lobes, left thalamus, right pons, and bilateral cerebellar hemispheres. These infarcts provided a complete explanation for his presented symptoms. An ultrasound examination detected a mild stenosis in his right Common Carotid Artery (CCA) and both vertebral arteries were hypoplastic. Computed Tomography Angiography (CTA) confirmed the presence of a mild stenosis in his right CCA, accompanied by calcified plaques. Additionally, the bilateral vertebral arteries were hypoplastic and occluded at the C1-C2 vertebral body level. A Persistent Hypoglossal Artery (PHA) originated from the right Internal Carotid Artery (ICA), traversed through an enlarged right hypoglossal canal, and formed the basilar artery. This anatomical variant was further confirmed by Digital Subtraction Angiography (DSA). His electrocardiogram and echocardiogram were both within normal limits, and no underlying cause of cardiogenic embolism was identified.

During his hospitalization, the patient was orally administered 100 mg/d of aspirin and 20 mg/d of atorvastatin. Over the course of treatment, his symptoms of vertigo and speech difficulty gradually improved, enabling him to be discharged from the hospital two weeks later.

Discussion

PHA, a rare anatomic variant, occurs when the hypoglossal artery fails to regress during embryonic development, resulting in a persistent link between the carotid and vertebral artery systems [2,3]. Reported incidence rates range from 0.03% to 0.26% [2,3]. Typically, PHA originates as a branch of the ICA at the level of the C1-C3 vertebral body [4,5]. Ascending anterior to the vertebral artery, it traverses the enlarged hypoglossal canal, enters the posterior cranial fossa, and joins the lower portion of the basilar artery [4,5]. This anatomic variant often coincides with hypoplasia of the vertebral artery and circle of Willis, making it a significant factor in posterior circulation strokes [6]. Although PHA is often discovered incidentally, its identification is clinically significant for understanding the etiology of ischemic stroke [2,7]. Multi-territorial infarcts are typically indicative of cardiogenic embolism in stroke patients. In our case, however, the concurrent involvement of both anterior and posterior vascular territories, which closely aligned with the supplying regions of the patient's right ICA and its branch of PHA, suggested a more likely cause of

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artery-to-artery embolism.

Conclusion

PHA, a rare carotid-vertebrobasilar artery anastomosis, often serves as the primary blood supply route for the posterior circulation, particularly when accompanied by hypoplasia of the Vertebral Artery (VA). Therefore, a precise assessment of PHA is crucial for determining the etiology of strokes in clinical settings.

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