

Vertebral Artery Occlusion Causing Facial Colliculus and Opalski Stroke Syndromes Simultaneously

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Abstract: Brainstem stroke syndromes refer to a group of vascular disorders that occur secondary to occlusion of small perforating arteries of the posterior circulation. Although such syndromes result in a characteristic clinical picture, they are challenging to localize. In practice, such syndromes are rarely seen in their pure form, and the clinical consequences of a given vascular lesion are probably less predictable than for arteries in the anterior circulation. If 2 or more brainstem strokes happen simultaneously, localization becomes much harder. In this article, we present a case with 2 embolic strokes that occurred simultaneously because of left vertebral artery occlusion. The first in the right facial colliculus causing right sixth and seventh cranial nerves' palsy. The second happened in the left lateral medulla extending below the pyramidal decussation causing a classic left lateral medullary syndrome with ipsilateral hemiparesis, which constitutes a left Opalski stroke syndrome. To our knowledge, this is the first reported case of these 2 brainstem stroke syndromes happening simultaneously.

Key Words: facial colliculus syndrome, Opalski stroke syndrome, vertebral artery occlusion, stroke

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Brainstem stroke syndromes refer to a group of vascular disorders that occur secondary to occlusion of small perforating arteries of the posterior circulation. Although such syndromes result in a characteristic clinical picture, they are challenging to localize.¹ In practice, such syndromes are rarely seen in their pure form, and the clinical consequences of a given vascular lesion are probably less predictable than for arteries in the anterior circulation. If 2 or more brainstem strokes happen simultaneously, localization becomes much harder.² In this article, we present a case with right facial colliculus and left Opalski stroke syndromes happening simultaneously and causing neurological deficits with very interesting localizations. The 2 embolic strokes occurred simultaneously because of left vertebral artery occlusion.

CASE REPORT

A 62-year-old man with a history of diabetes and hypertension presented to a community hospital with sudden onset neck pain, diplopia, swallowing difficulty, and slurred speech. He reported a mild headache as well. His initial computed tomography (CT) of the head ruled out

intracranial hemorrhage and did not show acute ischemic changes. He was transferred to our center a day after. He had normal general and cardiovascular examination. On neurological examination, he was alert, dysarthric, with left Horner syndrome, reduced sensation in the left side of the face, right sixth and nuclear seventh cranial nerves' palsy, and absent gag reflex. Motor examination showed left hemiparesis. Sensory examination revealed reduced sensation to pain and temperature in the right arm and leg. He also had dysmetria in the right arm and leg. His CT angiogram showed left vertebral artery dissection (Fig. 1). Magnetic resonance imaging of the brain showed restriction diffusion and T2/FLAIR signal changes in the right facial colliculus and left lateral medulla extending caudal to the pyramidal decussation (Figs. 2, 3). The patient was started on anti-ischemic measures including antiplatelet (clopidogrel) and cholesterol-lowering agent (atorvastatin). In addition, the patient was started on both physical and occupational therapy. Swallowing team assessment showed gradual improvement of his bulbar functions. The patient recovered completely and was discharged home after 1 week of hospitalization.

DISCUSSION

Dissection of the vertebral artery is a common cause of stroke in middle-aged and young patients, with an incidence of 10% to 25%. This condition is rare in old patients and represents only 2% of all ischemic strokes among all ages. The etiology is either idiopathic or traumatic (coughing, vomiting, blunt trauma, chiropractic procedures, etc.). Patients with connective tissue disorders are at an increased risk. It presents with vague signs and symptoms, which make the diagnosis challenging in some cases.³ Neuroimaging including either magnetic resonance angiography or CT angiogram can identify occluded vertebral artery or mural thrombus and may also demonstrate posterior fossa ischemia or subarachnoid hemorrhage. Vertebral artery occlusion results in ischemia in the medulla and/or cerebellum if the occlusion is proximal near the origin of the vertebral artery (extracranial). Ischemia in the middle and distal vertebrobasilar artery result in locked-in syndrome and top of the basilar syndrome, respectively, with catastrophic consequences. In our case, although not confirmed radiologically, the most likely diagnosis is dissection of the proximal part of the vertebral artery with subsequent occlusion. The presence of sudden onset neck pain is in favor of this diagnosis. Management is usually medical with anticoagulation or antiplatelet therapy. However, endovascular or surgical treatment is reserved for patients with concomitant complications or unsuccessful medical therapy.⁴

