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Case Report

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ARTERY OF PERCHERON INFARCT- A CASE REPORT

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ABSTRACT

Bilateral thalamic infarcts are rare occurrences. The typical clinical features of occlusion of the Artery of Percheron include altered level of consciousness, behavioral changes, memory impairment, and vertical gaze palsy. Herein we report a 44 year old lady who presented with Artery of Percheron infarction.

KEYWORDS: Artery of Percheron, Thalamic infarct, Vertical gaze palsy.

INTRODUCTION

Thalamus is pathway of connection between the cerebral cortex and the midbrain. It is responsible for regulating consciousness, and sleep. The vascular anatomy of the thalamus is usually from dual arterial contribution from the anterior and posterior cerebral circulations. Bilateral thalamic infarcts are rare occurrences.

CASE REPORT

A 44 year old lady, with systemic hypertension, diabetes mellitus and dyslipidemia, presented with sudden onset of increased sleepiness and decreased alertness. There was no history of loss of consciousness, involuntary movements, bowel or bladder incontinence, motor weakness or cranial nerve symptoms. On examination, patient had (BMI 27.34kg/m_2, pulse rate 90/minute, BP 170/90 mm of Hg.She was drowsy and had vertical gaze palsy. Pupils were equal in size bilaterally and reacting normally to light. She did not have any motor or sensory deficits or ataxia. Investigation showed Hemoglobin 12.8g/dl, Total leucocyte count 9600/mm3, Platelet count 3.2 lakhs/mm3, fasting blood sugar 150 mg/dl, blood urea 33mg/dL, serum creatinine 1 mg/dl, serum cholesterol 242 mg/dl, serum triglycerides 202mg/dl, HDL 32 mg/dl, LDL 170 mg/dl. MRI brain showed bilateral asymmetrical ill defined T2 hyper intensity involving ventromedial thalamus and adjacent areas, centre of the lesions show T2 hypo intensity with peripheral rim enhancement. MR angiography was normal. CSF study did not reveal any abnormality. She was managed conservatively with antiplatelets, antihypertensives, oral hypoglycemic agents and statins. She improved after around one week. Repeat MRI after one month showed a recent infarct left MCA territory involving left gangliocapsular region and corona radiata with focal gliosis in paramedian aspect of bilateral thalami extending to meso-diencephalic junction – suggestive of Artery of Percheron territory old infarct.

DISCUSSION

AOP infarcts account for .1 % to 2 % of all ischemic strokes[1] and from 4 % to 18 % of all thalamic strokes. The blood supply of the thalamus is by vessels originating from the posterior communicating artery and from the P1 and P2 segments of the posterior cerebral artery (PCA). With regard to the blood supply, the thalamus can be divided into 4 territories: anterior, paramedian, inferolateral, and posterior.[1] The anterior territory is supplied by the polar or thalamotuberal arteries arising from the posterior communicating artery. The paramedian territory is usually supplied by the paramedian (or thalamoperforating) arteries, which arise from the P1 segment of the PCA. The inferolateral territory is supplied by the thalamogeniculate arteries, which arise from the P2 segment of the PCA. The posterior territory is supplied by the posterior choroidal arteries, which arise from the P2 segment of the PCA. There are many variations in the blood supply of the paramedian territory of the thalamus. The most common occurrence is the Type I pattern, where the supply is from each P1 segment. In the Type III variant there is a single artery which bridges the two P1 segments and an arcade of perforating arteries supplying the paramedian territories of both the thalami arise from this single artery. Artery of Percheron, or the Type II variant (which is seen in about one third of humans) arises from one P1 segment and splits to supply the both the thalami and rostral midbrain. The rostral mid brain is supplied by rubral arteries which may arise either from the P1 segment of the PCA or may share a common origin with the paramedian arteries.

The typical clinical features of occlusion of the Artery of Percheron include altered level of consciousness, behavioural changes, memory impairment, and vertical gaze palsy. This patient also presented with sudden onset of sleepiness and vertical gaze palsy. The altered mental

status, behavioral changes and memory defects are explained on the basis of interruption of the connections of the thalamic nuclei with the anterior, orbitofrontal and medial prefrontal cortices.Vetrical gaze palsies occur due ischemia of the rostral interstitial nucleus of the medial longitudinal fasciculus (MLF).[2] The involvement of the rostral midbrain is a variable feature of AOP infarct. Vertical gaze palsy can also occur without midbrain affection. This is explained on the basis of the disruption of the fibres which pass through the thalamus on their way to the riMLF.The involvement of the rostral mid brain is responsible for the findings like hemiplegia,cerebellar ataxia, involuntary movements and third cranial nerve palsy which may sometimes be seen in AOP infarct. The prognosis is generally good. The neurological deficits usually improve. Rarely there may be persistent visual field defects. In this case on review after two months she did not have any residual neurological deficits.

Early diagnosis of AOP infarct is best made by a diffusion-weighted imaging (DWI) sequence using MRI.The occluded Artery of Percheron is not usually visible on MR angiography[3] as in this case .A characteristic finding of AOP infarct is the V-shaped hyper intense signal intensity on axial FLAIR and DWI images along the pial surface of the midbrain in the interpeduncular fossa. This finding has been reported to have 67% sensitivity.[4]

The differential diagnosis for bilateral thalamic infarcts include the top of the basilar syndrome, deep cerebral venous thrombosis ,metabolic and toxic processes like Wernicke's encephalopathy, Wilson's disease, extrapontine myelinolysis, Creutzfeldt-Jakob disease, infection and neoplasm including bilateral thalamic glioma.[5]

The diagnosis of Artery of Percheron is likely to be missed because of the atypical features of stroke, that is decreased level of consciousness and absence of motor and sensory deficits. In patients with sudden onset of altered level of consciousness, presence of vertical gaze palsy is a clue to the diagnosis of this clinical syndrome, which can prompt earlier investigations and thrombolytic therapy if feasible.

CONCLUSION

We are presenting a case of bilateral thalamic infarct with the typical clinical features .AOP is likely to be missed unless the physicians keep a strong suspicion for this condition. The presence of the sudden onset of altered level of consciousness with vertical gaze palsy should prompt us to think of this possibility.

There is no conflict of interest. Consent form is available.

Legend of the figures MRI brain showing bilateral asymmetrical ill defined T2 hyper intensity involving ventromedial thalamus and adjacent areas, centre of the lesions showing T2 hypo intensity with peripheral rim enhancement.

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