Trigeminal artery and microemboli to the brain stem

Report of two cases

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The authors report two cases of transient ischemic attacks (TIA's) involving the brain stem. The TIA's were due to microemboli that originated from a carotid bifurcation atherosclerotic plaque and travelled through a persistent trigeminal artery.

KEY WORDS • persistent trigeminal artery • microemboli • transient cerebral ischemia

The relationship between carotid circulation transient ischemic attacks (TIA's) and emboli arising from ulcerated atherosclerotic carotid artery bifurcation plaques is well recognized. To our knowledge there are no previous reports of TIA's involving the brain stem secondary to arterial microemboli originating in the internal carotid artery. We are reporting two cases of brain-stem TIA's presumed secondary to ulcerated embolization of the carotid bifurcation in association with a persistent trigeminal artery supplying the posterior circulation.

Case Reports

Case 1

A 54-year-old otherwise healthy man developed acute paresis and numbness of the entire left face that resolved over a 1-week period. This was followed by the abrupt onset of right facial numbness that resolved over several days. He denied extremity paresis, sensory disturbance, or amaurosis fugax, and had no symptoms of generalized vertebrobasilar ischemia.

Examination. A lower motor neuron left facial paresis and hypesthesia of the entire left face in the distribution of the trigeminal nerve were noted on initial examination and resolved over 7 days. Right facial and corneal hypesthesia, which appeared abruptly, were noted several days later and cleared over 3 days. The remainder of the neurological examination was normal. The patient was normotensive with equal brachial blood pressures and had no carotid, subclavian, or vertebral artery bruits.

A cerebral angiogram demonstrated hypoplastic vertebral arteries, normal intracranial circulation, a widely patent basilar artery...
supplied by a large persistent left trigeminal artery, and an ulcerated left carotid bifurcation atherosclerotic plaque without significant stenosis (Fig. 1 left). A moderate degree of stenosis was noted in the midportion of the trigeminal artery, but the angiogram did not demonstrate ulceration as a source of microemboli in that vessel. It was therefore felt that the patient's symptoms and neurological findings were due to microemboli originating at the left carotid bifurcation and travelling through the trigeminal artery to the brain stem.

Operation. A left carotid endarterectomy was performed. A mildly stenotic atherosclerotic plaque was found at the carotid bifurcation. There was a large ulcer crater with a rough, irregular base in the posterolateral wall (Fig. 2).

The postoperative course was unremarkable and the neurological examination remained normal. The patient was seen in follow-up review 18 months postoperatively and had remained asymptomatic.

Case 2

A 62-year-old woman developed acute weakness of her left hand followed by left facial numbness, bilateral visual blurring, vertigo, and loss of consciousness. She was not incontinent. Upon regaining consciousness, the patient was mildly confused and nauseated but her other neurological symptoms had resolved.

Examination. The neurological examination was normal. The patient was normotensive with equal brachial blood pressures and the carotid pulses were full bilaterally without bruits.

A cerebral angiogram demonstrated hypoplastic vertebral arteries, a large right persistent trigeminal artery, and posterior communicating artery supplying the posterior circulation. A right carotid bifurcation atherosclerotic plaque was found to be ulcerated and mildly stenotic (Fig. 1 right). It was felt that microembolization from the carotid bifurcation plaque to the posterior
circulation might explain the episode of cerebral ischemia before admission.

Operation. A right carotid endarterectomy revealed an ulcerated plaque at the right carotid bifurcation. The postoperative course was unremarkable. At follow-up examination 4 months following surgery, the patient had remained asymptomatic.

Discussion

The trigeminal artery is a fetal carotid-basilar anastomosis that generally disappears early in embryonic life. The incidence of persistent trigeminal arteries has been reported to be in the range of 0.1% to 0.6% of patients undergoing cerebral angiography.1,4 While the trigeminal artery has been reported as a location of aneurysms1,3 and has produced trigeminal nerve compression,2 to our knowledge it has not been previously reported to be a pathway for arterial microemboli to the brain stem. This anomaly is most unusual, and has been an indication for surgery in only two of over 450 patients who have undergone carotid endarterectomy by the neurosurgical service in our center. While the pathway of embolization is extraordinary, the concept of embolization from an ulcerated carotid bifurcation plaque is sound, and should be considered in patients with posterior circulation transient ischemic attacks.

References


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