

Neuroimaging in Acute Posterior Cerebral Artery Infarction

Pasquale F. Finelli, MD

Background: The role of neuroimaging in acute posterior cerebral artery (PCA) territory infarction is less well appreciated compared with the anterior cerebral circulation because PCA infarction occurs less frequently and more often is associated with limited neurologic deficit not qualifying for thrombolytic therapy. On occasion, however, hemiparesis and/or visual field defect accompanies PCA infarction that would warrant thrombolytic therapy.

Review Summary: As neuroimaging plays a central role in the diagnosis and treatment of acute stroke in the thrombolytic era, a series of case studies is presented to illustrate the role of computed tomography and magnetic resonance imaging in the setting of acute PCA infarction.

Conclusion: Familiarity with the neuroimaging features of acute PCA infarction can facilitate management in those select patients that qualify for thrombolytic therapy.

Key Words: thrombolytic therapy, MR imaging, CT scan, posterior cerebral artery infarction, neuroimaging

(The Neurologist 2008;14: 170–180)

The etiology, pathogenesis and clinical features of posterior cerebral artery (PCA) infarction are well described,^{1–3} yet the neuroimaging is less well appreciated compared with the anterior cerebral circulation. As neuroimaging plays an ever increasing part in the diagnosis and treatment of cerebrovascular disease, a series of case studies is presented (Table 1) to illustrate the role of computed tomography (CT) and magnetic resonance (MR) imaging in the setting of acute PCA infarction (Figs. 1–14).

ANATOMIC CONSIDERATIONS

The paired PCA arteries are formed by the bifurcation of the basilar artery and are the main blood supply to the uppermost midbrain, thalamus, and posterior inferior portions of the cerebral hemispheres, including the occipital lobes, the medial and inferior temporal lobes, and the inferior medial parietal lobes.⁴ The first portion of the PCA artery is the P1 segment whose branches supply the medial midbrain, hypothalamus, and thalamus, followed by the P2 segment whose

branches supply the cerebral peduncles and lateral thalamus and more distal branches that supply the cerebral cortex.⁵ The polar artery arising from the posterior communicating artery (PCOM) is unique in that its blood supply may come alternatively from the anterior or posterior circulation to supply the frontal most area of the thalamus. Variant circulation is noted particularly with a dominant PCOM (ie, fetal-type PCA) as seen in 20% to 30% of patients where the internal carotid artery is the principle blood supply of the PCA, and paramedian perforators that occasionally originate from a common trunk (Percheron's artery) to supply the upper midbrain and both thalami.¹ Depending on the location of the vascular obstruction, any combination of midbrain, thalamus, and PCA supplied cerebral hemisphere may be involved by PCA stroke. The most common pattern is hemispheric involvement followed by thalamus and hemisphere, followed by isolated thalamus, and lastly a combination of midbrain, thalamus, and/or hemisphere.

STROKE MECHANISM AND CLINICAL FEATURES

The etiology of isolated PCA territory infarction most

Depending on the location of the vascular obstruction, any combination of midbrain, thalamus, and posterior cerebral artery supplied cerebral hemisphere may be involved by posterior cerebral artery stroke.

commonly is cardioembolic.¹ However, involvement of the cerebellum or brainstem in association with PCA infarction more commonly is due to vertebral or intracranial vertebrobasilar artery disease.¹ The diverse areas of brain supplied by the PCA that include brainstem, thalamus, and cerebral hemisphere account for the protean clinical and neuroimaging manifestations. The 3 most frequent and prominent symptoms and signs of PCA infarction are visual field defect (95%), most commonly homonymous hemianopia, paresthesias (40%), and headache (50%). Distinct from middle cerebral artery (MCA) infarction, in PCA territory infarcts, patients are usually aware of their visual loss, and in many

From the Department of Neurology, Hartford Hospital and University of Connecticut School of Medicine, Hartford, Connecticut.

Reprints: P. F. Finelli, MD, Hartford Hospital, 80 Seymour Street, Hartford, CT 06102-5037. E-mail: pfinell@harthosp.org.

