

Magnetic Resonance Correlate of Hemiparesis in Posterior Cerebral Artery Infarction

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Background: Magnetic resonance (MR) imaging of posterior cerebral artery infarction with hemiparesis (PCAH) is limited to case reports with T2-weighted lesions involving the cerebral peduncle. *Objective:* We sought to extend the MR imaging correlate of PCAH. *Methods:* The case histories and MR/MR angiography findings of 4 patients with PCAH were examined. *Results:* Distinct from previously described MR imaging findings with PCAH, diffusion-weighted imaging and/or FLAIR changes were noted in the thalamogeniculate artery (TGA) territory in one patient; in the distribution of the TGA and lateral posterior choroidal artery (PChA) in two patients; and in the TGA and lateral PChA combined with the peduncular perforating arteries of the cerebral peduncle in one patient. *Conclusion:* Diffusion-weighted imaging changes of infarction in the distribution of the TGA alone or combined with lateral PChA involvement with or without cerebral peduncle infarction correlate with PCAH. **Key Words:** Cerebral infarction—diffusion-weighted imaging—posterior cerebral artery—hemiparesis—magnetic resonance angiography. © 2008 by National Stroke Association

Posterior cerebral artery (PCA) infarction with hemiparesis (PCAH) may mimic middle cerebral artery (MCA) territory infarction¹⁻⁵ with T2-weighted imaging lesions localized to the cerebral peduncle.^{2,3} Subsequent reports suggest more superficial PCA (SPCA) infarction accounts for the majority of cases of PCA with hemiparesis, yet no corresponding neuroimaging changes have been demonstrated.⁵ We describe 4 patients and correlate magnetic resonance (MR) changes remote from the cerebral peduncle with this stroke syndrome.

Case Studies

Case 1

A 76-year-old woman was admitted to our hospital with left-sided weakness, numbness and tingling, and difficulty seeing on the left side.

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Neurologic Examination

The patient did not recognize her left hand as hers and had a left homonymous hemianopia. Motor testing showed 2/5 left hemiparesis and left-sided hyperreflexia with an extensor plantar response on the left. After a negative computed tomography (CT) scan on admission she was treated with intravenous (IV) tissue plasminogen activator (tPA) for a suspected MCA stroke. National Institutes of Health Stroke Scale (NIHSS) score was 8 on admission and an electrocardiogram showed atrial fibrillation. MR imaging (MRI)/MR angiography (MRA) showed right PCA territory infarction with occlusion of the P2 segment (Fig 1).

Case 2

An 88-year-old woman with a history of atrial fibrillation was admitted to hospital with left-sided weakness, neglect, and visual field defect. She was on warfarin.

Neurologic Examination

Language and cranial nerves were normal. Visual field testing showed a left homonymous hemianopia. Motor testing showed 3/5 left-sided hemiparesis, and sensation to light touch and pinprick was decreased on the left side. Electrocardiogram showed atrial fibrillation. Her international normalized ratio was subtherapeutic on

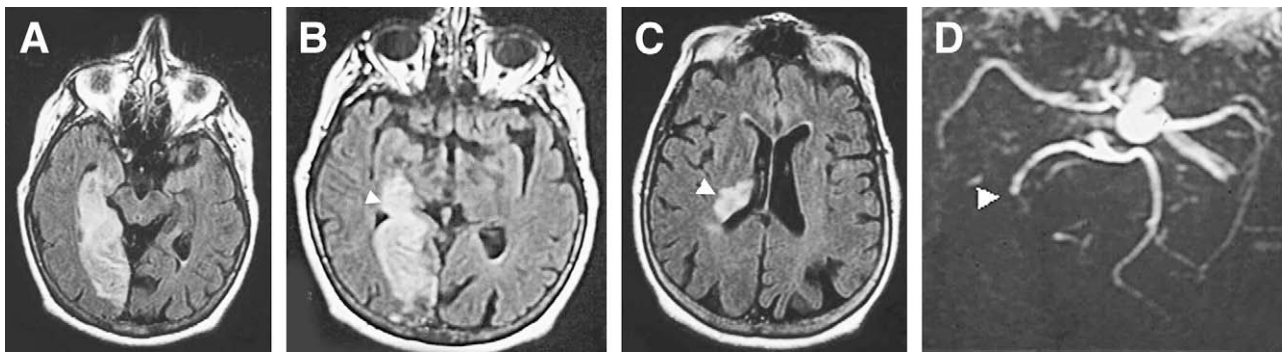


Figure 1. MR-FLAIR sequence showing right PCA infarction (A to C) with involvement of cortical, TGA (B) (arrowhead), and PChA (C) (arrowhead) territory. MRA with occlusion of P2 segment of right PCA (D) (arrowhead).

admission at 1.48. NIHSS score on admission was 6. MRI/MRA showed right PCA territory infarction with occlusion of the P1 segment (Fig 2).

