CASE REPORT

Primitive Hypoglossal Artery: A case report

Mohamed Samir Shaaban *

Diagnostic and Interventional Radiology Department, Faculty of Medicine, Alexandria University, Egypt

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KEYWORDS
Carotid artery anomalies; Circle of Willis; Persistent hypoglossal artery; Basilar artery

Abstract We report a case of a left sided Primitive Hypoglossal Artery detected by multi-detector CT cerebral angiography, performed for a 47 years old female patient complaining of sudden onset of drowsiness, who had undergone non-contract CT of the brain which revealed small ischemic infarcts in the left cerebellar peduncle. On MDCT cerebral angiography, the left internal carotid artery passes through the left hypoglossal canal, to form the basilar artery, while the left carotid canal was empty. The left anterior and middle cerebral arteries were formed through patent and prominent anterior communicating artery.

1. History

47 years old female suffered sudden onset of drowsiness and vertigo, with mild headache. Her vital signs and routine laboratory tests were normal. Upon initial non-contrast CT of the brain, small hypodense foci were detected in the left cerebellar peduncle, diagnosed as ischemic infarcts. Doppler examination of the carotid arteries was normal. MDCT angiography of the cerebral circulation was performed by 20-Detectors Siemens SOMATOM Definition AS (Siemens Medical Solutions, Malvern, PA), with 20 × 0.6 collimation, 0.5 Rotation time, and 1.4 Pitch. Reconstruction was performed by 0.75 mm slice thickness and 0.5 increment. Images were processed by Syngo CT Workplace VA44A with multiplanar reformatting and Volume Rendering Technique. The patient was injected with 100 mL of meglumine ioxitalamate 350 mg I/ml using a power injector (Medrad, Vistrone CT) at the rate of 5 ml per second through an 18 gauge venous cannula in the left antecubital vein.

2. Imaging findings

Upon the initial non-contrast CT of the brain, small hypodense ischemic infarcts were detected in the left cerebellar peduncle and left cerebellar hemisphere, in the territory of the left superior cerebellar artery (Fig. 1). On CT cerebral Angiography, The upper cervical parts of both internal carotid arteries were normal in course and caliber (Fig. 2). The left internal carotid artery deviated postero-medially at the level of CV1, to enter the skull cavity via the left hypoglossal canal, leaving the left carotid canal empty (Fig. 3). It then curved upward and then medially to form the basilar artery which gave origin to the posterior cerebral arteries bilaterally and the right superior cerebellar artery. The left superior cerebellar artery was markedly attenuated, which explains the left cerebellar peduncle infarcts (Fig. 4). The left posterior communicating artery was hypoplastic, while the right posterior communicating artery was patent (Figs. 5 and 6). The canalicular, cavernous and supra-clinoid portions of the left internal
Fig. 1  Axial non-contrast CT scan at the level of cerebellum showing ischemic infarct in the left cerebellar peduncle and left cerebellar hemisphere (yellow arrow).

Fig. 2  Axial CT cerebral angiography scan in arterial phase at the level of CV1–CV2, showing both internal carotid arteries of both sides at their normal sites (yellow arrows).
Fig. 3  Axial CT cerebral angiography scan in thin MIP reformatting at the level of skull base showing the left internal carotid artery passing into the left hypoglossal canal (yellow arrow).

Fig. 4  Coronal CT cerebral angiography scan thin MIP reformatted in arterial phase (a) showing the left internal carotid artery emerging from the left hypoglossal canal to form the basilar artery (yellow arrow). The left superior cerebellar artery is markedly attenuated (white arrow), while the right is patent (red arrow). (b) The corresponding thin VRT scan. Both vertebral arteries are hypoplastic.
carotid artery were absent till the bifurcation into the left anterior and middle cerebral arteries, supplied via the patent anterior communicating artery. Both vertebral arteries were hypoplastic (Fig. 4).

3. Discussion

Multidetector computed tomographic (CT) angiography has been considered as the first line technique to assess the cerebral circulation in the cases of acute stroke and subarachnoid hemorrhage (1,2). Variations of the cerebral circulation, in particular of the circle of Willis, are common, and it is important to understand the appearance of these normal variants, their prevalence, and their clinical relevance, as they might be mistaken for arterial occlusions in cases of hypoplastic or aplastic arteries, and as some of these variants are associated with aneurysm formation (3,4). The sensitivity and specificity of multidetector CT angiography for detection of intracranial vascular anomalies are reported to be up to 81 to 90% and 93%, respectively (5).

Variants of the cerebral circulation can be classified into fenestrations/duplications, circle of Willis variants, persistent anastomoses between the carotid and basilar arteries, and others (5–7). Persistent carotid–basilar anastomoses originate due to faulty development of the internal carotid arteries with faulty anastomosis at three major sites with the paired longitudinal neural arteries that constitute the primitive vertebrobasilar system (8). These arteries are named the trigeminal, otic, and hypoglossal arteries. There are seven transversely oriented arteries in the cervical region. The most cephalic of these arteries is the proatlantal intersegmental artery (8,9). Failure of these vessels to regress during embryonic development results
in various persistent carotid–vertebrobasilar anastomoses, including Persistent Trigeminal Artery, Primitive Hypoglossal Artery, Proatlantal Intersegmental Artery, Persistent Otic Artery, Persistent Dorsal Ophthalmic Artery, and Persistent Primitive Olfactory Artery (3). The persistent hypoglossal artery is the second most common carotid–vertebrobasilar artery anastomosis, following the persistent trigeminal artery, with a prevalence of 0.02–0.10% (4,10). It originates from the internal carotid artery at the levels of the upper cervical spines, courses through the hypoglossal canal, and anastomoses with the basilar artery (11,12). In 79% of cases, the posterior communicating arteries are hypoplastic, and in 78% of cases, the vertebral arteries are hypoplastic (3).

In our case, the facts that the anomalous artery arises from the level of C1-3, then passes through the hypoglossal canal to form the basilar artery which is filled only distal to the point of junction, and that the ipsilateral posterior communicating artery is severely hypoplastic, all meet the “Lie criteria” used to diagnose a Primitive Hypoglossal Artery (13,14).

Conflict of interest

The author declares that they have no conflict of interest.

References


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