Copyright © 2008 by Lippincott Williams & Wilkins

ISSN: 1074-7931/08/1403-0170

DOI: 10.1097/NRL.0b013e3181627679

TABLE 1. Case Vignettes

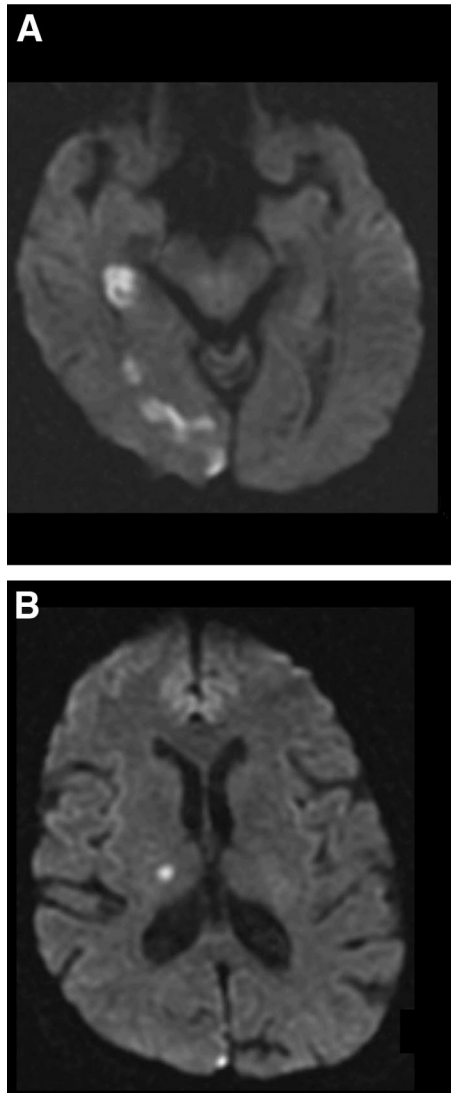
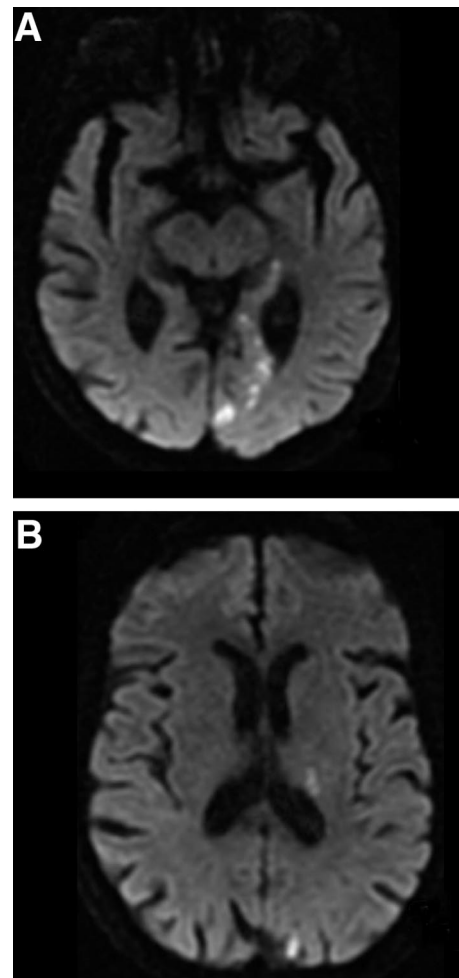
Case 1.	A 70-yr-old woman presented with a 2-d history of left-sided numbness and left visual field defect. EKG was normal and TTE showed a patent foramen ovale and TEE showed an interatrial septal wall aneurysm. Fig. 1. Right PCA infarction (thalamus/hemisphere) on DWI (A, B) with negative initial CT. MR shows the advantage of DWI for location and extent of lesion.
Case 2.	A 83-yr-old woman presented with several days of confusion on a background of 1 yr of forgetfulness. Her recent memory and calculations were impaired, and she did not know the name of the governor of the state. She had a visual field impairment, and the diagnosis of progressive dementia was considered. Fig. 2. Left PCA infarction (thalamus/hemisphere) on DWI (A, B) with negative initial CT. Her symptoms mostly resolved at 6 wk. Alzheimer disease was considered until PCA infarction was seen. MR shows the advantage of DWI for location and extent of lesion in a patient primarily with neuropsychological features.
Case 3.	A 70-yr-old woman who presented with sudden right-sided weakness and sensory loss with right homonymous hemianopia and impaired recent memory was treated with IV tPA. Fig. 3. Neuroimaging shows dense vessel sign on CT (A arrowhead), left PCA infarction (thalamus/hemisphere) on DWI (B) and left P2 segment PCA occlusion on CTA (C arrowhead) with recanalization by MRA and CTA (D, E).
Case 4.	A 75-yr-old man awakened with right arm numbness and right leg weakness that improved over 24 h. CT at an outside hospital was normal and carotid Doppler showed a preocclusive left internal carotid artery stenosis. Warfarin was started but patient developed right hemiparesis, aphasia, and left gaze preference. Fig. 4. Left PCA infarction (thalamus/hemisphere) on DWI (A) with preocclusive left internal carotid artery stenosis MRA (left), CTA (right) (B arrowheads) and fetal-type PCA on left, MRA (C arrowhead). Repeat DWI (D) showed progression of infarction. Heparin may have been a better choice in the short term. Imaging shows role of the carotid artery in PCA infarction with fetal-type circulation and value of CTA and MRA in delineating the vascular anatomy.
Case 5.	A 20-yr-old woman presented to ER with a 3-d history of double vision and droopy right eyelid, sleepiness, and unsteady gait. She was 3 mo postpartum, smoked 1/2 pack of cigarettes/d and was treated with Depoprovera. TTE showed a PFO with aneurysmal wall dilatation. Fig. 5. Discrete circular shaped lesion of right hypothalamus and thalamus seen on CT and DWI (A) most likely is an infarction in a thalamoperforate artery. Follow-up MR at 1 mo showed almost total resolution of the lesion. The circular appearance of this infarct leads to the misdiagnosis of neoplasm by several vascular specialists.
Case 6.	A 70-yr-old man presented with transient 30 min of numbness of left side of tongue and mandibular region. Next day he had a recurrence including numbness of fingers of the left hand and weakness of the left leg. A TTE was negative. Fig. 6. CT on admission was normal. DWI (A, B) study showed a discrete infarct of the right cerebral peduncle and thalamus.
Case 7.	A 76-yr-old woman admitted with left sided numbness and tingling and difficulty seeing on the left side. Examination showed a dense left hemiparesis and left homonymous hemianopsia. Patient was treated with IV tPA. Repeat EKG showed atrial fibrillation and carotid Doppler showed a right carotid occlusion. Fig. 7. CT (A) at 2 h was negative and DWI (B) at 24 h showed a right PCA infarction (thalamus/hemisphere). MRA (C) of the neck confirmed the right carotid bifurcation occlusion and MRA (D arrowhead) of the head showed evidence of a right P2 segment PCA occlusion and no evidence of fetal-type PCA. This case illustrates the value of neuroimaging when PCA infarction mimics MCA infarction.
Case 8.	A 71-yr-old man with prior stroke was admitted with 1 wk history of word-finding difficulty and memory problems. A TTE showed PFO with an interatrial septal aneurysm. Fig. 8. Left thalamic infarction in the distribution of the polar artery that arises from the PCOM artery. Imaging delineates the vascular anatomy of fetal PCA with hypoplastic P1 segment on MRA (B arrowhead) and infarction location on CT (A) and DWI (C).
Case 9.	A 74-yr-old woman transferred from outside hospital with decreased consciousness and acute myocardial infarction following chest pain and syncope. She followed 1-step commands and drifted off. There was a suggestion of a left visual field defect. Fig. 9. Right PCA (thalamic/hemisphere) and bilateral thalamic infarctions, initial CT (A) negative, DWI (B–D) positive. Decreased level of consciousness without hemiparesis with a suggestion of field defect and negative early CT suggests bilateral thalamic infarction as seen on DWI.
Case 10.	A 63-yr-old man was admitted with the new onset of a right visual field defect. EKG was consistent with atrial fibrillation. Fig. 10. CT (A, B) showed left temporal tip decreased attenuation. FLAIR (C) showed the temporal tip lesion to be old (probable arachnoid cyst) and DWI (D) identified a new area of acute infarction in the left PCA territory. Imaging illustrates how MR sequences help with the diagnosis by determining the age of a lesion.
Case 11.	A 75-yr-old man was admitted with a right upper extremity seizure. One year ago, he suffered bilateral occipital infarction and 8 mo ago was diagnosed with lung cancer. Fig. 11. Neuroimaging showed a left occipital hypodensity on CT (A) and DWI (B) and increased signal on FLAIR (C) reported to be consistent with infarction. A subsequent T1-weighted enhanced MR (D, E arrowhead) showed a ring-enhancing lesion consistent with metastasis. Vasogenic edema can be appreciated on the unenhanced images.
Case 12.	A 75-yr-old woman collapsed in church and admitted to an outside hospital with a left hemiparesis and a right gaze deviation. CT at 1 h showed bilateral occipital lobe decreased attenuation. EKG showed new atrial fibrillation. She was treated with IV tPA and transferred to our hospital. Fig. 12. Bilateral PCA territory with decreased attenuation, CT (A), early changes of a right MCA territory infarction, CT (B) and an acute right MCA infarction, DWI (C), FLAIR (D, E), and ADC (F). The bilateral PCA infarctions were old and the right MCA infarction new. Neuroimaging demonstrates the advantage of DWI, FLAIR, and ADC sequences over CT in distinguishing between acute and chronic infarctions.

