Late de novo basilar aneurysm after carotid artery injury

Case illustration

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Traumatic carotid artery dissections are rare, and the majority of these lesions are clinically silent. They may present with a delayed stroke related to thromboembolic dissemination from the site of the dissection.2 Less frequently, the dissection may progress to cause significant luminal stenosis or occlusion with subsequent hemodynamic consequences.1

This 37-year-old female presented with Grade V subarachnoid hemorrhage (SAH) secondary to basilar aneurysm rupture. She had sustained bilateral carotid artery dissections and mandibular fractures 12 years previously after a motor vehicle accident. No aneurysms were noted on the angiogram report. She received no specific treatment for the dissections and made a full recovery without any neurological sequelae, with no subsequent follow-up.

Computed tomography demonstrated diffuse subarachnoid and intraventricular blood and a filling defect anterior to the pons (Fig. 1A). Angiography demonstrated a large 10-mm basilar apex aneurysm (Fig. 1C), an occluded right internal carotid artery (ICA; Fig. 1B), and a string sign involving the left ICA (Fig. 1D). The anterior circulation was supplied through external collaterals (Fig. 1D) and large posterior communicating artery (PCoA) vessels filled retrograde by the posterior circulation (Fig. 1C). The patient remained in poor neurological condition, her family elected to withdraw life support, and she died.

Intracranial aneurysm formation has not been recognized in the literature as a possible long-term complication of blunt carotid artery injury. We present the first such case, in which a patient with prior known bilateral traumatic carotid artery dissection developed a de novo basilar apex aneurysm, which may relate to the increased flow in the posterior circulation. (DOI: 10.3171/JNS/2008/108/3/0607)

References


Fig. 1. A: Noncontrast-enhanced CT scan demonstrating diffuse SAH, temporal horn dilation, and the aneurysm’s location anterior to the upper pons. B: Computed tomographic angiogram demonstrating absent opacification of the right ICA and narrowed filling of the left ICA vessel (black arrow). C: Lateral projection vertebral angiogram demonstrating the basilar apex aneurysm. The posterior circulation supplies the PCoA vessels (black arrow) to provide collateral flow to the anterior and middle cerebral territories. D: Lateral projection of left and right common carotid angiograms. Bilateral ICA dissections and severe stenoses are noted, with external carotid artery collateralization via ethmoidal and middle meningeal vessels (asterisks).

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