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Clinical case

Opalski's syndrome: a case report and review of the literature

Syndrome Opalski : à propos d'un cas revue de la littérature

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Résumé

Le syndrome d'Opalski est un syndrome du tronc cérébral, rarement décrit dans la littérature. Elle est une variante du syndrome de Wallenberg associée à une hémiplégie.

Sa présentation clinique est caractéristique et l'arrivée de l'IRM cérébrale a permis de confirmer le substratum anatomique. Nous rapportons le cas clinique d'un homme de 57 ans, qui va présenter un syndrome de Wallenberg droit associé à une hémiparésie droite, l'imagerie par résonance magnétique (IRM) cérébrale montrant un infarctus latérobulbaire avec thrombus de l'artère cérébelleuse postéro-inferieur droite.

Mots-clés : syndrome Opalski, IRM cérébrale, Gabon.

Abstract

Opalski syndrome is a brainstem syndrome, rarely described in the literature. It is a variant of Wallenberg syndrome associated with hemiplegia. Its clinical presentation is characteristic and the arrival of cerebral MRI has confirmed the anatomical substratum. We report the clinical case of a 57-year-old man who presented with right Wallenberg syndrome associated with right hemiparesis. Cerebral magnetic resonance imaging (MRI) showed a laterobulbar infarction with thrombus of the right posteroinferior cerebellar artery.

Keywords: Opalski syndrome, brain MRI, Gabon.

Introduction

Opalski is the eponym of Opalski syndrome discovered in 1946. It is a variant of Wallenberg syndrome characterized by a hemiparesis homolateral to the lesion respecting the face associated with a lateral bulbar syndrome [1]. It is a rare vascular syndrome of the brain stem secondary to a laterobulbar infarction located below the pyramidal decussation. We report the clinical case of a 57-year-old man, chronically hypertensive for 10 years, smoker at 20 pack

years, who presented a right Wallenberg syndrome associated with a right hemiparesis.

Clinical case

We report the clinical case of a 57-year-old man, hypertensive for 10 years under treatment, who smoked 20 packs of cigarettes on average per day for 40 years. He will suddenly present phonation and swallowing disorders. He will describe thereafter headaches in helmet without photophobia nor phonophobia nor vomiting but dizziness. The clinical examination revealed a Claude Bernard Horner on the right side, swallowing disorders, a nasal tract, a hypoesthesia of the right face, a monoparesis of the right upper limb with a muscle strength rated at 4/5, a Babinski sign and a hemihypoesthesia of the left upper limb. The patient was admitted to the neurovascular unit outside the thrombolysis period. The clinical picture was suggestive of an Opalski syndrome confirmed on brain imaging. Cerebral CT was normal and we completed the study with a cerebral MRI showing a diffusion hypersignal in ADC restriction in the lateral part of the bulb (Figure 1) evoking a laterobulbar infarction with the presence of a thrombus in the right posteroinferior cerebellar artery visible in T2* (Figure 2).

The biological workup was normal, HIV and syphilitic serology were negative. The cardiac workup showed atrial extrasystoles, the cardiac ultrasound was normal, the Doppler ultrasound of the neck vessels showed atheroma without significant stenosis at the vertebrobasilar level. The patient was put on antiplatelet medication at 160 mg and a beta-blocker, physical therapy and speech therapy, the evolution was favorable after 2 weeks, the National Institutes of Health Stroke Scale (NIHSS) went from 2 to 0.

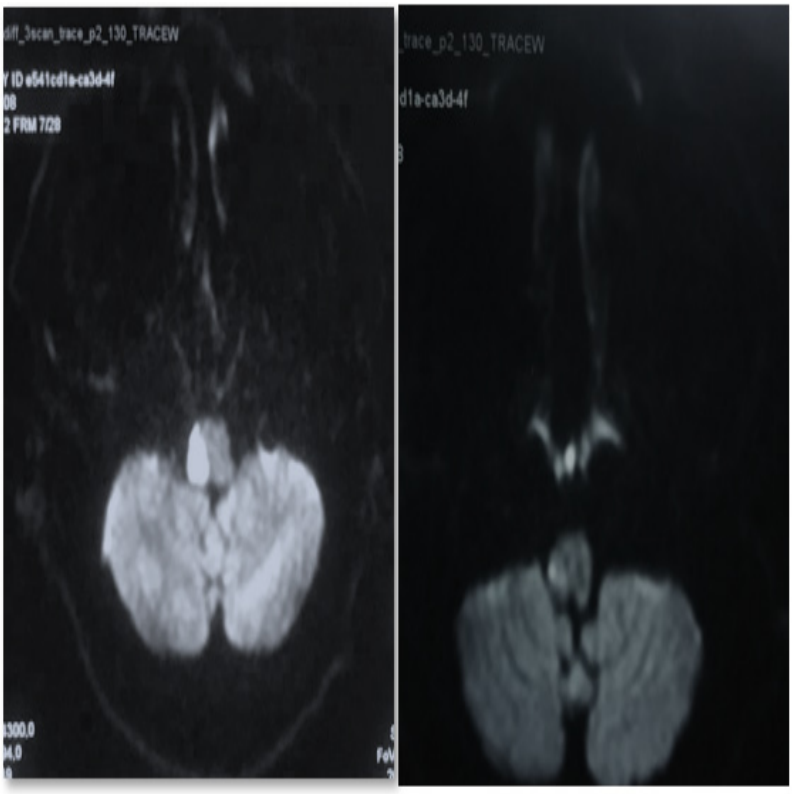


Figure 1 : right lateral bulbar infarction (diffusion sequence)