Lateral medullary infarction, also called Wallenberg syndrome, is a common vertebrobasilar vascular syndrome. However, ipsilateral hemiparesis as part of lateral medullary infarction is rare (known as Opalski syndrome).⁵ The first description of Opalski syndrome was in 1946 in 2 patients who were reported by Opalski.⁶ They presented with ipsilateral facial loss of pain and temperature sensation, hemiplegia, Horner syndrome, ataxia, and contralateral loss of pain and temperature sensation in the limbs. Reported causes of this syndrome include vertebral artery occlusion, stenosis, and/or dissection.⁷

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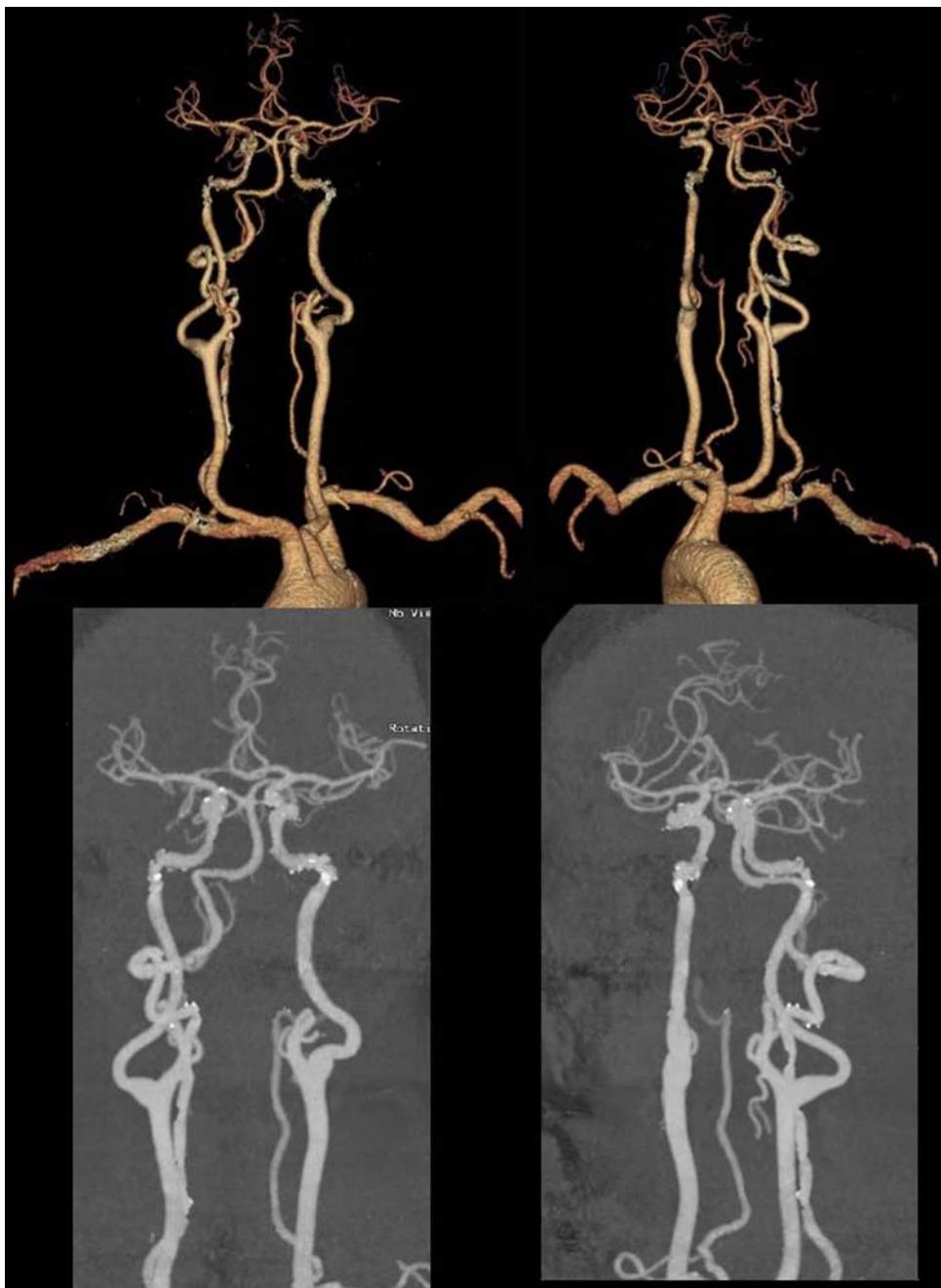


