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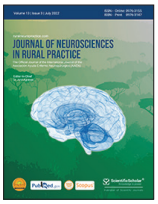

Letter to Editor

Artery of Percheron infarct - classical imaging findings in two cases

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Dear Editor,

Artery of Percheron (AP) infarction is an infrequent type of ischemic stroke involving bilateral paramedian thalami.[1,2] It is responsible for 4–18% of all thalamic infarctions[1] and 0.1–2% of all cerebral strokes[1,2] and can have a variable clinical presentation as detailed below.

A 66-year-old hypertensive male was referred 30 h following the sudden onset of right-sided weakness, slurring of speech and diplopia, postural imbalance, and ataxia. Initial computed tomography scans done elsewhere were normal. Magnetic resonance imaging (MRI) of the brain revealed hyperintensities in T2 and fluid-attenuated inversion recovery (FLAIR) sequences in bilateral paramedian thalami as well as brightness suggesting diffusion restriction in diffusion-weighted images (DWI) and corresponding hypointensities in apparent diffusion coefficient (ADC) sequences suggestive of a recent infarction [Figure 1].

Another 42-year-old male presented 24 h after sudden-onset drowsiness progressing to loss of consciousness and decreased movement of all four limbs. There were no localizing signs. MRI of the brain showed bilateral thalamic hyperintensities in T2 and FLAIR sequences and corresponding diffusion restriction in DWI images [Figure 2].

AP is the name given to the anatomical entity wherein a single vessel arising from one P1 segment feeds bilateral thalami. The thalamoperforating arteries that supply bilateral paramedian thalami arise from the proximal segment (P1 segment) of the posterior cerebral arteries (PCA).[3] They are classified into 4 types based on their number and on whether they arise unilaterally or bilaterally. In Type 1 (20% cases), several vessels for each thalamus arise from respective individual P1 segments; in Type 2 (33%), a single perforator supplying both thalami arises from one P1 segment while the other has no branch; in Type 3 (40%), single perforators for each thalamus originate from the respective P1 of the same side; and in Type 4 (7% cases), multiple vessels arise from any one P1 segment supplying each thalamus separately while the other P1 has no branch.

Yet another classification system[4,5] varies slightly and holds that Type 1 consists of multiple thalamoperforating vessels from both P1 segments (similar to Type 1 described above), Type IIa consists of separate vessels for both thalami arising from a single P1 segment (similar to Type 4 described above), Type IIb consisting of a single thalamoperforator supplying thalami of both sides arising from any P1 segment (similar to Type 2 described above), and Type III multiple vessels arising from an arterial anastomosis connecting the right and left proximal PCAs. Both these classifications have been shown in the schematic diagram [Figure 3].

AP was first described by Percheron in 1973[6] and while occlusion of this vessel gives rise to nearly symmetric bithalamic infarctions as shown in these cases, occasionally, there is additional infarction of the rostral part of the midbrain[7] because the superior mesencephalic (rubral) artery that arises from the P1[7] may be occluded as well. Rarely there may also be involvement of the anterior thalamus particularly if the polar artery of the thalamus (usually a branch of the posterior communicating artery) is absent and the supply is taken over by the AP.

Lazzaro *et al.*[7] have described 4 patterns of AP infarctions - Type 1 having bilateral paramedian thalamic and midbrain infarction, Type 2 having bilateral paramedian thalami without midbrain infarction, Type 3 consisting of bilateral paramedian, anterior thalamic and midbrain infarctions, and Type 4 bilateral paramedian and anterior thalamic but no midbrain infarction.

Both our cases were Type 2 as there was only bilateral paramedian thalamic involvement with no midbrain or anterior thalamic involvement.

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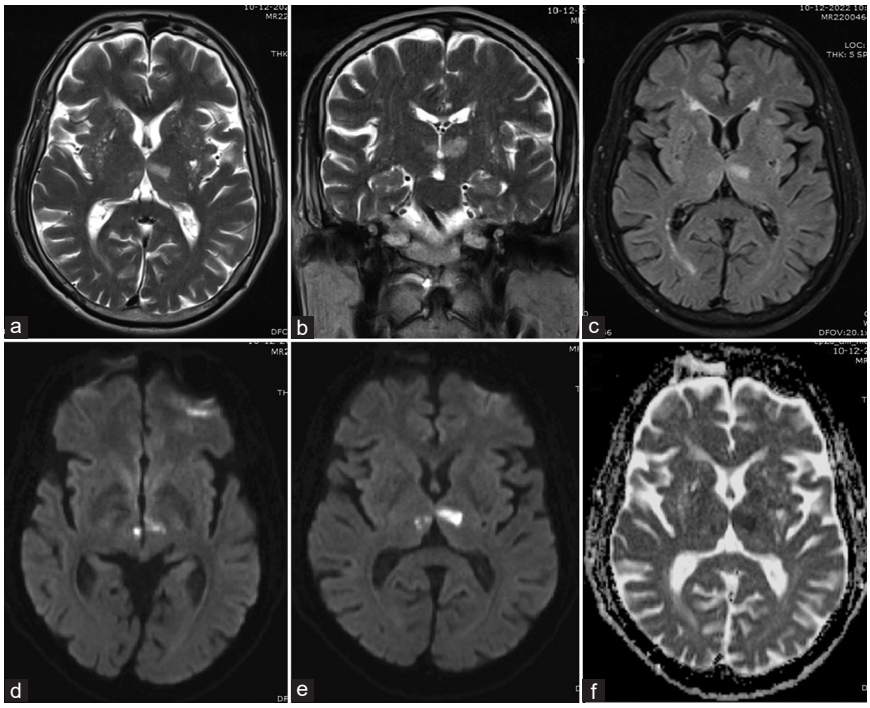