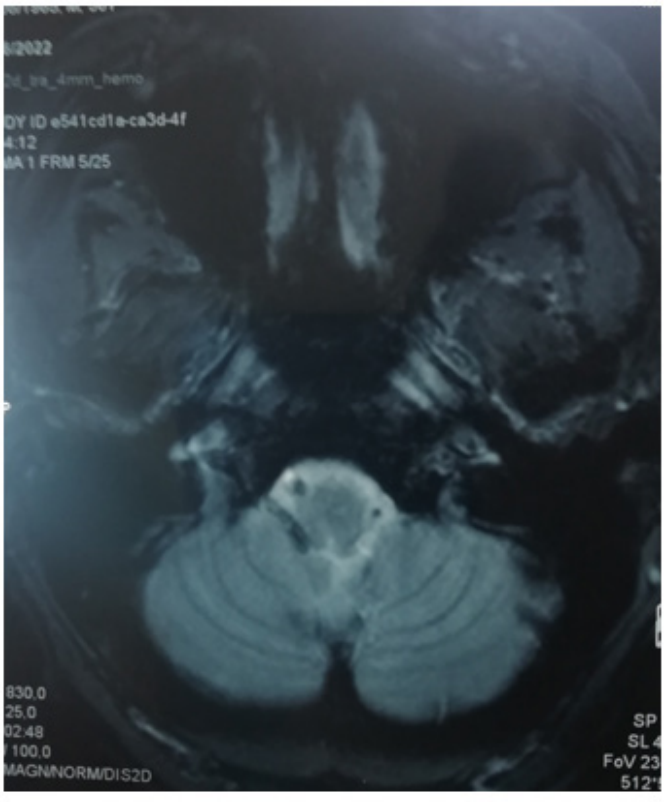


Figure 2: Thrombus in the right posteroinferior cerebellar artery (T2* sequence)

Discussion

Bulbar infarcts account for 7% of all strokes in the brainstem [2; 3]. Lateral bulbar infarcts are more frequent, i.e. 5 times more frequent than in the medial territory [4]. In the lateral bulbar infarcts, we can mention as vascular syndrome: Wallenberg's syndrome which is the most frequent and Opalski's syndrome which is little described in the literature. Opalski was the first to describe this variant of lateral bulbar syndrome with ipsilateral hemiparesis in 1946 under the name of partial posterior spinal artery syndrome [1]. It is a relatively rare stroke with a slight male predominance [5].

The classic presentation is thermoalgesic sensory disturbances of the brainstem and extremities contralateral to the infarction and numbness of the face ipsilateral to the infarction. Other signs of the syndrome include vertigo, nystagmus, dysphagia, homolateral cerebellar signs, and Claude Bernard Horner syndrome. Studies suggest that hemiparesis occurs in less than 7% of cases in laterobulbar infarction [4]. The cause of hemiparesis remains controversial in Opalski syndrome.

Opalski in his description attributed it to the extension of the ischemia from the lateral bulb to the superior cervical medulla involving the corticospinal fibers under the pyramidal decussation.

He also considered that the ischemia was due to additional damage to the posterior spinal artery. However, studies suggest the involvement of the perforating arteries that supply the pyramidal fibers after decussation [6]. The perforating arteries of the lateral part of the bulb are supplied by the posteroinferior cerebellar artery at the bottom, the vertebral artery in the middle, and at the top by a part of the basilar artery and the anteroinferior cerebellar artery. These arteries penetrate the bulb in the lateral fossa of the bulb [7]. Other authors evoke a failure of the regional perfusion of the frontal zone of the spinal cord which is located between the anterior and posterior spinal arteries involving the pyramidal fibers. Some authors question the existence of hemiparesis because they attribute the weakness to a spinocerebellar hypotonic syndrome rather than to damage of the pyramidal tract [5]. In our clinical picture the hemiparesis could be the consequence of a pyramidal damage under the decussation (Figure 2).

Bulbar infarcts most often result from hypoperfusion secondary to intracranial vertebral artery stenoses (73%) and arterial dissections (26%), they are rarely caused by cardiac emboli [4]. The presence of thrombus in the posteroinferior cerebellar artery visible on T2* echogradient may suggest either a cardioembolic cause or thrombus from an ulcerated atheromatous plaque. The prognosis of strokes in the brain stem is varied because it is the control center for several vital functions, such as breathing and cardiac regulation.

Thus, despite the small size of the lesions, they often have a poor prognosis and require monitoring in a neurovascular unit, as in our case. The treatment depends on the etiology.

Conclusion

This case illustrates a rare entity of brainstem syndrome known as Opalski's syndrome with clinical originality and characteristic imaging. It shows that a careful neurological examination is sufficient to make the diagnosis, pending further investigations.

Author contributions

All authors received in the development of this manuscript. All authors have read and approved the final version of the manuscript.

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