Case 3

An 83-year-old woman with a history of atrial fibrillation suddenly collapsed with right-sided weakness, confusion, and garbled speech. After a negative CT scan she was treated with IV tPA at an outside hospital.

Neurologic Examination

She was alert and oriented to place and person with intact comprehension but exhibited mild expressive difficulty. Cranial nerve testing showed a right homonymous hemianopia. Motor testing showed 3/5 right-sided hemiparesis with extinction on double simultaneous stimulation on the right. NIHSS score on admission was 7. MRI/MRA showed left PCA territory infarction with occlusion of the P1 segment (Fig 3).

Case 4

An 85-year-old man suddenly noted left-sided numbness and weakness and was admitted to the hospital.

Neurologic Examination

He was alert with no language abnormality. Motor testing showed dense left-sided hemiplegia graded 0/5 in-

cluding the face. There was decreased sensation to pinprick on the left side. Electrocardiogram showed sinus bradycardia. CT scan at 2 hours from onset of symptoms showed no evidence of infarction and IV tPA was administered. After several hours the hemiparesis dramatically improved to 4/5 motor weakness on the left. NIHSS score on admission was 9. MRI/MRA showed right PCA territory infarction with occlusion of the P2 segment (Fig 4).

Discussion

Hemiparesis is the main clinical feature distinguishing proximal PCA (PPCA) infarction from other types of PCA strokes⁶ and is generally regarded as a result of infarction of the cerebral peduncle as seen on neuroimaging.¹⁻⁴ More recently, PCAH is described in as many as 18% of patients with PCA infarcts⁵ with some 66.7% secondary to SPCA territory infarction, differing from previous reports where PPCA infarcts were considered the likely cause of the hemiparesis.¹ No corresponding neuroimaging findings, however, were presented to correlate a specific stroke location with hemiparesis in the cases with SPCA infarction.

The patients with PCAH presented here, 3 of whom had unrecognized, untreated, or inadequately anticoagulated atrial fibrillation, showed diffusion-weighted imaging (DWI) change in an area other than, or in addition to, the cerebral peduncle or cerebral cortex, namely in the distribution of the thalamogeniculate artery (TGA) and lateral posterior

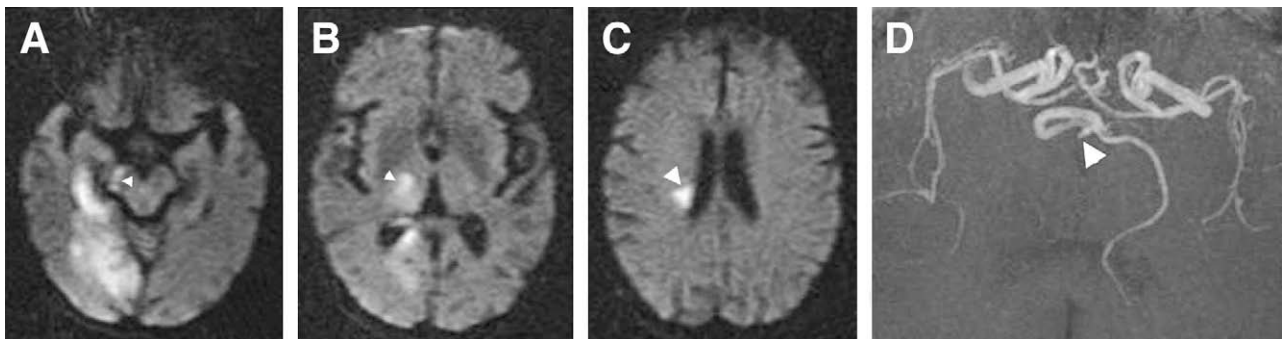


Figure 2. DWI showing restricted diffusion caused by right PCA infarction (A to C) with involvement of cortical, TGA (B) (arrowhead), and PChA (C) (arrowhead) territory and right cerebral peduncle (A) (arrowhead). MRA with occlusion of P1 segment of right PCA (D) (arrowhead).

