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Flow-diversion headaches in a patient with high-grade internal carotid artery stenosis

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Abstract

Background—High-grade stenosis of the internal carotid artery (ICA) may result in flow diversion to the external carotid artery (ECA) and its branches. Head and facial pain secondary to flow-diversion to ECA and increase in regional blood flow are under-recognized and unreported.

Design/methods—We report a patient with high-grade ICA stenosis and recurrent ipsilateral headaches that resolved after revascularization.

Results—A 69-year-old woman presenting with right-arm weakness had neuroimaging evidence of ipsilateral high-grade ICA stenosis and left hemispheric ischemic stroke. Her history was significant for unilateral recurrent headaches that clinically resolved after left ICA angioplasty and stenting. With prior evidence supporting extracranial vasodilatation as the source of pain in migraine, and current clinical observation, we propose flow-diversion phenomenon as the connection between high-grade ICA stenosis and ipsilateral headache.

Conclusion—Recognition of flow-diversion into the ECA as a possible mechanism for headaches may help in further understanding of ICA disease, its progression, and the effects of carotid revascularization on quality of life. Headache as a likely surrogate marker of carotid stenosis with flow-diversion warrants more research, and may be critical in the early identification of significant ICA stenosis and prevention of TIA or stroke.

Keywords

Carotid artery stenosis; flow diversion; carotid stenting; headache; facial pain; magnetic resonance imaging; carotid angiography

Background

Transient ischemic attack (TIA) and ischemic stroke are recognized symptoms of internal carotid artery (ICA) stenosis secondary to distal embolism or regional cerebral hypoperfusion [1,2]. High-grade stenosis of the ICA may result in diversion and increase of the regional blood flow into the branches of external carotid artery (ECA). Such increase is expected based on mechanical diversion of the common carotid artery flow and activation of collaterals from ECA to ICA via anastomoses from internal maxillary and ophthalmic arteries or the ascending cervical artery within the dura. Symptoms of head and facial pain secondary to such flow-diversion and increase of the regional blood flow into the branches of ECA are under-recognized and unreported. We present a patient with ICA stenosis who developed recurrent ipsilateral headaches with resolution after carotid revascularization.

Case description

A 69-year-old woman initially presented with unilateral (left-sided) headache, peri-orbital and temporal in location. She denied any radiation of the headache to the contralateral side. Headache frequency was reported as daily and severity was 6/10, on a 10-point severity scale. She described the headache as constant and throbbing in character. She denied aura with the headache, but reported associated nausea without vomiting. No particular

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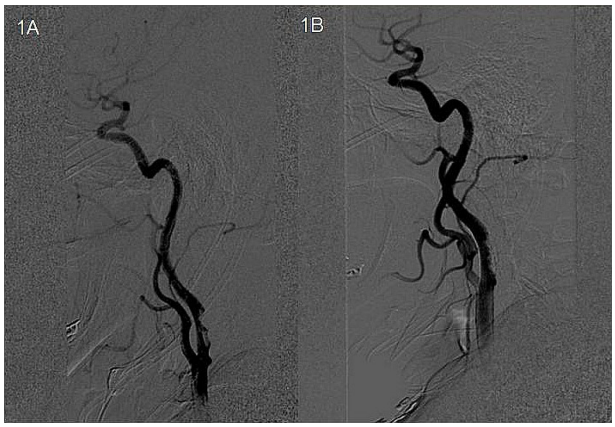

Figure 1. Lateral views of carotid angiogram illustrating high-grade stenosis of the left ICA, with pre- (a) and post-stent placement (B) images shown below.

triggers associated with the headache were reported. She recalled several visits in the past to the primary care physician and emergency department (ED) for treatment of headache. Her treatment included only abortive therapy with Ibuprofen 600 mg and provided marginal pain relief.

After 18 months from onset of headaches, she presented again to ED with transient unilateral right-arm weakness. Magnetic resonance imaging (MRI) of the brain revealed distinct lesions of restricted diffusion within the left cerebral hemisphere involving the fronto-parietal and parieto-occipital areas. The lesions corresponded to areas of decreased signal on the apparent diffusion coefficient map, consistent with subacute ischemic stroke. Chronic focal infarcts within the right frontal lobe and basal ganglia; and scattered chronic microvascular ischemic changes were noted. MR angiography of the neck with and without gadolinium enhancement was obtained which demonstrated a hemodynamically significant stenosis in the left proximal ICA. The stenosis was most severe at 1.6 cm superior to the bifurcation, resulting in a reduction of the cross-sectional diameter estimated between75% and 80%. No evidence of any hemodynamically significant stenosis in the right ICA was noted.

