Carotid Stump Syndrome: A Rare Cause of Stroke

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Background

The cessation of ipsilateral ischemic events following the occlusion of an internal carotid artery (ICA) has been claimed widely. Recurrent ischemic events following the occlusion of ICA are rarely reported but they are commonly known as carotid stump syndrome.

Case Presentation

A 64-year-old smoker man with a past medical history of hypertension and stroke presented in the emergency department (ED) with a 3-day history of speech impairment. He also endorsed a transient right-sided weakness a few days back. Six months prior to this visit, he had left-sided watershed infarct involving frontal and parietal lobe on Magnetic Resonance Imaging (MRI) and was found to have complete occlusion of left Internal Carotid artery (ICA) (Figure 1) and 20-40% stenosis of the right ICA on CT angiography (Figure 2). The patient was on aspirin 81 mg, atorvastatin 80 mg, and took clopidogrel 75 mg for 3 weeks. His vitals on current ED visits showed blood pressure of 81/60 mmHg; Pulse: 116 beats per minute; respiratory rate: 16 per minute; temperature: 98.6 F, and SPO2: 98% on room air. Physical exam was significant for mild expressive aphasia. Repeat magnetic resonance imaging revealed small areas of cortical restricted diffusion in the left parietal and temporal lobes, compatible with acute infarct. Transthoracic echocardiography was unremarkable for intracardiac thrombi or patent foramen ovale. The patient was managed conservatively with aspirin, clopidogrel, and atorvastatin and was referred to a neurovascular surgeon for possible endovascular stenting.

Discussion

Carotid stump syndrome (CSS) is defined as an ischemic stroke (IS) that occurs following occlusion of the ipsilateral Internal Carotid artery (ICA). It is estimated that CSS is responsible for causing 3 to 5% of ipsilateral IS.[1] Multiple mechanisms have been proposed for ipsilateral IS following occlusion of ICA: 1) embolus originating from the stump and traveling in retrograde flow to external-intracranial carotid anastomosis causing ipsilateral IS; 2) embolus originating from contralateral carotid artery traveling through the circle of Willis towards contralateral side of the brain and 3) profound hypotension leading to ipsilateral watershed infarct.[2] However, attempts were made to eliminate hypoperfusion as the cause of stroke in patients with CSS by creating bypass between the external-intracranial carotid artery but most patients still developed ipsilateral infarct making microembolization as the most likely cause.[3] The diagnosis of CSS can be assumed once all other embolic sources have been excluded. The risk factors for CSS include advanced age, smoking, hypertension, hypercholesterolemia, diabetes, and obesity. A history of stroke at the time of diagnosis is associated with a high incidence of subsequent stroke and mortality whereas transient ischemic attack does not help with the prediction of outcome. Endarterectomy of the ipsilateral ICA has been the mainstay of treatment however it remains controversial. In a large study, surgical treatment was not found superior compared with conservative medical treatment.[4] In a recent study, anticoagulation with warfarin has also been used.[5] The endovascular stenting is an emerging modality for managing the patient with CSS.[6] Further large prospective studies are warranted for effective treatment of this rare disease.

References


Figure 1: CT angiography with contrast neck showing Left Internal Carotid Artery Stump (green arrow).

Figure 2: CT angiography with contrast neck showing Right Internal Carotid Artery narrowing (arrow red).