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Rare Bilateral Congenital Anomalies of the Internal Carotid Artery Revealed by an Ischemic Stroke: A Case Report and Literature Review

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Abstract **Case Report**

Congenital anomalies of the internal carotid artery (ICA) are exceptionally rare vascular developmental abnormalities. Although frequently asymptomatic because of collateral cerebral circulation, these anomalies may be revealed by ischemic or hemorrhagic cerebrovascular events. We report the case of a 66-year-old hypertensive patient presenting with language disturbances and right-sided paresthesias. Brain CT and MRI demonstrated a subacute ischemic lesion involving the left thalamus associated with bilateral ICA anomalies characterized by agenesis of the right ICA and severe hypoplasia of the left ICA. Multimodal imaging, including MR angiography and CT angiography, confirmed the congenital nature of the anomalies and demonstrated collateral supply through enlarged posterior communicating arteries. This observation highlights the importance of recognizing congenital ICA dysgenesis and the major role of imaging in diagnosis, characterization of collateral pathways, and detection of associated vascular complications.

Keywords: Internal Carotid Artery Agenesis, ICA Hypoplasia, Congenital Vascular Anomaly, Ischemic Stroke, MR Angiography, CT Angiography, Collateral Circulation.

INTRODUCTION

Congenital absence or hypoplasia of the internal carotid artery is an uncommon vascular anomaly with a prevalence estimated at less than 0.01% of the population. The term congenital ICA dysgenesis encompasses agenesis, aplasia, and hypoplasia. Agenesis corresponds to complete absence of the vessel and carotid canal, whereas hypoplasia refers to incomplete arterial development associated with a narrow carotid canal.

Most patients remain asymptomatic because of the development of efficient collateral circulation through the circle of Willis and persistent embryonic vascular pathways. However, some patients may present with ischemic stroke, transient ischemic attacks, headache, or intracranial aneurysms resulting from altered cerebral hemodynamics.

Recognition of these anomalies is essential because they may influence surgical and endovascular planning and may mimic acquired carotid occlusive disease. We report a rare case of bilateral congenital ICA anomalies revealed by an ischemic stroke and discuss the radiological, embryological, and clinical aspects of this entity.

CASE REPORT

A 66-year-old patient with a history of hypertension presented with progressive language disturbances and right-sided paresthesias evolving over five days. No associated loss of consciousness, visual symptoms, or seizures were reported.

Brain MRI demonstrated a left thalamic lesion involving the anterior polar region, characterized by FLAIR hyperintensity, subtle diffusion hyperintensity, and elevated ADC values, consistent with a subacute ischemic infarction.

Time-of-flight MR angiography demonstrated absence of visualization of the intracranial segments of the right internal carotid artery. The left ICA appeared diffusely hypoplastic, particularly at the cervical and petrous segments, with absence of visualization of its cavernous and supraclinoid portions. The basilar artery and posterior cerebral arteries were dilated and tortuous, supplying the anterior circulation through enlarged

posterior communicating arteries. The anterior and middle cerebral arteries appeared slender and were predominantly supplied by the posterior circulation.

CT angiography confirmed the reduced caliber of both common carotid arteries. The right ICA was not visualized beyond the post-bulbar segment, while the left ICA demonstrated severe hypoplasia involving its cervical and petrous portions. Bone window images demonstrated bilateral hypoplasia of the carotid canals, supporting the congenital origin of the anomalies. No intracranial aneurysm was identified.

The imaging findings were consistent with bilateral congenital ICA dysgenesis associated with collateral compensation through the vertebrobasilar circulation.

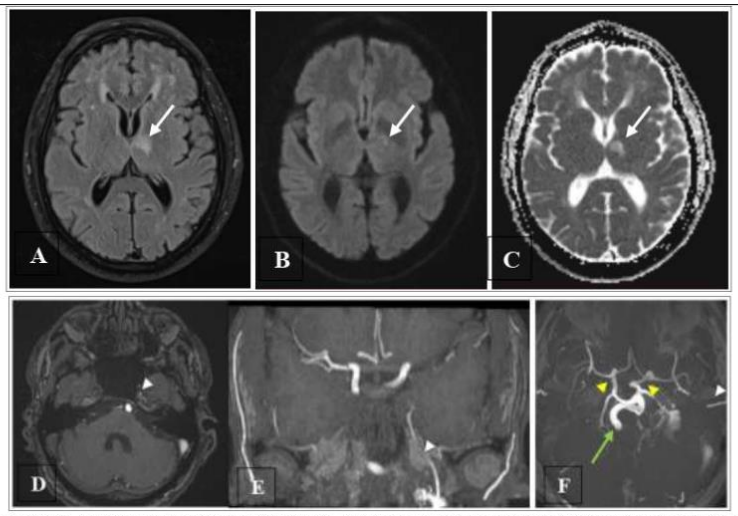

Fig. 1: Axial FLAIR (A), Diffusion B1000 (B) with ADC mapping and arterial TOF angiography (D,E and F) showing: Signal abnormality in the anterior polar region of the left thalamus, characterized by FLAIR hyperintensity, subtle diffusion hyperintensity, and elevated ADC (arrows)

On arterial TOF angiography sequences: Absence of visualization of the intracranial segments of the right internal carotid artery (ICA). The left ICA appears slender with a hypoplastic aspect at its proximal cervical and petrous segments (arrow heads), with no visualization of its remaining intracranial segments. The basilar trunk is tortuous and dilated (green arrow), without any clearly detectable aneurysmal image, giving rise to two posterior cerebral arteries which also appear dilated (yellow arrow heads). The middle, anterior, and anterior communicating cerebral arteries are slender and are supplied by the posterior communicating arteries, which appear tortuous with increased caliber.

