

Artery of Percheron Infarction-MRI Imaging

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Abstract: The Artery of Percheron is an uncommon anatomic variation in which a single arterial trunk arises from the posterior cerebral artery (PCA) to supply both sides of the thalamus and midbrain. The incidence of AOP infarcts occurs in approximately 0.1 to 0.3 % of total ischemic stroke. A bilateral paramedian thalamic infarct is characterized by a triad of altered mental status, vertical gaze palsy, and memory impairment. These disorders usually occur with acute onset and may persist until death. Our cases come under the 2nd type of ischemic pattern by N.A.Lazzaro *et al.* The classical MRI imaging feature in AOP infarction is diffusion restriction in both paramedian regions on diffusion-weighted MRI. AOP occlusion can be life-threatening, so the knowledge about AOP and its occlusion is essential to make an accurate diagnosis and timely management.

Keywords: Artery, Percheron, Infarction.

INTRODUCTION

The Artery of Percheron (AOP) was first described by Gerard Percheron in 1973. The AOP is an uncommon anatomic variation in which a single arterial trunk arises from the posterior cerebral artery (PCA) to supply both sides of the thalamus and midbrain [1]. The incidence of AOP infarcts occurs in approximately 0.1 to 0.3 % of total ischemic stroke [2].

Early diagnosis of AOP infarction can be challenging as it is rare and early computed tomography (CT) or magnetic resonance imaging (MRI) may not be positive. Thus, it can be difficult to be differentiated from other neurological conditions like tumors and infections [3].

CASE REPORT

Case 1

A 55-year-old male with bilateral upper and lower limb weakness (right > left) and disoriented for two hours. The patient is a known case of hypertension and diabetes mellitus for the past 5 years and is on

irregular medications. On physical examination, Glasgow coma scale 8 and following initial vital signs: pulse – 58 beats per minute, respiratory rate of 15 breathes per minute and blood pressure 150/100 mmHg. Mild mydriasis seen in both eyes. Ocular movements showed vertical gaze palsy. No history of fever and meningeal signs. Laboratory tests were unremarkable. CT performed initially showed no early signs of ischemia and acute hemorrhage. MRI was performed 2 hours later revealed acute infarct in the paramedian location of both thalami (left < right) and shows a restriction on diffusion and low on ADC map (Figure 1).

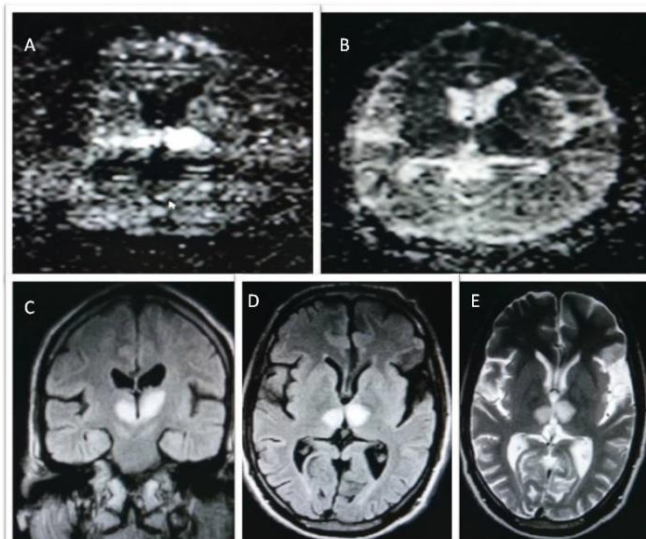


Fig-1: MRI axial section (A, B) shows a restriction on diffusion and low on ADC map, FLAIR (C, D) coronal, axial and T2 axial section (E) shows high signals on the bilateral paramedian area of the thalamus

Case 2

A 64-year-old female with disorientation and weakness of the right side of the face, bilateral upper and lower limbs for last 6 hours and referred to the radiology department for MRI study of the brain. Initial CT was performed elsewhere and no significant

abnormality was noted. The patient is a known case of hypertension. No history of fever and meningeal signs. Laboratory tests were unremarkable. MRI study of the brain revealed acute infarct in the paramedian location of both thalami (left < right) (figure 2).

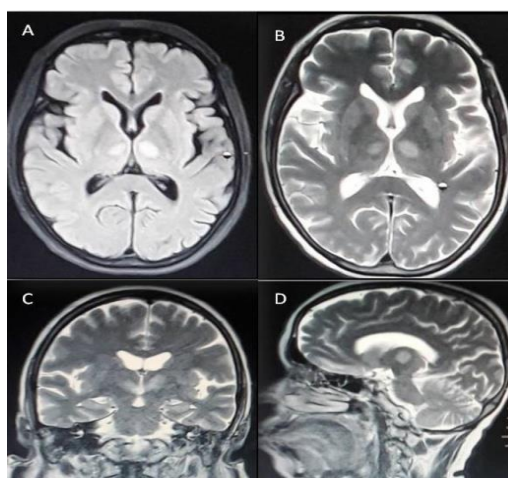


Fig-2: MRI axial section, FLAIR axial (A), T2 axial, coronal and sagittal section (B, C, D) shows high signals on the bilateral paramedian area of the thalamus

DISCUSSION

The thalamus is mostly supplied by multiple small branches from the posterior communicating artery (PCA) and P1 and P2 segments of the PCAs. Even though there are significant variation and overlap in thalamic vascular supply, it is categorized into 4 territories: anterior, paramedian, inferolateral, and posterior. Bilateral paramedian thalamic infarct is characterized by a triad of altered mental status, vertical gaze palsy, and memory impairment. These disorders usually occur with acute onset and may persist until death. Cases of complete recovery have also been documented. According to N. A. Lazzaro *et al.*

described 4 ischemic patterns of AOP infarction 1) Bilateral paramedian thalamic with midbrain (2) Bilateral paramedian thalamic without midbrain, 3) Bilateral paramedian thalamic with anterior thalamus and midbrain and 4) Bilateral paramedian thalamic with anterior thalamus without midbrain. Our cases come under 2nd type of ischemic pattern by N. A. Lazzaro *et al.* [4]. The classical MRI imaging feature in AOP infarction is diffusion restriction in both paramedian regions on diffusion-weighted MRI [5] which is also noted in our cases. The differential diagnosis in imaging of bithalamic lesions is extensive and comprises of arterial and venous occlusion, neoplasm, infectious and

inflammatory lesions. Diagnosing an AOP infarction is critical to direct the proper management and to prevent additional unnecessary procedures [4].

CONCLUSION

AOP occlusion can be life-threatening. Hence the knowledge about AOP and its occlusion leading to bilateral paramedian thalamic infarction is essential to make an accurate diagnosis and timely management.

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