Wallenberg Syndrome: A Case Report
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INTRODUCTION

Wallenberg syndrome or lateral bulbar syndrome or lateral bulbar infarction was first described by Adolf Wallenberg, a German neurologist. It is an alter neurological syndrome resulting from posterolateral involvement of the medulla oblongata. It is initially manifested by a great inaugural vertigo [1].

OBSERVATION

This is a 65-year-old patient, type II diabetic under insulin for 30 years. He presented with an atrial flutter that was treated by radiofrequency ablation and subsequently put on anticoagulants.

He consulted us following the development of a right hemiplegia with vertigo, dysphagia, dysphonia and facial hemiparesis. He was referred to our department for a brain scan at H48 of the beginning of the symptomatology which was normal. A magnetic resonance imaging was performed at D4 which showed:

Right lateral hyper signal of the bulb on T2, FLAIR and diffusion sequences in discrete T1 hyper signal.

Time-of-flight 3D TOF and gadolinium-injected AngioMR sequences showed distal occlusion of the right vertebral artery upstream of the PICA origin.
DISCUSSION

Anatomically, the brainstem is composed of the midbrain, the pontine, and the bulb.

These three levels are the seat of the cranial nerve nuclei that have connections with the cerebral cortex, thalamus and spinal cord.

The trunk is irrigated by branches of the vertebral artery: the posterior communicating artery, the superior cerebellar artery, the posterior inferior cerebellar artery, and the anteroinferior cerebellar artery [2].

Brainstem infarcts may be staged and in association with downstream hemispheric infarcts. This results in alternating syndromes that are defined by:
- Homolateral cranial nerve damage
- An attack on a sensitive or motor pathway, contralateral to the lesion

Wallenberg’s syndrome or alternating sensory syndrome results from lateralized ischemic damage to the medulla oblongata.

The initial presentation is dominated by vertigo, headache and balance disorder [3].

Secondarily, it is manifested by:
- On the side homolateral to the lesion: mixed nerve involvement (dysphonia, dysphagia); VIII involvement (vestibular syndrome with nystagmus); V involvement (hypoesthesia of the hemiface), Claude Bernard Horner syndrome.
- On the side contralateral to the lesion: thermo-algesic anesthesia of the hemifield.

MRI is the examination of choice for the diagnosis of lateral bulbar infarction. It detects the lesion regardless of its size. On conventional sequences, the ischemia appears in hyposignalT1 from the first hours, a hyposignalT2 and FLAIR is noted in 80% of cases at 24H. The exploration of the brain stem is better with the FLAIR sequence.

Diffusion allows visualization of lesions before the 6th hour while the T2 sequence is still normal with a specificity exceeding 95%.

Angiographic sequences allow the detection of occlusions and stenoses even of small vessels [4].

The CT scan fails most of the time in the diagnosis of Wallenberg syndrome. It is performed in order to eliminate a hemorrhage in an emergency context [5].

CONCLUSION

The brainstem is a very complex anatomical structure. It can be the site of minimal infarction with spectacular clinical translation [6].

Imaging, particularly magnetic resonance imaging, has revolutionized the diagnosis and management of these lesions.
REFERENCES