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Short Communication
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Artery of Percheron Infarct

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Keywords

Hyperintensity; Artery of Percheron's territory; Ischemic event

Clinical Information

Patient information

Age: 72 years

Gender: Female

Clinical history and presentation

A 72-years-old female, complaining of diplopia, lateral deviation of the left eye, left ptosis for the last 3 days, and altered consciousness for the last day. There was no history of fever. She was a 40 pack year's ex-smoker.

Physical exam

At the emergency department, she presented with impaired consciousness, dysarthria, bilateral paralysis of the third cranial nerve (oculomotor nerve), bilateral ptosis, and symmetric grade 4 strength.

Other diagnostic testing

Normal blood and urine tests.

Diagnosis

Final diagnosis

Artery of Percheron Infarct: More than half of the cases have a hyperintensity region in the shape of a "V" involving the medial surfaces of the cerebral peduncles and the rostral midbrain. MRI with DWI sequence is the method of choice and shows diffusion restriction in the affected areas. T2-weighted/FLAIR sequences show hyperintensity in the medial thalamus.

Differential diagnoses

Basilar artery thrombosis: The most important differential diagnosis is thrombosis of the basilar artery. The most affected region is the rostral midbrain, occipital lobes, superior vermis, and thalamus. In contrast to deep cerebral venous thrombosis, which is also a differential diagnosis, the posterior region of the internal capsule, the entire thalamus, and the basal ganglia are affected. On the MRI T2 sequences, we find the "blooming" artifact in the deep cerebral veins.

Acute Disseminated Encephalomyelitis (ADEM): Acute disseminated encephalomyelitis: A disease often affecting the thalamus bilaterally and causing swelling. It is often associated with injury to the white matter, optic nerves, and spinal cord. Shows hyperintense T2 enhancement on MRI.

Discussion

Clinical presentation and general epidemiology

The AOP exists in up to 33% of the population [1], and studies have indicated that AOP occlusion accounts for up to 2% of all ischemic strokes, and 4% to 18% of all thalamic strokes [1,2].

The thalamopeduncular syndrome is among the commonest presentations of AoP obstruction, and its clinical manifestations include cervical dystonia, abnormal eye movements, ataxia, cognitive impairment, and behavioral disorders [1,2].

Imaging features

An early brain non enhanced computed tomography usually does show abnormalities. Hypoattenuating areas in both thalamus that extend into the central area of the midbrain may

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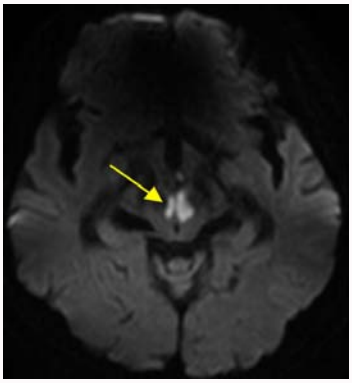

Figure 1: Axial DWI sequence (b1000) showed restricted diffusion in the paramedian thalamus and central midbrain (yellow arrow). The ADC map showed a low apparent diffusion coefficient (image not shown).

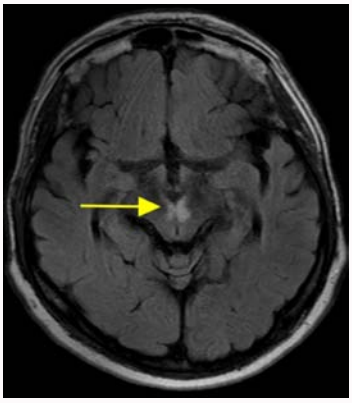

Figure 2: Axial FLAIR sequence shows hyperintensity in the paramedian thalamus and central midbrain (yellow arrow).

appear in later stages [4] (Figure 1).

The MRI with DWI sequence is the exam of choice and reveals restriction to diffusion in the affected areas. The T2/FLAIR-weighted sequences show a hyperintensity in the medial thalamus. In just over half of the cases, a region with a "V" shaped hyperintensity affects the medial surfaces of the cerebral peduncles and the rostral midbrain [2,4].

The differential diagnosis of bithalamic lesions is broad [2], potentially mimicking non convulsive status epilepticus, subarachnoid hemorrhage, metabolic or toxic encephalopathy, and encephalitis [3] (Figure 2). Basilar artery thrombosis should be included, however, it is more extensive and involves part or all of the rostral midbrain, occipital lobes, upper vermis, and thalamus [3,4] (Figure 3). Deep

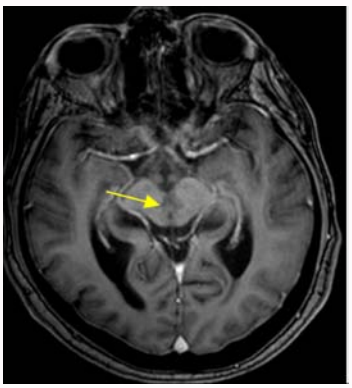

Figure 3: Axial gadolinium-enhanced T1 sequence shows no contrast enhancement. The ischemic area shows hypointensity (yellow arrow).

cerebral venous thrombosis is also a differential diagnosis, however, it usually involves the basal ganglia [3,4], posterior limb of the internal capsule, and the entire thalamus, with T2*- weighted MRI sequences (GRE, SWI) showing "blooming" artifact in the deep cerebral veins [4].

Prognosis, treatment or therapeutic options

Effective first-line treatment options for acute AOP occlusions are intravenous heparin and thrombolysis with tissue plasminogen activator, followed by the use of long-term anticoagulants. Non-permanent cases, without midbrain involvement, can be treated with rehabilitation and ongoing monitoring. Analysis of treatment options for AOP occlusion is important [5].

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