

CASE REPORT

Ascending pharyngeal artery arising from a hypoplastic internal carotid artery

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ABSTRACT

Normal vascular variants often have clinical/surgical significance and can be misinterpreted for pathology. We report a case ascending pharyngeal artery arising from a hypoplastic internal carotid artery. We provide clues to differentiate between dysgenesis and disease/thrombosis of the internal carotid artery.

Key words: Carotid canal, dysgenesis of internal carotid artery, hypopharyngeal artery, vascular variants

INTRODUCTION

Dysgenesis of the ICA is a rare developmental anomaly seen in <0.01% of the population.^[1,2] The term incorporates agenesis (no carotid canal or vascular remnant), aplasia (vascular remnant and hypoplastic carotid canal), or hypoplasia (small caliber, patent lumen). These abnormalities often have clinical/surgical significance and can be misinterpreted for pathology. We report a case ascending pharyngeal artery arising from a hypoplastic internal carotid artery.

CASE REPORT

A 15-year-old African American girl, with past medical history significant for juvenile onset diabetes mellitus, underwent sinonasal surgery for fungal sinusitis. The preoperative magnetic resonance imaging (MRI) of the sinuses had shown absence of the flow void from the cavernous portion of the right internal carotid artery (ICA) while the left sided flow void was normal [Figure 1]. Surgery did not document fungal disease extension into the either cavernous sinus. The patient was referred to a postoperative computed tomography angiography (CTA) of the head and neck for better evaluation of the right ICA.

Computed tomography angiography showed a normal caliber of the right common carotid artery (CCA), external

carotid artery (ECA) as well as the proximal 1 cm of the right ICA. After the normal first centimeter, the ICA became of narrow caliber, without evidence of thrombus or dissection, and remained of homogeneous small caliber all the way into a hypoplastic carotid canal. Findings confirmed the congenital nature of the small ICA [Figure 2].

Arising from the medial aspect of the proximal ICA was a small caliber vessel that ascends vertically between the ICA and pharynx, to the under surface of the base of the skull, lying on the longus capitis muscle. The course of this artery was consistent with an ascending pharyngeal artery (AscPA). This artery did not provide any intracranial branches or any feeders to the ICA distribution.

The remainder of the CTA was normal, namely the ICA was of normal course and caliber, and there was no evidence of intracranial aneurysms. The left AscPA was not identified with certainty.

DISCUSSION

Dysgenesis of the ICA is a rare developmental anomaly seen in <0.01% of the population.^[1,2] The term incorporates agenesis (no carotid canal or vascular remnant), aplasia (vascular remnant and hypoplastic carotid canal), or hypoplasia (small caliber, patent lumen).^[3] The abnormality is

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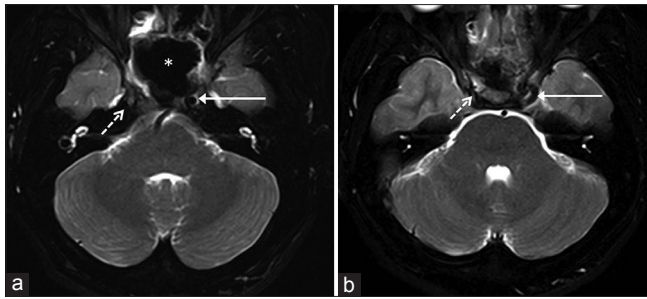


Figure 1: (a) Axial T2-weighted fat-saturated images at the level of the cavernous sinus normal flow void (solid arrow) from the precavernous segment of the left internal carotid artery (ICA). There is the loss of the flow void (dashed arrow) from the same segments of the right ICA. Patient has expansion of the sphenoid sinus that is densely opacified on computed tomography (CT) (not shown) and shows a signal void on T2-weighted (asterisk) consistent with fungal disease ICA: Internal carotid artery. (b) Axial T2-weighted fat-saturated images at the level of the cavernous sinus show normal flow void (solid arrow) from the cavernous segment of the left ICA. There is the loss of the flow void (dashed arrow) from the same segments of the right ICA. Patient has expansion of the sphenoid sinus that is densely opacified on CT (not shown) and shows a signal void on T2-weighted (asterisk) consistent with fungal disease ICA: Internal carotid artery

usually unilateral with nearly 3:1 left-sided predominance.^[4] Bilateral dysgenesis has been described however.^[5]

Etiology is unknown,^[5] but it is postulated that all the three variants of dysgenesis represent a spectrum related to an intrauterine insult to the developing embryo. The ICA is thought to arise from the dorsal aorta and the third aortic arch at 4–5 mm embryonic stage, with the full development of the ICA by 6 weeks.^[3,5] The artery usually forms, followed soon after by the bony carotid canal. Hence, the size of the canal is essential for making the diagnosis of a hypoplastic ICA. A small ICA is considered hypoplastic if the ICA canal is small; however a small ICA in the setting of a normal sized carotid canal is abnormal secondary to atherosclerotic disease, dissection, vasculitis, etc. By the same token, an absent ICA is considered congenital agenesis rather than thrombosis if the ICA canal is not developed.