Figure 1: Magnetic resonance imaging showing bilateral paramedian thalamic hyperintensities in (a) T2 axial and (b) coronal (c) images as well as axial fluid-attenuated inversion recovery sequences. (d and e) The lesions show diffusion restriction on diffusion-weighted images sequences and (f) corresponding hypointensities on apparent diffusion coefficient sequences.

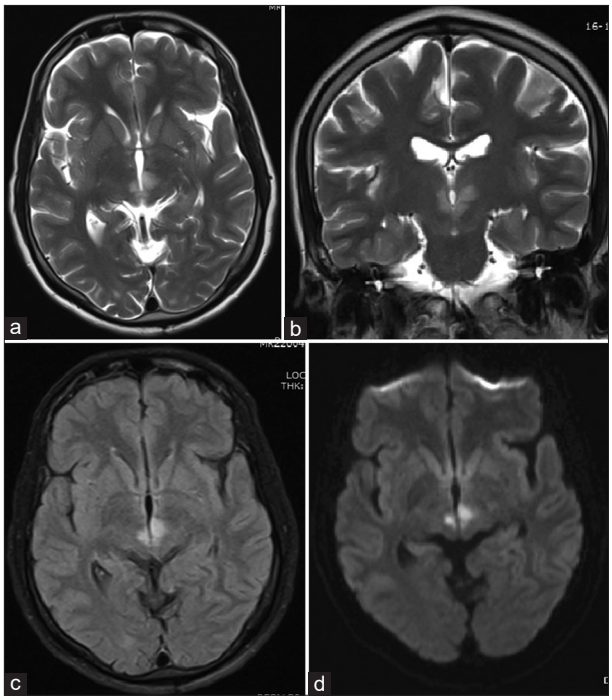

Figure 2: Magnetic resonance imaging showing bilateral paramedian thalamic hyperintensities in (a) T2 axial and (b) coronal images and (c) axial fluid-attenuated i nversion recovery (d) sequences with diffusion restriction on diffusion-weighted images sequences.

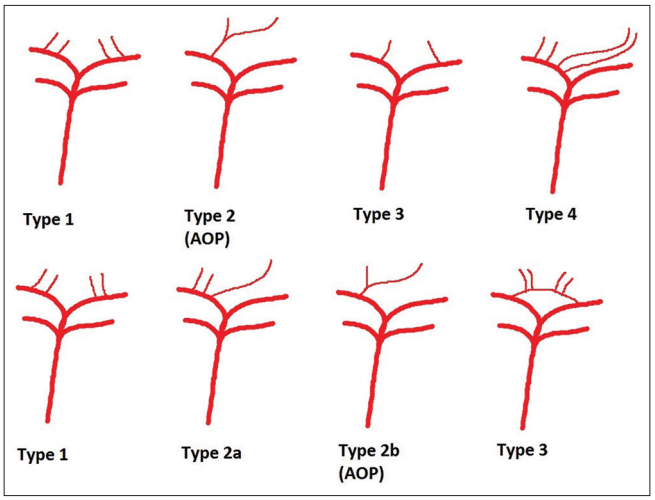

Figure 3: Schematic diagram showing two types of classifications (in two rows) of variations of the thalamoperforating vessels.

Various authors[1,8,9] have stressed that AP infarctions can have varied clinical presentations though the three main symptoms are upward gaze palsy, memory disturbances, and altered sensorium[9] and Khanni *et al.*[8] state that as the classical signs of stroke are absent, these patients are usually diagnosed outside the window for thrombolysis. This happened in both our cases with one presenting with slurred speech, diplopia, and ataxia and the other with coma and quadriparesis.

Lamot *et al.*[9] noted that the AP is usually too small to be visualized by conventional methods and in none of our cases too could we appreciate any vessel occlusion on MR angiography. It was only the classical findings on MRI that led us to this diagnosis.

To conclude, AP infarctions have classical imaging findings (kissing infarctions in bilateral paramedian thalami) on MRI even though they have varied presentations. Knowledge of this will help a physician to clinch the diagnosis even in patients who present with atypical non-localizing symptoms unlike those found in "usual" strokes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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