(Continued)

TABLE 1. (Continued)

Case 13.	A 49-yr-old man was admitted with slurred speech and a right sided weakness and a right homonymous hemianopia. He was treated with IV tPA. Fig. 13. Neuroimaging shows dense left PCA P2 segment, CT (13 arrowhead) and absent left PCA with a fetal-type right PCA, CTA (B arrowhead, arrow), with mismatch in the left PCA territory infarction. CTP (CBV, CBF, MTT) (C–E). Color scale of the CTP images indicate the state of cerebral perfusion where blue represents a decrease in cerebral blood flow (CBF) and prolonged mean transit time (MTT) (D, E) more so than the decrease in cerebral blood volume (CBV) (13C). Left PCA infarction was seen, DWI (F) with recanalization of the left PCA, MRA (G).
Case 14.	A 62-yr-old woman presented with an 18 h history of left arm and leg numbness and weakness. Neurologic examination showed mild motor and sensory deficits confined to the extremities on the left. Electrocardiogram showed new onset of atrial fibrillation and MR imaging showed evidence of recent proximal PCA infarction. She was anticoagulated and discharged after 3 d without deficit. Fig. 14. Neuroimaging shows restricted diffusion in the distribution of the right ATA (A–C) and thalamostriate arteries (D), and proximal occlusion of the right PCA on MRA (E arrowhead).

DWI indicates diffusion-weighted image; PCA, posterior cerebral artery; EKG, electrocardiogram; TTE, transthoracic echocardiogram; TEE, transesophageal echocardiogram; WNL, within normal limits; PFO, patent foramen ovale; IV tPA, intravenous tissue plasminogen activator; CTA, CT angiography; CTP, CT perfusion; MRA, MR angiography; FLAIR, fluid attenuation inversion recovery; ADC, acquired diffusion coefficient; PCOM, posterior communicating artery; ATA, anterior temporal artery.