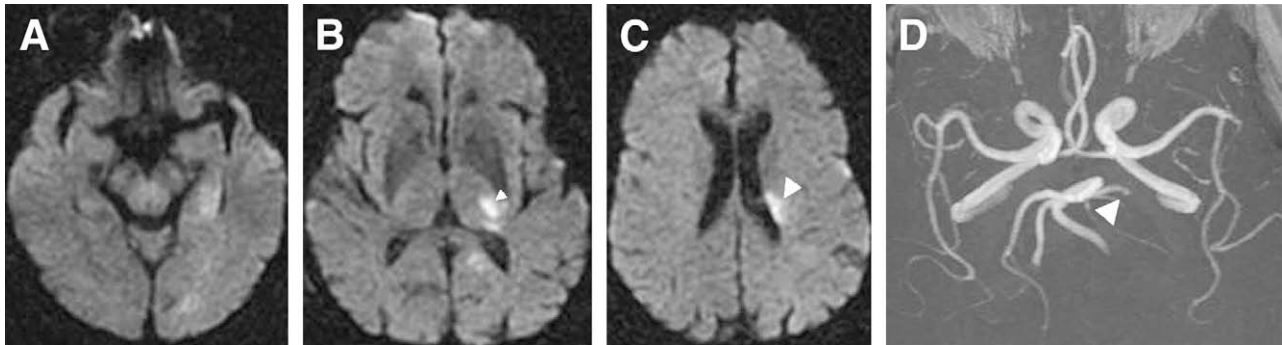


Figure 3. DWI showing restricted diffusion caused by left PCA infarction (A to C) with involvement of TGA (B) (arrowhead) and PChA (C) (arrowhead) territory. MRA with occlusion of P1 segment of left PCA (D) (arrowhead).

choroidal artery (PChA). Given the sensitivity and relative specificity along with the development of signal change within minutes of ischemia, DWI is ideally suited to correlate signs and symptoms of stroke with lesion location as compared with CT imaging, particularly in these two arterial territories. Similarly, DWI identifies small MCA territory strokes that may occur coincident with PCA infarction.

The initial portion of the PCA, before the posterior communicating artery anastomosis from the internal carotid artery, is the precommunal or P1 segment of the PCA from which arises the paramedian mesencephalic, thala-

moperforate, and medial PChA whereas the peduncular perforate, TGA, and lateral PChA and anterior temporal artery arise from the postcommunal or P2 segment.⁶ The medial PChA supplies the medial geniculate body, and the medial nucleus and the pulvinar while the lateral PChA vascularizes part of the lateral geniculate body, part of the dorsomedial nucleus, and part of the pulvinar and lateral wall of the lateral ventricle (LWLV).^{7,8}

TGA territory infarction results from PPCA occlusion or TGA branch occlusion with similar clinical features including: (1) hemisensory loss, hemiataxia, and