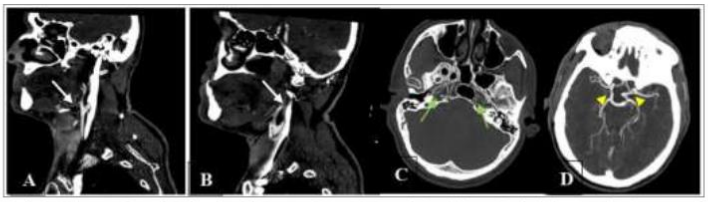

Fig. 2: A CT angiography with Sagittal images through the right (A) and left (B) carotid planes, axial bone window slice (C), and axial MIP slices of the circle of Willis (D) showing : The common carotid arteries have a typical origin but appear reduced in caliber. There is no visualization of the post-bulbar segment of the right internal carotid artery

DISCUSSION

The internal carotid arteries provide the major arterial blood supply to the cerebral hemispheres. Congenital anomalies of the ICA are rare developmental abnormalities resulting from disruption of embryological vascular formation during the fourth to sixth weeks of gestation. These anomalies include agenesis, aplasia, and hypoplasia, which differ according to the degree of embryological development failure.

The embryogenesis of the ICA is complex and involves fusion of several embryonic vascular segments derived from the first and third aortic arches as well as the dorsal aorta. Failure of normal development of one or more embryonic segments may result in complete absence or incomplete formation of the artery. Lasjaunias and Santoyo-Vazquez described seven embryological segments involved in ICA formation, providing an anatomical basis for the diversity of congenital ICA anomalies.

Congenital ICA dysgenesis may remain asymptomatic for decades because of collateral cerebral circulation. Several collateral pathways have been described, including compensation through the circle of Willis, persistent embryonic arteries, intercavernous anastomoses, ophthalmic arteries, and transcranial branches from the external carotid artery system. Lie and

Hage classified six major collateral circulation patterns associated with ICA agenesis and hypoplasia. In our patient, collateral supply predominantly originated from the vertebrobasilar circulation through markedly enlarged posterior communicating arteries, corresponding to a variant of type E collateralization.

Differentiating congenital ICA anomalies from acquired carotid occlusive disease is crucial. Acquired ICA occlusion caused by atherosclerosis, dissection, vasculitis, fibromuscular dysplasia, or Moyamoya disease usually occurs in the presence of a normally developed carotid canal. In contrast, congenital ICA agenesis or hypoplasia is associated with an absent or narrow carotid canal because carotid canal development depends on the presence of the ICA during embryogenesis.

Imaging plays a central role in diagnosis. Doppler ultrasound may demonstrate reduced caliber and decreased blood flow within the ICA. MRI and MR angiography allow evaluation of ischemic lesions, collateral pathways, and associated vascular abnormalities. CT angiography with multiplanar and three-dimensional reconstructions provides excellent evaluation of vascular anatomy and skull base osseous structures, particularly the carotid canals. In our case, the demonstration of bilateral hypoplastic carotid canals strongly supported the congenital nature of the anomaly.

One of the major clinical implications of ICA dysgenesis is the increased incidence of intracranial aneurysms. The prevalence of aneurysm formation in patients with congenital ICA anomalies is significantly higher than in the general population because of chronic hemodynamic stress within collateral vessels. Rupture of these aneurysms may result in subarachnoid hemorrhage or intracerebral hemorrhage. Consequently, long-term vascular imaging surveillance is recommended even in asymptomatic patients.

Although many patients remain asymptomatic, some may develop ischemic cerebrovascular events because of insufficient collateral perfusion or embolic phenomena. In our patient, the ischemic thalamic lesion was likely related to altered cerebral hemodynamics in the setting of bilateral ICA dysgenesis.

No standardized therapeutic strategy currently exists for congenital ICA anomalies. Management is mainly preventive and focuses on control of vascular risk factors, particularly hypertension and smoking. In patients with associated aneurysms, endovascular or surgical treatment may be required depending on aneurysm size, morphology, and rupture risk. Recognition of these anomalies is also essential before skull base surgery, carotid interventions, or endovascular procedures to avoid potentially catastrophic complications.

CONCLUSION

Congenital agenesis and hypoplasia of the internal carotid artery are rare vascular developmental anomalies that may remain asymptomatic until revealed by ischemic or hemorrhagic cerebrovascular events. Recognition of these entities is essential to avoid misdiagnosis and inappropriate management. Multimodal imaging, including MRI, MR angiography, CT angiography, and skull base CT, plays a pivotal role in diagnosis, assessment of collateral circulation, and detection of associated aneurysms. Long-term clinical and radiological follow-up is recommended because of the increased risk of cerebrovascular complications.

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