**FIGURE 1.** Right PCA infarction.**FIGURE 2.** Left PCA infarction.

patients the only accompanying symptoms are hemisensory.¹ Hemiparesis is uncommon and when present is mild and transient. Neuropsychological abnormalities associated with PCA infarction are varied and include, memory dysfunction followed by aphasia, dyslexia, dyscalculia, color anomia, disorientation, visual agnosia, and palinopsia, seen in up to

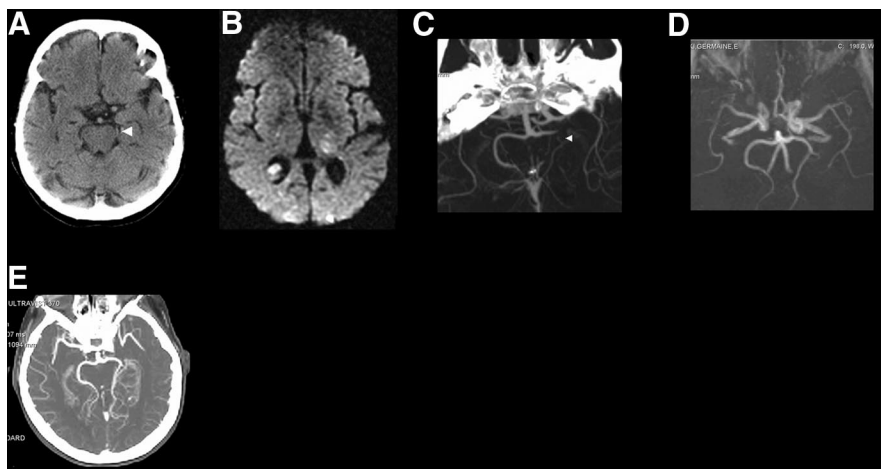


FIGURE 3. Left PCA infarction.

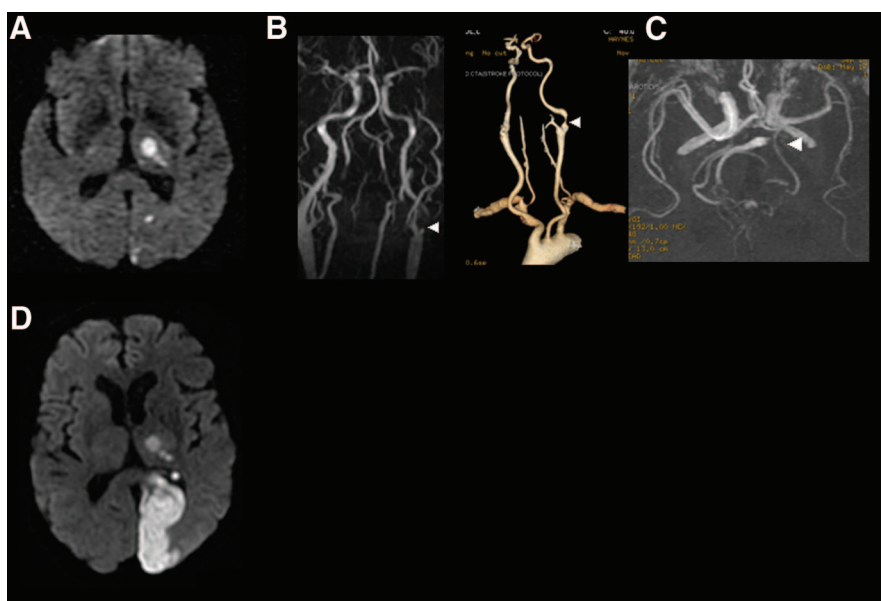


FIGURE 4. Left PCA infarction.

32% of patients.^{6,7} Thalamic infarction particularly when bilateral may be associated with profound lethargy.

NEUROIMAGING FEATURES

Much has been written about the neuroimaging in

As with middle cerebral artery territory stroke, computed tomography may show a dense vessel sign of the basilar or posterior cerebral artery as the earliest feature of posterior circulation stroke.

cerebrovascular disease, mostly in regard to the internal

carotid artery and MCA, the arterial territories most commonly involved in stroke. The PCA, the second most common arterial territory involved in cerebral infarction, accounts for some 25% of ischemic strokes^{5,8} and can have similar neuroimaging applications in the acute stroke setting (Table 2) as described with MCA territory infarction. The diagnosis of stroke is primarily clinical and the principle goal of neuroimaging in acute stroke management is to determine suitability for intravenous thrombolytic therapy by excluding a nonischemic cause of the patient's symptoms and determining whether there is salvageable brain.¹⁰ In select centers, with resources and physician expertise in interventional studies, the role of imaging is extended to identify cerebral thromboembolism amenable to interventional therapy such as intra-arterial (IA) thrombolytic therapy and/or mechanical clot retrieval.¹¹ Neuroimaging features apart from those directly related to thrombolytic therapy that contribute to the overall understanding of the various aspects of PCA infarction as mechanism, etiology, and response to therapy are noted (Table 2).