As ICA dysgenesis is not associated with ECA or CCA abnormalities, it is suggested that the latter two arteries have a different embryogenesis.^[5]

Many collateral pathways have been described in the setting of ICA dysgenesis.^[3] A simplified classification includes three collateral pathways: Collateral flow through the circle of Willis (most frequent), collateral flow via persistent fetal circulation, and reconstitution of the ICA through skull base collaterals from the ECA.^[2,5]

These patients are at a much higher risk of developing aneurysms. The risk is estimated at 24–34% when compared to 2–4% prevalence in the general population.^[5] Altered flow dynamics along with congenital defects of the vessel wall are

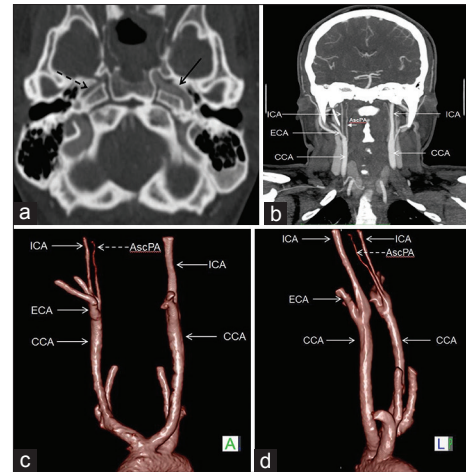


Figure 2: (a) Axial computed tomography angiography the petrous carotid canal is normal on the left (solid black arrow) and hypoplastic on the right (dashed black arrow) Arising from the medial aspect of the proximal internal carotid artery (ICA) is a small caliber vessel consistent with ascending pharyngeal artery (AscPA), AscPA: Ascending pharyngeal artery, ECA: External carotid artery, ICA: Internal carotid artery. (b) Coronal maximum intensity projection 1 cm after its origin, the right ICA is of diffusely narrow caliber, without evidence of thrombus or dissection, and remained of homogeneous small caliber all the way into the skull Arising from the medial aspect of the proximal ICA is a small caliber vessel consistent with AscPA, AscPA: Ascending pharyngeal artery, CCA: Common carotid artery, ECA: External carotid artery, ICA: Internal carotid artery. (c) Three-dimensional volumetric reconstruction 1 cm after its origin, the right ICA is of diffusely narrow caliber, without evidence of thrombus or dissection, and remained of homogeneous small caliber all the way into the skull. Arising from the medial aspect of the proximal ICA is a small caliber vessel consistent with AscPA, AscPA: Ascending pharyngeal artery, CCA: Common carotid artery, ECA: External carotid artery, ICA: Internal carotid artery. (d) Three-dimensional volumetric reconstruction 1 cm after its origin, the right ICA is of diffusely narrow caliber, without evidence of thrombus or dissection, and remained of homogeneous small caliber all the way into the skull. Arising from the medial aspect of the proximal ICA is a small caliber vessel consistent with AscPA, AscPA: Ascending pharyngeal artery, CCA: Common carotid artery, ECA: External carotid artery, ICA: Internal carotid artery

thought to be the cause of this increased risk.^[1,5] Follow-up vascular imaging criteria are not established.

Our patient had juvenile onset diabetes mellitus and family history of cardiac and peripheral vascular disease all of which make her a high-risk patient for cerebrovascular disease. This vascular variant also increases her risk for intracranial aneurysms. She was counseled about the need for aggressive glucose level control and regular medical follow-ups.

CONCLUSION

A hypoplastic ICA is a rare developmental anomaly that can be misdiagnosed for a thrombosed ICA on MRI. Radiologists should be aware of this variant and be able to differentiate between dysgenesis of the ICA and a ICA disease/thrombosis based on the size of the ICA canal on computed tomography. This entity is clinically significant as it places the patient at significantly increased risk of intracranial aneurysms.

To the best of our knowledge, an AscPA arising from a hypoplastic ICA has never been described. It is difficult to establish if this combination represents an incidental coexistence of two rare developmental anomalies or whether this represents part of the spectrum of ICA hypoplasia. As indicated above, ICA dysgenesis has not been associated with ECA or CCA abnormalities, suggesting that the latter two arteries have a different embryogenesis,^[5] therefore we favor that the findings in this case are a mere coincidence.

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