FIGURE 1. Computed tomography angiogram showing abrupt occlusion of the distal V2 and V3 segments of the left vertebral artery suggestive of dissection.

The cause of hemiparesis in Opalski syndrome remains controversial. Opalski attributed it to an extension of the infarct to involve the corticospinal fibers caudal to the pyramidal decussation.⁶ Subsequent studies showed that lesions that cause this syndrome are located caudal to the lesions in Wallenberg syndrome and involve the corticospinal fibers after the decussation. These arteries branch from the vertebral artery, which is considered the most common artery involved in Wallenberg syndrome.⁸

The facial colliculus is an elevated area occupying the dorsal pons on the floor of the fourth ventricle. It is formed by

fibers from the motor nucleus of the seventh cranial nerve as they loop over the nucleus of the sixth nerve.⁹ Facial colliculus syndrome is a group of neurological signs caused by a lesion at the facial colliculus involving the sixth and seventh cranial nerve along with fibers at the genu medial longitudinal fasciculus. The lesion will result in ipsilateral facial paralysis and ipsilateral unopposed eye medial deviation. The cause is variable depending on the age. In the young, causes include tumors, demyelination (eg, multiple sclerosis), and viral infection (eg, rhombencephalitis). In older people, the cause is typically vascular (eg, stroke).¹⁰

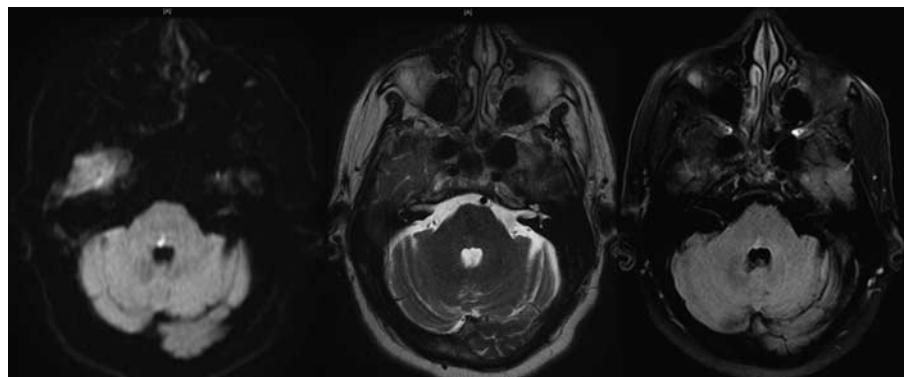


FIGURE 2. Magnetic resonance imaging of the brain showing restricted diffusion in the right facial colliculus.