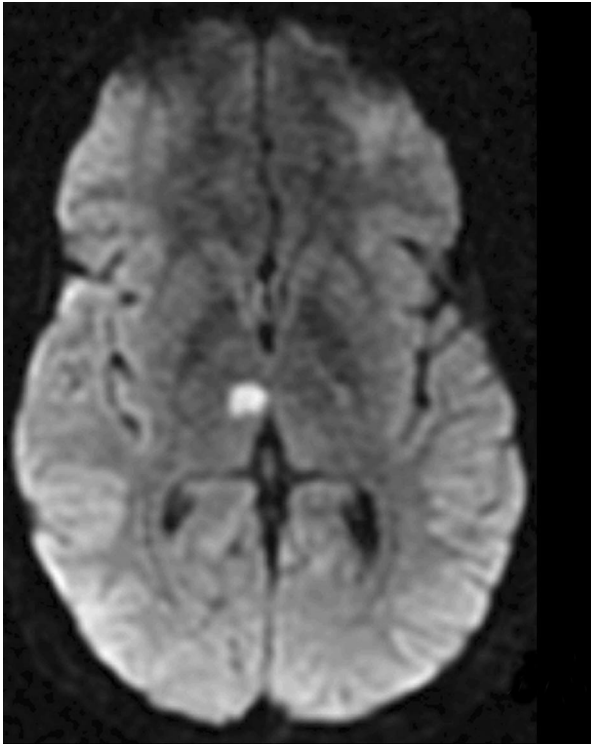


FIGURE 5. Right thalamic infarction.

Neuroimaging now plays a central role in hyperacute stroke management and its availability in many medical centers includes CT imaging with CT angiography (CTA) and CT perfusion (CTP), and increasingly MR imaging with MR angiography (MRA) without and with contrast enhancement (CE MRA), diffusion-weighted imaging (DWI), and to a lesser extent MR perfusion. Familiarity with these modalities can provide the information necessary in the urgent management of PCA infarction and obviate the need for more invasive procedures as conventional cerebral angiography. Echocardiography, transcranial, and carotid artery doppler studies, an integral part of the evaluation of cerebrovascular disease, are not considered here.

CT, the most widely and readily available modality for brain imaging, may not show parenchymal changes for several hours after stroke onset, the time most critical for initiation of stroke therapy. Nevertheless, as with MCA territory stroke, CT may show a dense vessel sign of the basilar or PCA¹² as the earliest feature of posterior circulation stroke (cases 3 and 13). This CT sign is due to a calcific embolus or in situ atherothrombosis or a thromboembolic clot. CTA serves a similar role to that of MRA, however, only CE MRA depicts vessel anatomy with clarity similar to that of CTA. Recently, with high-field-strength MR imaging, 3D TOF-MRA with sensitivity encoding at 3T has resulted in substantial improvement of spatial resolution and advanced MRA as a reliable noninvasive diagnostic tool for the detection of steno-occlusive disease of major intracranial arteries¹³; however, this technology has limited availability at present. CTA does, however, involve ionizing radiation and

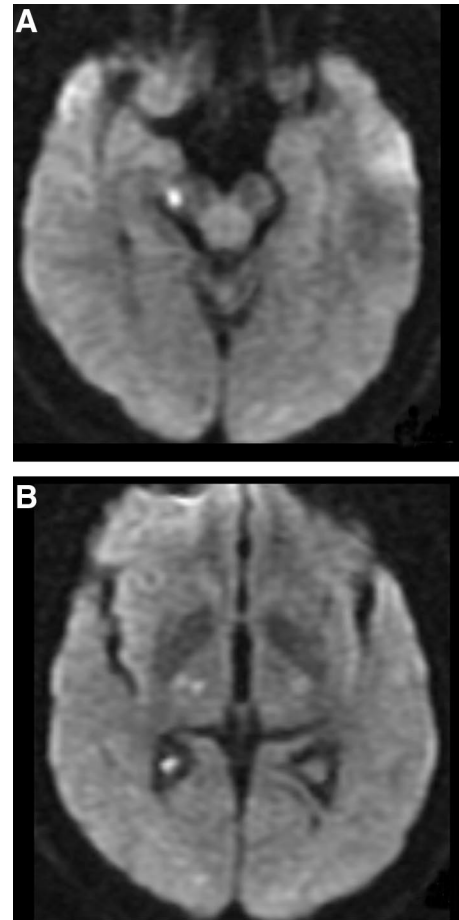


FIGURE 6. Right thalamic/peduncular infarction.

the risk of contrast induced nephropathy which is largely offset by sodium bicarbonate nephroprotection. In the acute stroke setting CTA assesses for stenosis, occlusion and plaque morphology of both the vertebrobasilar and internal carotid arteries as well as for arterial dissection and intracranial steno-occlusive and thromboembolic disease¹⁴ amenable to interventional modalities such as IA thrombolytic therapy and mechanical clot retrieval. Further, vessel wall calcification and anatomy with collateral and variant circulation that may relate to the PCA are defined. Arterial dissection involving the PCA is a rare cause of infarction of this arterial territory and has been demonstrated on MRA.¹⁵

A difference of opinion exists concerning the imaging modality of choice in hyperacute stroke evaluation, ie, MR versus CT. MR is favored by some¹⁶ while a trend toward a “triple study” of CT, CTA, and CTP (total time ~15 minutes) is recommended by others.⁹ A recent comparative study of CTA/CTP to MR gave comparable results when used to select stroke patients for thrombolytic therapy.¹⁷ Features favoring CT include: (1) less time consuming, (2) relative lack of contraindications, (3) more readily available in emergency department setting, (4) monitoring patient in CT room is easier, and (5) CT is generally less costly than MR. Nevertheless, individual institutional resources, expertise and particular patient needs might support the use of MR.

