

THE “BUTTERFLY WINGS” AND MIDBRAIN “V” SIGN OF ARTERY OF PERCHERON INFARCTION

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CONSENT STATEMENT

All parties gave their informed consent prior to their inclusion in this write up. Details that may disclose the identity of the patient in this manuscript have been omitted. Written consent of the subject/guardian/spouse has been obtained prior to submission.

An 80-year-old woman with history of hypertension presented to the emergency department following a fall. Her mental status quickly deteriorated and became obtunded. She was stuporous, and unresponsive to verbal stimuli. She had minimally reactive pupils and was moving extremities to painful stimuli. An MRI scan was performed following an unrevealing CT brain.

This case demonstrates classic imaging findings of ischemic stroke in the territory of the Artery of Percheron (AOP). The thalamus is mainly vascularized by the tubero-thalamic artery, thalamo-geniculate artery, paramedian thalamic artery and the posterior choroidal artery. These arterial branches arise from the posterior communicating artery and the posterior cerebral artery (PCA). Each of these major thalamic arteries has predilection for supplying a particular group of nuclei. The AOP is an anatomic variant where a single trunk arises off the P1 segment of PCA to supply both paramedian hemispheres of the thalamus and/or rostral midbrain(1).

MR imaging demonstrated symmetric infarctions of the bilateral paramedian thalami

and rostral midbrain with additional ischemic foci involving the PCA (Figure 1). MRA showed partial occlusion of the basilar artery with beaded appearance suggestive of intracranial atherosclerosis (Figure 2). The mechanism of stroke in this case is likely due to hypoperfusion with artery-to-artery distal embolization. In one of the largest series of AOP infarcts, bilateral paramedian thalamic and rostral midbrain involvement made up about 43% of cases(2). The midbrain V sign is also present in this case, characterised by hyperintense signal intensity along the pial surface of the midbrain interpeduncular fossa. This distinct pattern of V shaped hyperintensity has been described in 67% of AOP cases(2). The symmetrical hyperintensity involving bilateral rostral mesencephalon is also observed to resemble “butterfly wings”.

Other vascular etiologies that can cause bilateral thalamic infarctions include top of the basilar syndrome and deep cerebral venous thrombosis. Both differential diagnoses have been excluded as the tip of the basilar artery was patent on MRA while patent flow voids were

seen within the internal cerebral veins and venous sinuses on MRI.

The distinct pattern of infarction in this case, along with the “butterfly wings” and mid

brain “V” sign, should improve recognition of AOP infarction and assist with the neurologic evaluation and management of patients with thalamic strokes.