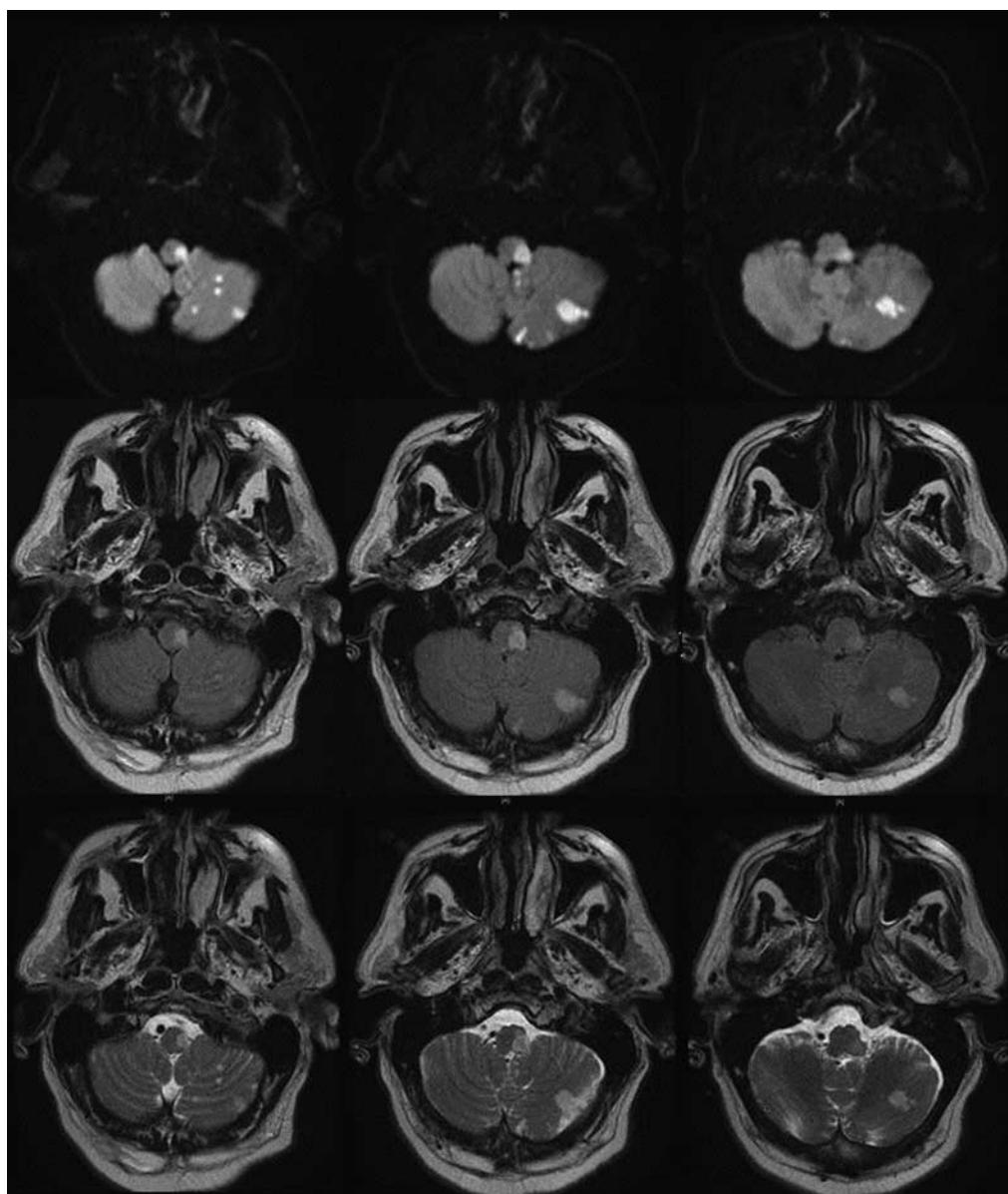


FIGURE 3. Magnetic resonance imaging of the brain showing images at the level of the lower medulla below the pyramidal decussation showing the restricted diffusion and increased T2 signal changes, respectively.

Facial colliculus syndrome typically presents with lower motor neuron seventh cranial nerve palsy proximal to geniculate ganglion. Therefore, it includes loss of sensation of taste in the anterior two thirds of the tongue and hyperacusis, horizontal conjugate gaze palsy, and diplopia. The facial palsy is a result of interruption of the ipsilateral facial nerve fibers at the genu as they arch behind the sixth cranial nerve nucleus. The conjugate gaze palsy is a result of the involvement of innervation of the ipsilateral sixth cranial nerve to lateral rectus, in addition to the interneurons projecting into the medial longitudinal fasciculus that innervates the contralateral medial rectus. Magnetic resonance imaging is the modality of choice, as it is able to detect a small focus of high signal in facial colliculus at the floor of the fourth ventricle.^{9,10}

CONCLUSIONS

Brainstem stroke syndromes are challenging to localize. In our case, 2 embolic strokes happened simultaneously. The first in the right facial colliculus causing the right sixth and seventh cranial nerves' palsy. The second happened in the left lateral medulla extending below the pyramidal decussation causing a classic left lateral medullary syndrome with ipsilateral hemiparesis, which constitutes a left Opalski stroke syndrome. To our knowledge, this is the first reported case of these 2 brainstem stroke syndromes happening simultaneously.

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