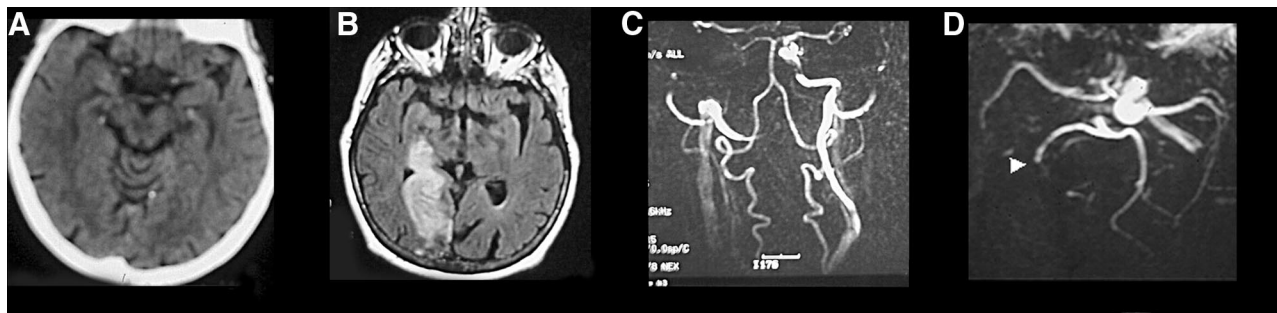


FIGURE 7. Right PCA infarction.

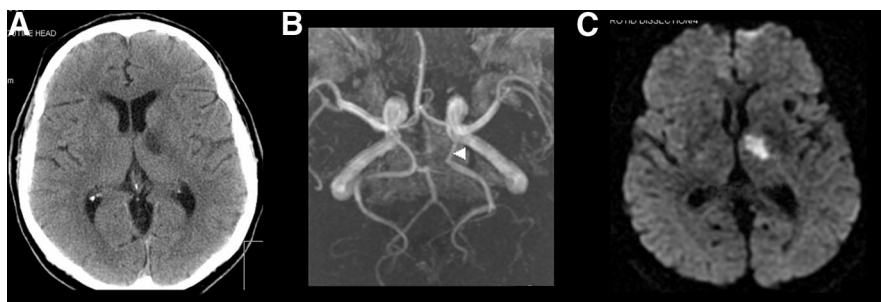


FIGURE 8. Left thalamic infarction.

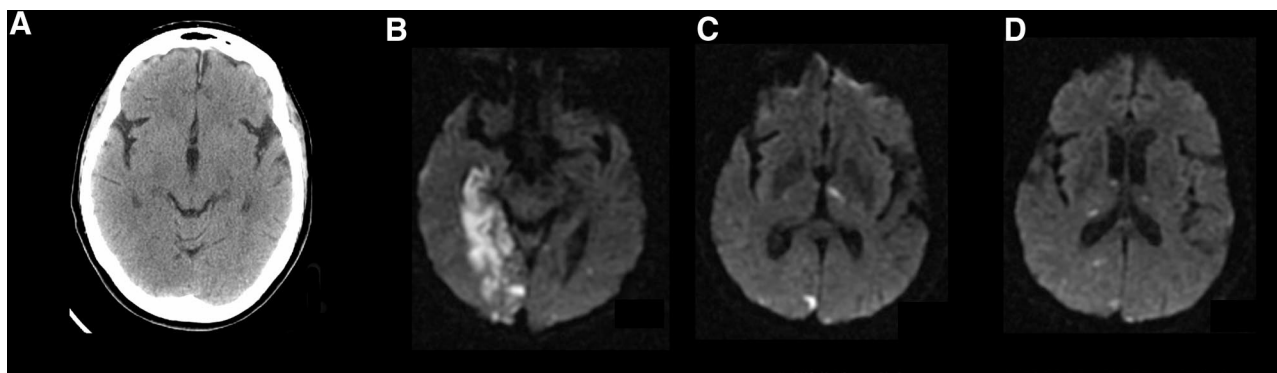


FIGURE 9. Bilateral PCA infarction.