Patient subsequently underwent a single vessel cerebral angiogram with catheterization of the left common carotid artery for angioplasty and stent placement. The lateral projection images demonstrated high-grade stenosis of 80% based on the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria [3] with an ulcerated, irregular plaque in the left ICA. Angioplasty and stent placement was performed using a 5F catheter to gain carotid artery access (Simmons-2), embolic protection device (Angioguard®), balloon dilator (Aviator™), stent system (Precise® Pro Rx®), and a closure device (Angio-Seal™). Procedure was performed under awake condition and post-stent angiogram revealed good stent opposition to vessel wall, and restoration of flow in the ICA after removal of distal protection device [see Figure 1(B)]. Patient's hospitalization was complicated with post-stent hypotension and fluctuation in neurological deficits which resolved prior to discharge. Patient was continued on aspirin and clopidogrel post-procedure. Post-procedure examination revealed no focal neurological deficits and after approximately a week following the procedure, patient reported complete resolution of the headaches. She currently remains headache-free after about 8 weeks since the intervention.

Discussion

Headache is a common neurological complaint and may occur as a primary headache such as migraine or secondary to an underlying pathology ranging from sinus disease to fatal subarachnoid hemorrhage [4]. We believe that the headache was related to the high-grade stenosis of left ICA because of ipsilateral location and resolution following stent placement. The relationship between ischemic stroke and headache, particularly migraine with aura has been reported as coincidental, co-morbid or causal [5]. The International Classification of Headache Disorders (ICHD) II recognizes "Headaches attributed to a cranial or cervical vascular disorders" as a category of secondary headaches and includes: "post-endarterectomy headache", "headache attributed to carotid or vertebral angioplasty", "headache attributed to an intracranial endovascular procedure", etc. [4]. Recogni-

tion of headache and possibly facial pain attributed to flow-diversion phenomenon, within this category of ICHD-II secondary headaches may hold significant diagnostic value and impact overall clinical care.

Flow-diversion to ECA in the setting of significant ICA stenosis is a consideration. Previously, in the rat model [6] with temporary occlusion of the ECA, a significant increase in regional cerebral blood flow to ipsilateral hemisphere was demonstrated [6]. Therefore, an increase in ECA blood flow by mechanical diversion or increased demand is likely in the settings of ICA stenosis. The extracranial terminal branches of ECA are recognized as a significant source of pain in migraine [7]. A prior review elucidates the available experimental, physiological, pharmacological, and clinical evidence supporting extracranial vasodilatation as the source of pain in migraine [7]. ECA with its branches primarily supplies the face and neck, and the meningeal branches; particularly the middle meningeal artery supplies the dura mater. Pain sensitive structures of cranium include the skin, subcutaneous tissue, periosteum, muscles, arteries, venous sinuses and branches, and the trigeminal, glossopharyngeal, and vagus nerves. Pain afferents from anterior scalp, face are carried by the ophthalmic, maxillary and mandibular branches of the trigeminal nerve; and the posterior scalp by cervical branches of nerves II and III. Pain afferents from the extracranial arteries are primarily carried by neighboring cutaneous nerves, and therefore pain may be localized to the area of artery that is inflamed or distended from abnormal flow. However, with involvement of trigeminal nociceptive afferents, the pain signals may be relayed to trigeminal nucleus caudalis (TNC) and eventually to the cortex via the thalamic centers. Moreover, continuous stimulation of TNC may lead to central sensitization [8–11].

This report is only a clinical observation suggesting a link between high-grade ICA stenosis and ipsilateral headache. Further studies are required to determine the varying grades of flow diversion and relationship to headaches. It also remains to be elucidated whether occurrence of flow diversion headaches relates to long-term risk of ischemic events.

Conclusions

Recognition of the flow-diversion phenomenon could be essential for further understanding the ICA disease and its progression. Headache as a likely surrogate marker of carotid stenosis with flow-diversion warrants more research, and may be critical in the early identification of significant ICA stenosis and prevention of TIA or stroke.

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