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Case Report

Artery of percheron infarction: case reports and literature review

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ABSTRACT

The artery of Percheron is a rare anatomic variant supplying the thalamus and the rostral midbrain. Infarct in this territory results in a wide array of neurological signs and symptoms causing diagnostic dilemma and management issues. We describe the clinical presentations in three cases admitted and evaluated for neurological symptoms and diagnosed as artery of percheron infarct after brain imaging. In one patient, the etiology turned out to be infective while the other two patients had cerebrovascular accident secondary to dilated cardiomyopathy and hyper homcystinimea respectively. Artery of percheron infarction is a rare entity and should be considered in patients with altered sensorium and behavioral manifestations with associated eye abnormalities. MRI brain is the investigation of choice to detect this rare variant of thalamic circulation.

Keywords: Artery of percheron, Infarction, Thalamus

INTRODUCTION

The complexities of the vascular supply of thalamus were initially evaluated by Duret and Hillemand and later by Percheron.[1]

Artery of percheron (AOP) is a solitary trunk originating from one of the posterior cerebral arteries (PCA, P1 segment usually) to provide arterial supply to paramedian thalami and the rostral midbrain. This artery supplies the medial thalamic nuclei, interpeduncular nucleus, decussation of the superior cerebellar peduncles, medial part of red nucleus, third and fourth cranial nerve nuclei and anterior portion of the periaqueductal grey matter.

Hence, occlusion of artery of percheron causes bilateral paramedian thalamic infarction with or without affection of the midbrain.[2,3] We describe three cases of infarction in the territory of artery of percheron secondary to different etiologies.

CASE REPORT

Case 1

A 48-years-old gentleman was admitted with fever of 3 months duration. It was associated with headache and vomiting. There was worsening in sensorium with restlessness, disorientation and confusion for 4 days. Upon arrival, he was febrile and had a left sided ptosis with a semi dilated pupil and a positive kernig's sign and neck rigidity. Systemic examination and blood investigations, chest X-ray and ultrasound of the abdomen was normal. A contrast MRI brain was done that showed bilateral paramedian thalamic infarcts and rostral mid brain infarction suggesting AOP involvement (Figure 1A and B). A cerebrospinal fluid (CSF) examination showed 200 cells (80% lymphocytes and 20% neutrophils) with reduced sugar (19mg/dL) and raised proteins (242mg/dL). Antigen testing and india ink for Cryptococci was negative. A provisional diagnosis of tubercular meningitis with arteritis was made and was

confirmed on a positive mycobacterium growth indicator tube (MGIT) test for tuberculosis. He was started on anti-tubercular treatment with steroids. He showed improvement in sensorium and was discharged in a stable condition.

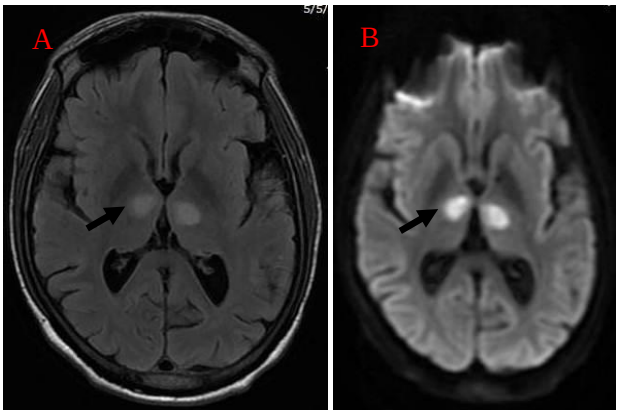

Figure 1: A and B- FLAIR and DWI showing B/L thalamic infarcts (arrows) in case 1.

Case 2

A 55-years-old lady had sudden onset giddiness and imbalance on walking of about 12 hours duration. She had progressive worsening in sensorium and was admitted in a drowsy state. On examination, she was drowsy, had muteness and hyper somnolence. There was a skew eye deviation with left hemiparesis.

A possibility of posterior circulation stroke was considered and MRI brain revealed B/L paramedian thalamic infarcts consistent with AOP involvement (Figure 2 A and B). MR angiogram of brain was unremarkable. Her ECHO cardiogram revealed dilated cardiomyopathy with a low ejection fraction of 28% and no evidence of clot or vegetations on ECHO testing. Anti-coagulation was started and she was discharged in a stable condition. While on regular follow up, her deficits have improved and she has mild left sided residual hemiparesis.

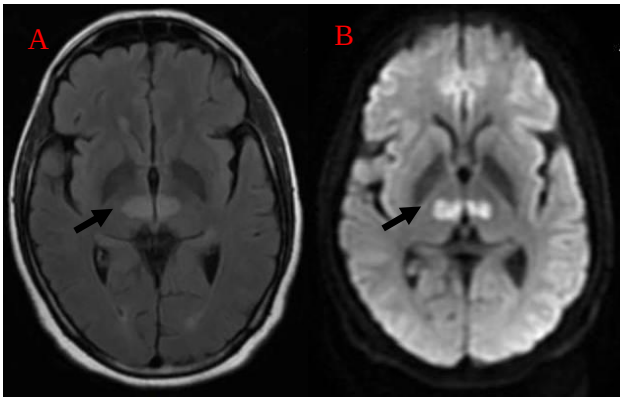

Figure 2: A and B- FLAIR and DWI showing B/L thalamic infarcts (arrows) in case 2.