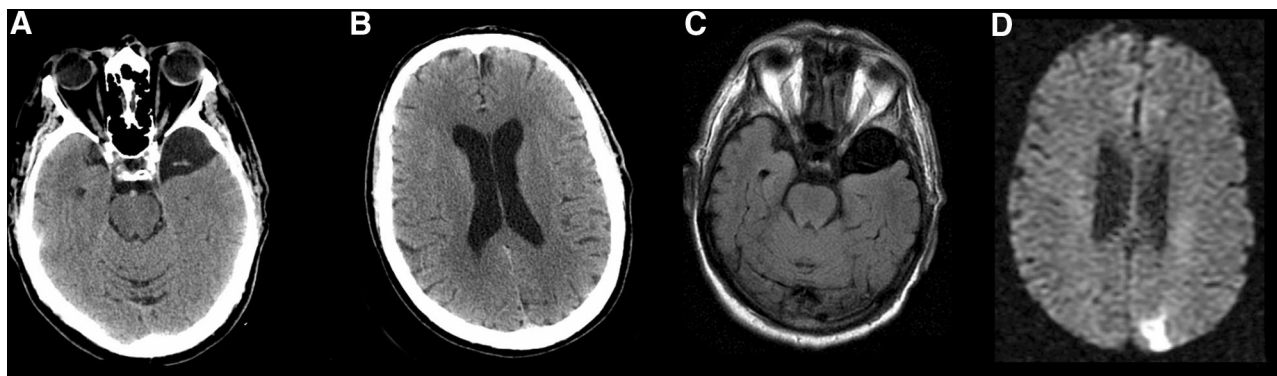


FIGURE 10. Left PCA infarction.

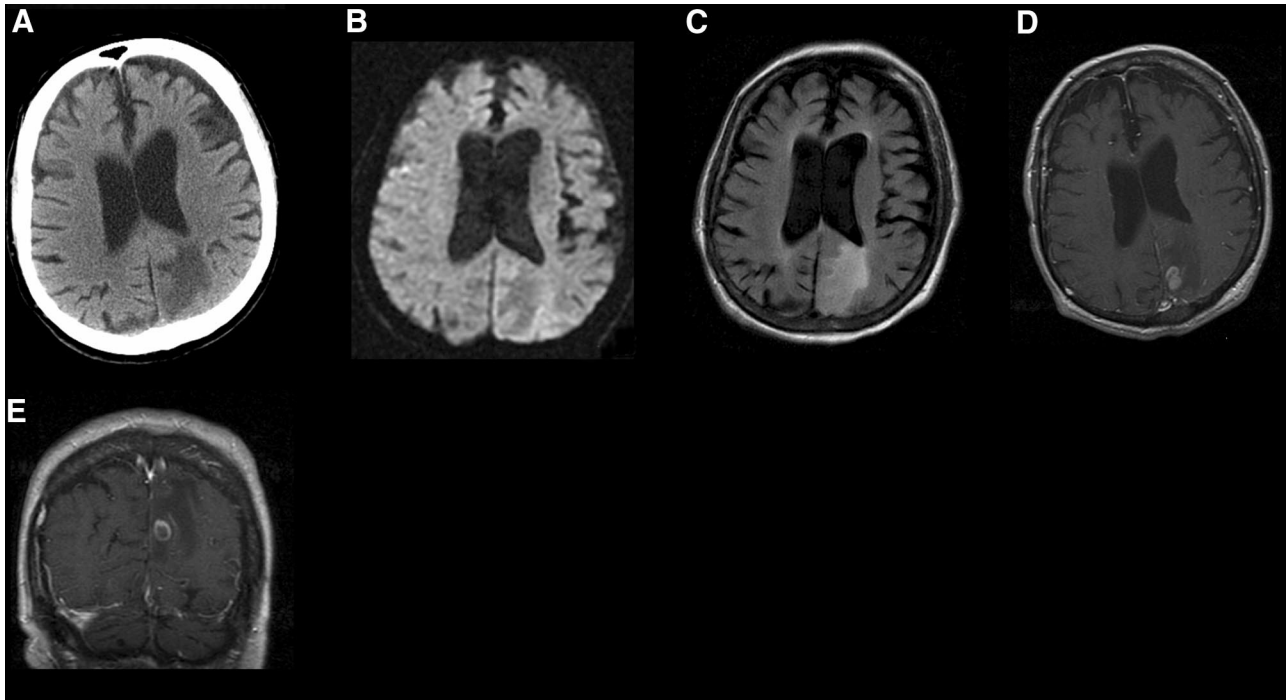


FIGURE 11. CNS metastasis.

