CASE REPORT

Congenital absence of the internal carotid artery in a patient with spinal cord injury: case report

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Study design: Case report.

Objective: To describe a rare case of congenital absence of the internal carotid artery (ICA) in the setting of acute spinal cord injury and review the existing literature.

Setting: Regional Spinal Cord Injury Center of the Delaware Valley, Philadelphia, PA, USA.

Clinical presentation: We report on a 47-year-old male who presented as a T2 ASIA A spinal cord injury with a T2–T3 fracture dislocation injury after a 25-foot fall from a tree. Magnetic resonance angiography imaging of the neck carried out as per routine spinal trauma protocol suggested an acute traumatic injury to the carotid artery. Therefore, computed tomographic angiography imaging of the neck was performed, which revealed absence of the left ICA and a diminutive left carotid canal at the skull base, suggesting congenital absence. Follow-up magnetic resonance imaging of the brain to evaluate for acute infarct showed no diffusion restriction contributing to the evidence that this was a congenital anomaly and not traumatic occlusion of the artery.

Conclusion: Congenital absence or hypoplasia of the ICA is a rare anomaly. It should be considered in the differential in patients who have absent or diminished carotid arteries on radiologic imaging in the setting of trauma.

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Introduction

Congenital internal carotid artery (ICA) absence, a term that includes agenesis, aplasia and hypoplasia, of the ICA is a rare vascular anomaly.¹ It is seen in less than 0.01% of the population.² The majority of cases are discovered incidentally on imaging of the head and neck.¹ Absence of the ICA has been associated with intracranial vascular abnormalities, particularly cerebral aneurysms, which may require further imaging and surveillance.³

Case report

A 47-year-old male with no significant medical history fell 25 feet from a tree. He was transferred from an outside institute and his examination was consistent with a T2 ASIA A injury. Plain radiographs of the chest and spine were obtained revealing a T2–T3 fracture dislocation injury. Computed tomography and magnetic resonance imaging (MRI) of the

spine with reformatted images detailed the T2–T3 injury and deformation of the spinal canal (Figure 1).

MRI of the cervical and thoracic spine with concurrent magnetic resonance angiography (MRA) of the neck vessels were obtained as per our spinal cord injury protocol. These studies revealed absence of the left ICA. Given the clinical picture, this was initially believed to represent traumatic occlusion of the artery. To confirm this, a computed tomographic angiography (CTA) of the head and neck was performed from the level of the heart to the vertex to evaluate both a possible aortic tear and the carotid circulation. These studies were suggestive of congenital absence of the left ICA rather than traumatic occlusion (Figure 2).

No evidence of aneurysm or dissection was found. There was no evidence of a left carotid canal suggesting developmental anomaly (Figure 3). The left middle cerebral artery originated from the left posterior cerebral artery. The left anterior cerebral artery originated from the anterior communicating artery.

An MRI with diffusion weighted imaging performed on hospital day 5 showed no evidence of acute ischemic event, essentially eliminating the possibility of an acute traumatic occlusion.

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Figure 1 Sagittal T2-weighted magnetic resonance imaging sequence revealing a severe T2–T3 fracture dislocation injury in the thoracic spine.

Figure 2 Coronal computed tomographic angiogram reconstruction showing patent right common and internal carotid arteries (black arrows), a diminutive common carotid on the left, and absent internal carotid artery on the left (white arrow).

Discussion

The first report of internal carotid hypoplasia was described in 1787.¹ Since that time, numerous reports of this anomaly have been published and the underlying developmental pathology has also been well described.^{2,4} Retrospective estimates of incidence of this anomaly vary between 0.01 and 0.24% of the population.⁵ Some authors believe that the true incidence in the general population is higher as most cases are clinically asymptomatic.^{1,2}

Lie¹ described two modes of collateral circulation in patients with unilateral absence of the internal carotid. In one, the ipsilateral middle cerebral artery arises from the posterior cerebral or basilar artery. This is the more common formation and was the case in the current patient. In the other, the ipsilateral middle cerebral artery is supplied by the anterior communicating artery. In both cases the ipsilateral anterior cerebral artery is supplied by the anterior communicating artery.

Figure 3 Non-contrast computed tomographic scan of the skull base demonstrating a carotid canal on the right side (black arrow) but not on the left.

The collateral blood supply through the circle of Willis often leads to findings of normal perfusion in the affected territories in these patients.⁵ This is in contrast to traumatic occlusion or stenosis where perfusion is reduced and the risk of ischemic events is higher.

Anomalies of the internal carotid are associated with intracranial aneurysms.⁴ The prevalence of cerebral aneurysms in the general population is estimated at 1–5%, whereas the rate in those with congenital ICA absence is 24–34%.² This increased risk is believed to be because of hemodynamic stress and abnormal flow through collateral circulations in these patients.²

The finding of asymmetric or absent carotid canals on skull base or plain head computed tomography should raise suspicion of ICA anomalies.³ A diminutive carotid canal may distinguish hypoplasia from atherosclerosis, dissection, or other acquired causes of a diminished vessel caliber. In the current case, this finding raised suspicion of a congenital cause for absence of the left internal carotid on imaging and led to further investigation. Some authors suggest that these findings alone should prompt further evaluation, even in asymptomatic patients, because of the association with cerebral aneurysms and subarachnoid hemorrhage.³

Conclusion

We report an interesting case of congenital absence of the ICA in the setting of a traumatic spinal cord injury. Given the clinical presentation, the radiologic finding was initially thought to represent traumatic occlusion of the artery. However, the findings of an absent carotid canal and collateral flow through an abnormal circle of Willis were more consistent with congenital absence.

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