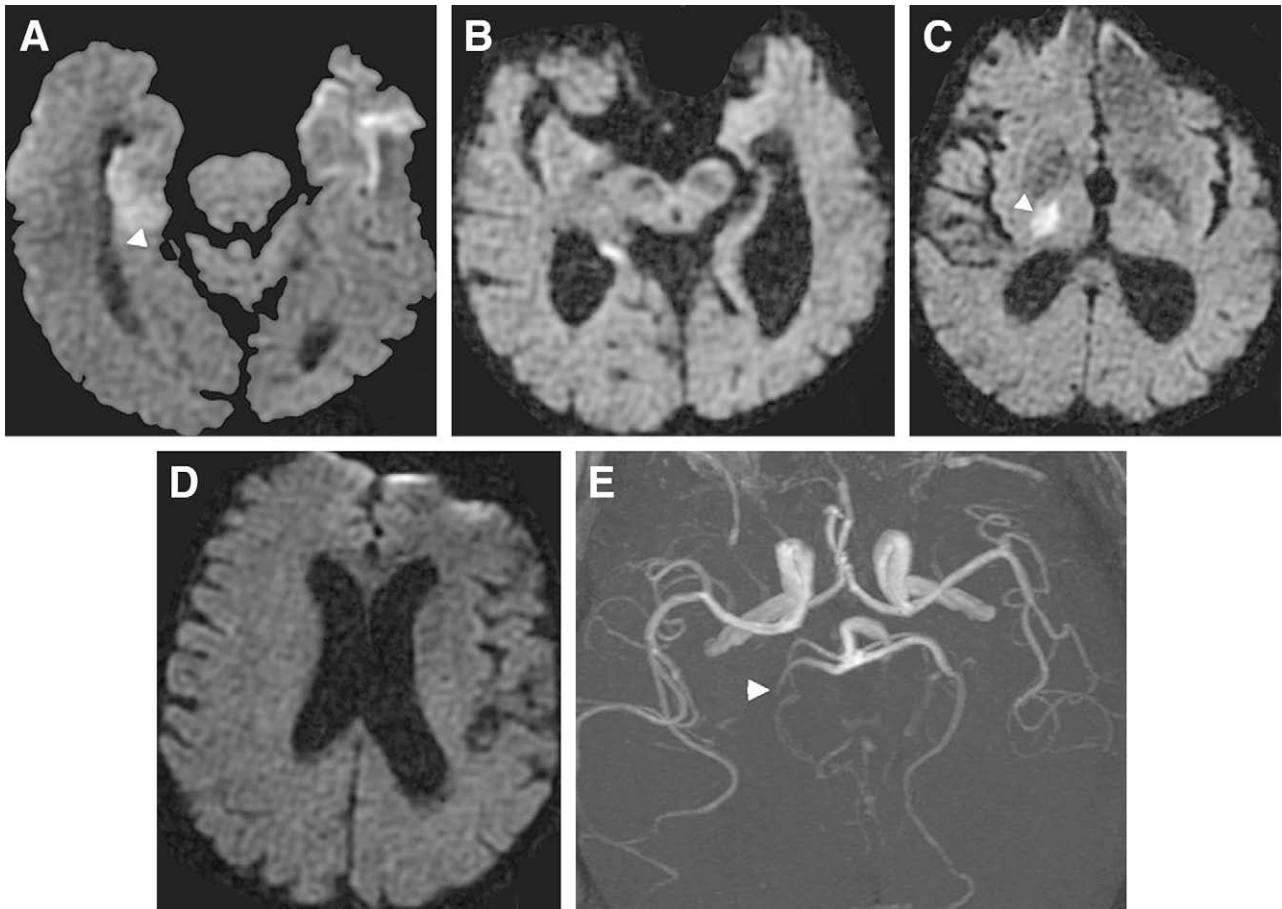


Figure 4. DWI showing restricted diffusion caused by right PCA infarction (A to D) with involvement of anterior temporal artery (A) (arrowhead) and TGA (C) (arrowhead). MRA with occlusion of P2 segment (E) (arrowhead).

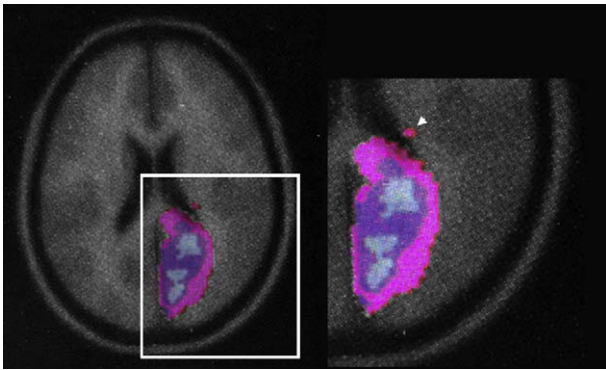


Figure 5. Digital map of PCA infarction (left) with visualization of lateral PChA territory in LWLV (inset, right) (arrowhead).¹⁴ Reprinted with permission.

involuntary movements; (2) pure sensory stroke; and (3) sensory-motor stroke with hemiparesis as a result of involvement of the posterior limb of the internal capsule adjacent to the thalamus.⁹

Although occlusion of the PPCA can result in infarction limited to the TGA territory, more often there are accompanying PChA and cortical infarcts.⁹

Neau and Bogousslavsky¹⁰ described 10 patients with PChA infarction and reviewed 10 others and noted hemiparesis in 8 with equal involvement of the lateral and medial PChA. They stated hemiparesis with PChA infarction may be related to the corticobulbar and corticospinal fibers in the adjacent posterior limb of the internal capsule, yet the origin remains unclear and with no visible involvement of the internal capsule or midbrain on CT or MRI. The absence of a prior neuroimaging correlate may relate to the fact that hemiparesis in their cases was mild and transient and DWI sequence was not obtained. It has been suggested that under-recognition of hemiparesis in PCA infarction may be a result of a high degree of recovery early on related to partial involvement of the posterior limb of the internal capsule, however, in the context of peduncular involvement severe hemiplegia is noted.^{11,12}

The lateral ventricular margin overlying the posterolateral thalamus is reported to be the most distal territory of the lateral PChA.^{8,13} This is in keeping with a digital map of PCA infarction¹⁴ where enlargement of the digital image shows a discrete abnormality in the LWLV (Fig 5). The area of restricted diffusion (cases 2 and 3) and fluid-attenuation inversion recovery (FLAIR) abnormality (case 1) in the LWLV corresponds to the aforementioned distribution of the lateral PChA.

The concept of stroke associated with PCAH remote from the cerebral peduncle⁵ is correlated with infarction

of the lateral PChA and/or TGA territory, areas best appreciated with DWI sequence. MRA accurately demonstrates the proximal portion of the PCA and in all 4 of our patients showed PPCA occlusion variably involving territory of the peduncular perforate, TGA, and/or lateral PChA on DWI. MRA and DWI findings described here further define infarct location associated with the PCAH stroke syndrome.

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