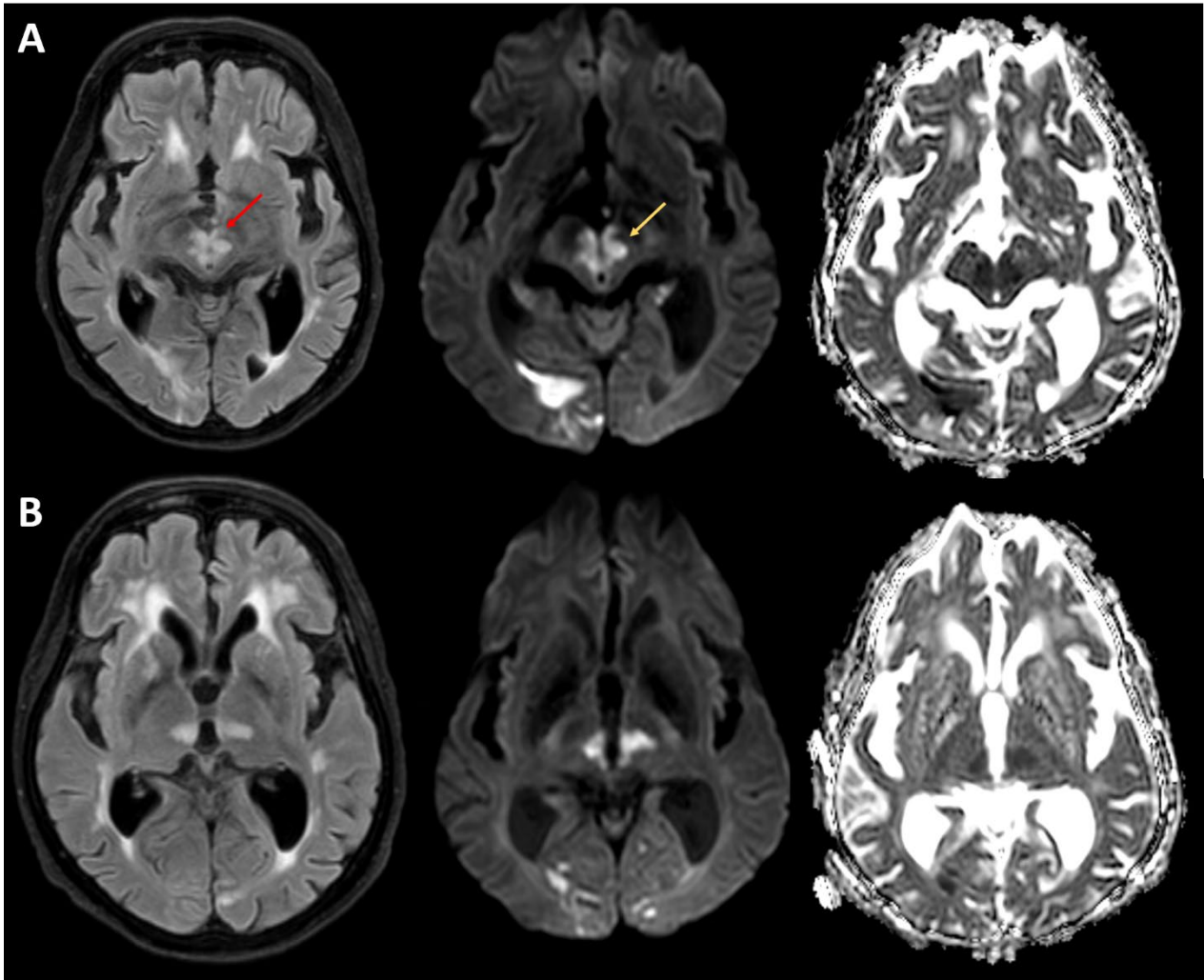


Figure 1:

Panel A showing FLAIR and DWI/ADC images at the level of the midbrain, demonstrating bilateral rostral midbrain infarction. Notice the hyperintense signal intensity along the pial surface of the midbrain interpeduncular fossa representing the V sign (red arrow). The symmetrical hyperintensity at the rostral mesencephalon resembled “butterfly wings” as a whole.

Panel B showing FLAIR and DWI/ADC images at the level of the thalamus demonstrating symmetrical paramedial thalamic infarction.

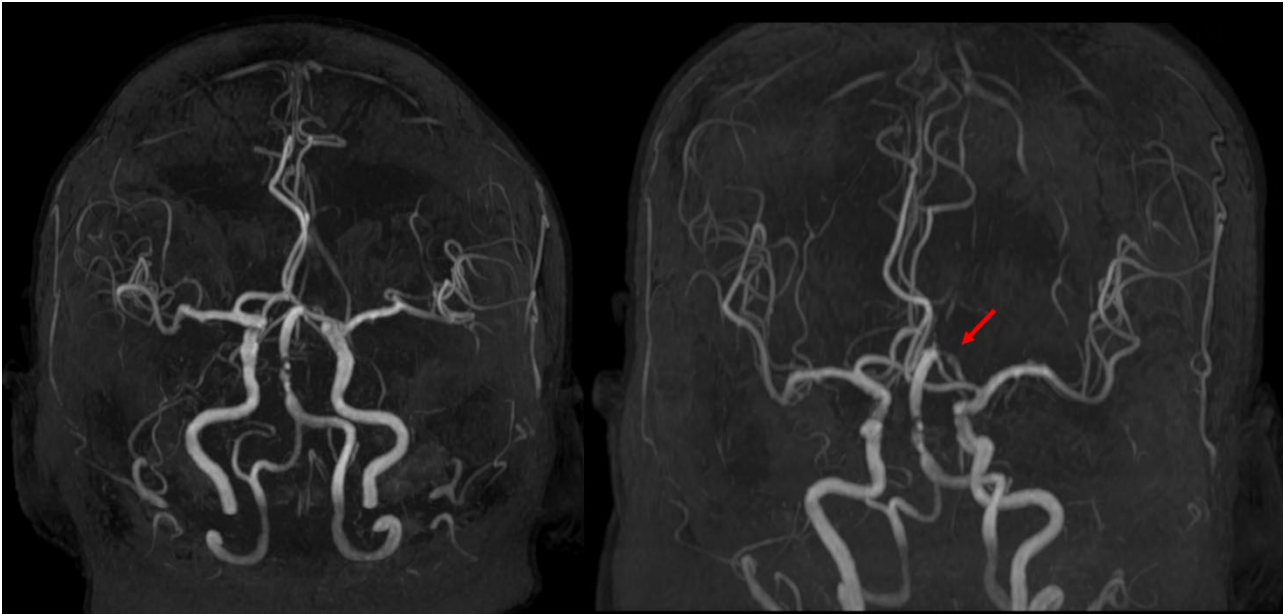


Figure 2: Magnetic resonance angiography showing partial occlusion and beading of the basilar artery suggesting that intracranial atherosclerosis as the aetiology of the stroke. Narrowing of the bilateral P1 segment of the PCA artery was also noted (red arrow).

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