

Opalski Syndrome Due to Left Vertebral Artery Dissection

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ABSTRACT

There are a variety of stroke syndromes with often overlapping clinical presentations. When ipsilateral hemiplegia is associated with symptoms of a lateral medullary syndrome, it corresponds to the submedullary syndrome of Opalski. A 32-year-old woman presented with sudden onset neck pain, headache, vertigo, and vomiting. Her clinical status gradually deteriorated. Computed tomography angiography showed signs of critical stenosis of the V3 segment of the left vertebral artery. Magnetic resonance angiography displayed restricted diffusion in the posterolateral portion of the bulb and in the left cerebellar hemisphere. Opalski syndrome is a rare condition that could develop in patients with Wallenberg 'plus' syndrome, and neuroimaging studies to identify arterial disease and brain stem lesions should be performed.

Keywords

Stroke, Emergency department, Opalski syndrome.

Introduction

Opalski syndrome is a rare variant of Wallenberg's syndrome, characterized by hemiparesis and displaying the typical findings of lateral spinal infarction. This syndrome, first described by Opalski in 1946 [1], is a very rare brain stem syndrome. Patients present with ipsilateral hypoesthesia, Horner syndrome, hemiparesis, hemiataxia, and hypoesthesia on the opposite side of the body. We report an admitted case of lateral medullary infarction and ipsilateral hemiparesis that was diagnosed as Opalski syndrome.

Case Report

A 32-year-old woman with a history of hypertension, diabetes, smoking, and obesity, was admitted to a community hospital with sudden onset neck pain, headache, vertigo, and vomiting. She was transferred to our center after two days. General and cardiovascular examinations findings were normal. On neurological examination, she was alert and presented with left Horner syndrome. Motor examinations showed left hemiparesis (muscle strength of 2/5).

Sensory examinations revealed left reduced facial sensations to pain and temperature that was crossed in the body, affecting the right limbs, and dysesthesias in her left hemibody. She was ataxic on her left side. Nystagmus was evoked by looking left. Reflexes and proprioception were normal. Babinski reflex was negative.

Laboratory test, chest radiography, and electrocardiography results were normal. Computed tomography angiography (Figure 1) showed signs of critical stenosis of the V3 segment of the left vertebral artery, immediately after its emergence from the C2 vertebral foramen. Magnetic resonance angiography displayed restricted diffusion in the posterolateral portion of the bulb and in the left cerebellar hemisphere, and a reduction of normal void flow from the intradural segment of the left vertebral artery with apparent hypersignal at T1 compatible with vertebral artery dissection.

The patient worsened in the emergency department with drowsiness, dysphagia, broncho-aspiration, and hypoxemia. A rapid intubation procedure was performed using propofol and rocuronium to protect her airway.



Figure 1: A computed tomography angiogram with critical stenosis of the V3 segment of the left vertebral artery.

Anticoagulation with heparin was not prescribed due to a risk of hemorrhagic transformation to prevent a cerebellar infarction. The patient received 300 mg of aspirin.

The patient presented clinical improvement on the fourth day after her stroke. She was extubated without complications. During the next week, the patient almost completely recovered from her weakness (National Institutes of Health Stroke Scale score of 1) and was discharged on the 10th day.

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 older 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 [2].

Neuroimaging, including either magnetic resonance angiography or computed tomography angiograms, can identify occluded vertebral arteries or mural thrombi and may also demonstrate posterior fossa ischemia or a 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 results in ‘locked-in syndrome’ and ‘top of the basilar syndrome’, respectively, with catastrophic consequences [2]. In our case, the most likely diagnosis was dissection of the proximal part of the vertebral artery. The presence of sudden onset neck

pain favors 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 therapies [3].

The cause of hemiparesis in Opalski syndrome remains controversial. In his original description, Opalski justified motor impairment resulting from an extension of the ischemia from the lateral medulla to the upper cervical cord involving corticospinal fibers caudal to pyramidal decussation. He also considered that the ischemia was related to occlusion of the posterior spinal artery. Subsequently, other authors have reported cases related to vertebral artery occlusion and dissection [4,5].

This case provides additional information about the anatomical basis of ipsilateral hemiparesis in the lateral medullary infarction that is still not well understood. As suggested by Opalski [1] and Garcia-Garcia [5], the area of the spinal cord involved could be considered a border between the anterior and posterior spinal arteries and between the vertebral and spinal blood supply. Failure of regional perfusion in this area may be the result of a hemodynamic mechanism due to stenosis or occlusion of the vertebral artery. Although we cannot determine the exact mechanism, the existence of a dissection suggests a compromise of the penetrating arteries as the cause of spinal and medullary ischemia in our patient.

In addition, our patient presented a probable and expected complication not described in the literature, which was dysphagia with airway protection difficulty and the need for orotracheal intubation. Finally, it has been demonstrated that, in this case, treatment with platelet anti-aggregation is safe.

Conclusion

Lateral medullary infarction, also called Wallenberg syndrome, is a common vertebrobasilar vascular syndrome. However, ipsilateral hemiparesis as part of a lateral medullary infarction is rare (known as Opalski syndrome) [6].

First described in 1946 [1], Opalski syndrome is considered a variant of a lateral medulla oblongata infarct (i.e., Wallenberg syndrome) with ipsilateral facial loss of pain and temperature sensations, hemiplegia, Horner syndrome, ataxia, and contralateral loss of pain and temperature sensation in the limbs.

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