Case 3

A 37-years-old lady came with sudden onset altered sensorium and right hemiparesis of 12 hours duration. She had right hemiplegia and left 3rd cranial nerve palsy on examination. MRI brain showed B/L thalamic paramedian and midbrain infarction consistent with infarction of AOP (Figure 3 A and B).

No abnormality could be seen on MR angiogram of brain and neck vessels. Anti-platelets were started. Stroke work up revealed high serum homocysteine levels (38.5micromol/L). Therapy for the same was started and she has shown signs of improvement after discharge.

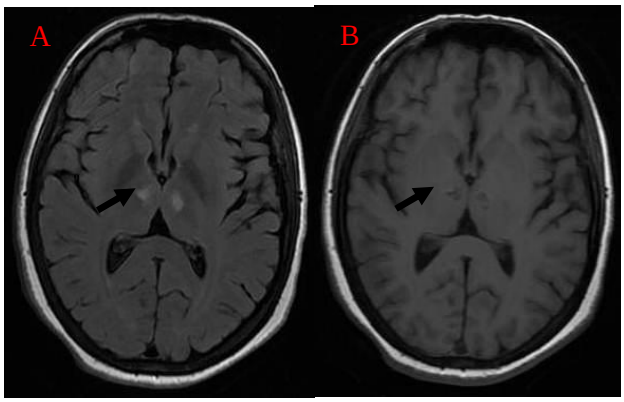

Figure 3: A and B- FLAIR and T1WI showing thalamic infarcts (arrows) in case 3.

DISCUSSION

All three cases had infarction in AOP territory though with different etiologies. Case 1 had arteritis secondary to tubercular meningitis; case 2 had an embolic infarction and case 3 a thrombotic infarct.

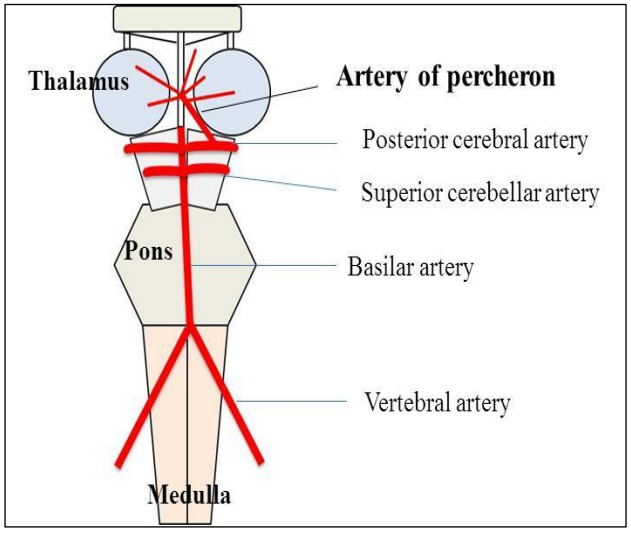

Figure 4: Artery of percheron originating from right posterior cerebral artery and supplying B/L paramedian thalami.

The paramedian thalami and midbrain are irrigated by two paramedian thalamic arteries stemming from each PCA. Percheron G, a French neurologist, described four variants of paramedian perforating arteries to the thalami. Of these, AOP is a single arterial trunk stemming off the P1 segment of one of the PCA and dividing to supply both thalami and the upper midbrain (Figure 4).[2]

AOP is a rare variant with Uz having noted only 1 AOP in 15 cadavers while Aaron et al observed AOP infarcts in 17 of 3589 patients with stroke (0.47%).[4,5] Various other series have reported the incidence of AOP infarcts to be 2% of all strokes to 4-18% of thalamic strokes.[6] In a large series of B/L thalamic infarcts by Lazarro et al, the commonest pattern of involvement was paramedian thalamic infarction with rostral midbrain infarct.[7]

The same pattern was observed in cases 1 and 3. The most common etiology of B/L thalamic infarction is cardio embolic source in young strokes < 45 years of age while cerebral atherosclerosis in older patients.[7,8] Kamasak et al reported two children with B/L thalamic infarcts after chicken pox and encephalitis.[9] Other infections that need to be considered in the differential diagnosis of AOP infarcts are tubercular meningoencephalitis, Japanese encephalitis, cerebral malaria, toxoplasmosis and west Nile virus encephalitis.[10] The most common manifestation of AOP infarcts is disturbance in the conscious levels.

Behavioral manifestations are common and brain stem affection causes eye abnormalities ranging from skew eye deviation to abnormalities in vertical eye movements and pupillary abnormalities, ptosis, and adduction deficits seen with rostral midbrain lesions.[6,8]

AOP infarcts are usually identified on MRI brain sequences that show characteristic B/L thalamic paramedian lesions with or without involvement of the mid brain. Lazarro et al described a distinct pattern of V-shaped hyperintensity on axial FLAIR and/or DWI sequences along the pial surface of the midbrain adjacent to the interpeduncular fossa in 67% of patients with midbrain involvement.[8]

The AOP is rarely visualized on conventional angiography. Prognosis after thalamic infarction is generally better compared with large vessel strokes both in children and adults. Persistence of cognitive and psychiatric manifestations after paramedian artery stroke and development of debilitating extra pyramidal symptoms has been reported.[7] Various studies report a mortality of about 12 % after AOP infarction.[6]

CONCLUSION

Sudden onset alteration in sensorium with behavioral manifestation and eye movement abnormalities are clues to artery of percheron infarction. MRI brain with diffusion sequences in the early hours is the investigation of choice for detecting AOP infarcts. Since embolism is the commonest cause of AOP infarcts, a detailed evaluation for cardiac or arterial source for embolism should be looked for.

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