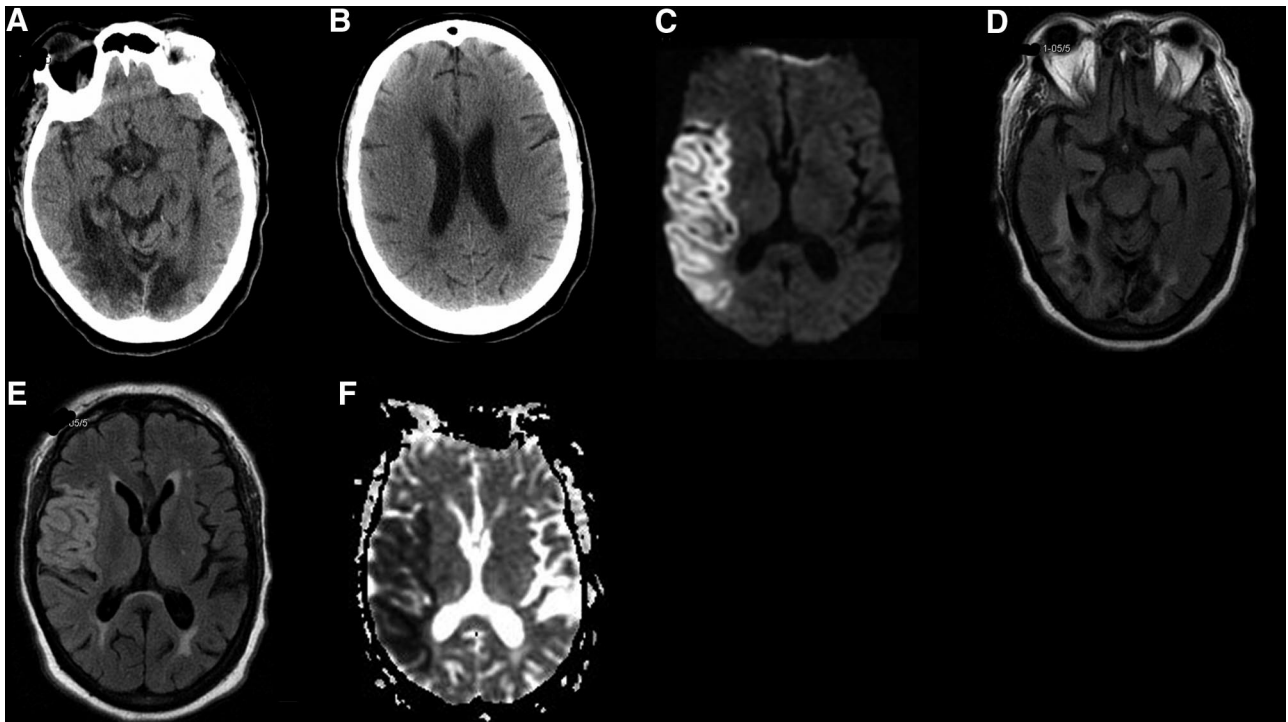


FIGURE 12. Bilateral PCA and right MCA infarction.

CE MRA can provide additional details of vascular anatomy and along with CTA show aspects of the cerebral vasculature previously only appreciated with conventional angiography. Additionally, both MRA and CTA play a role in

evaluating vessel patency to assess efficacy of thrombolytic therapy with arterial recanalization (cases 3 and 13).

CTP, which also requires iodinated contrast, but with possibly wider availability than MR perfusion can demon-

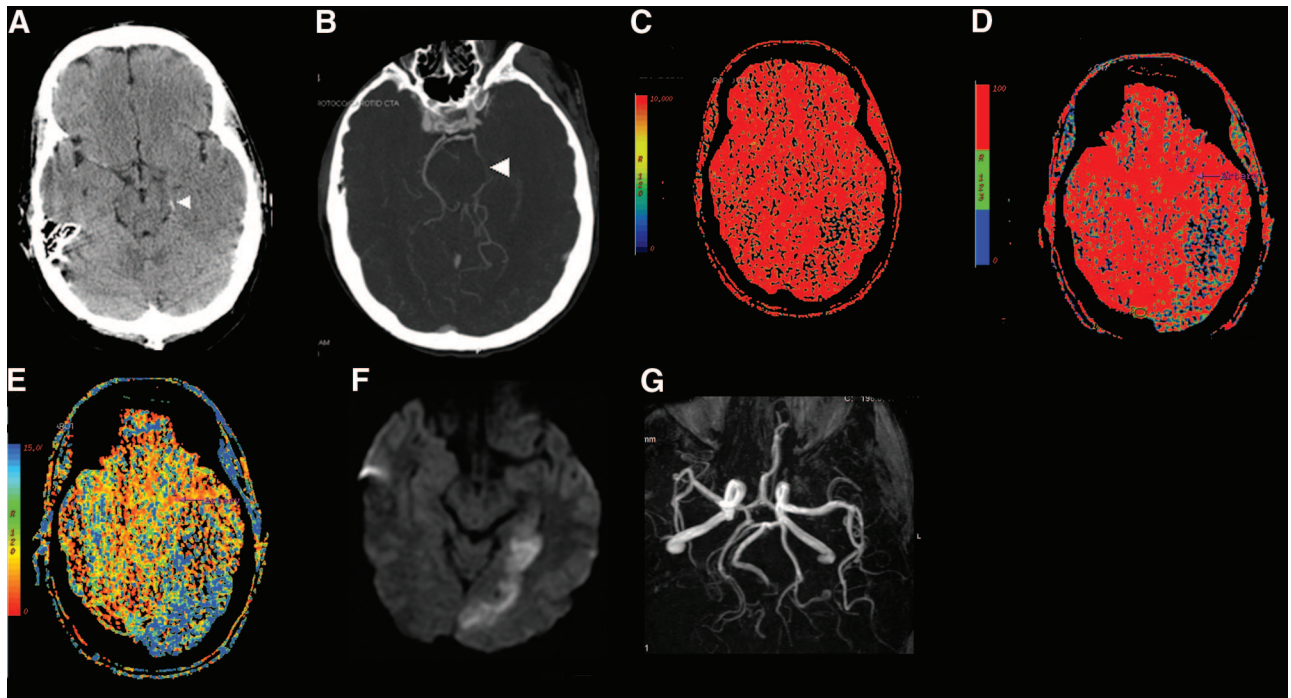


FIGURE 13. Left PCA infarction.

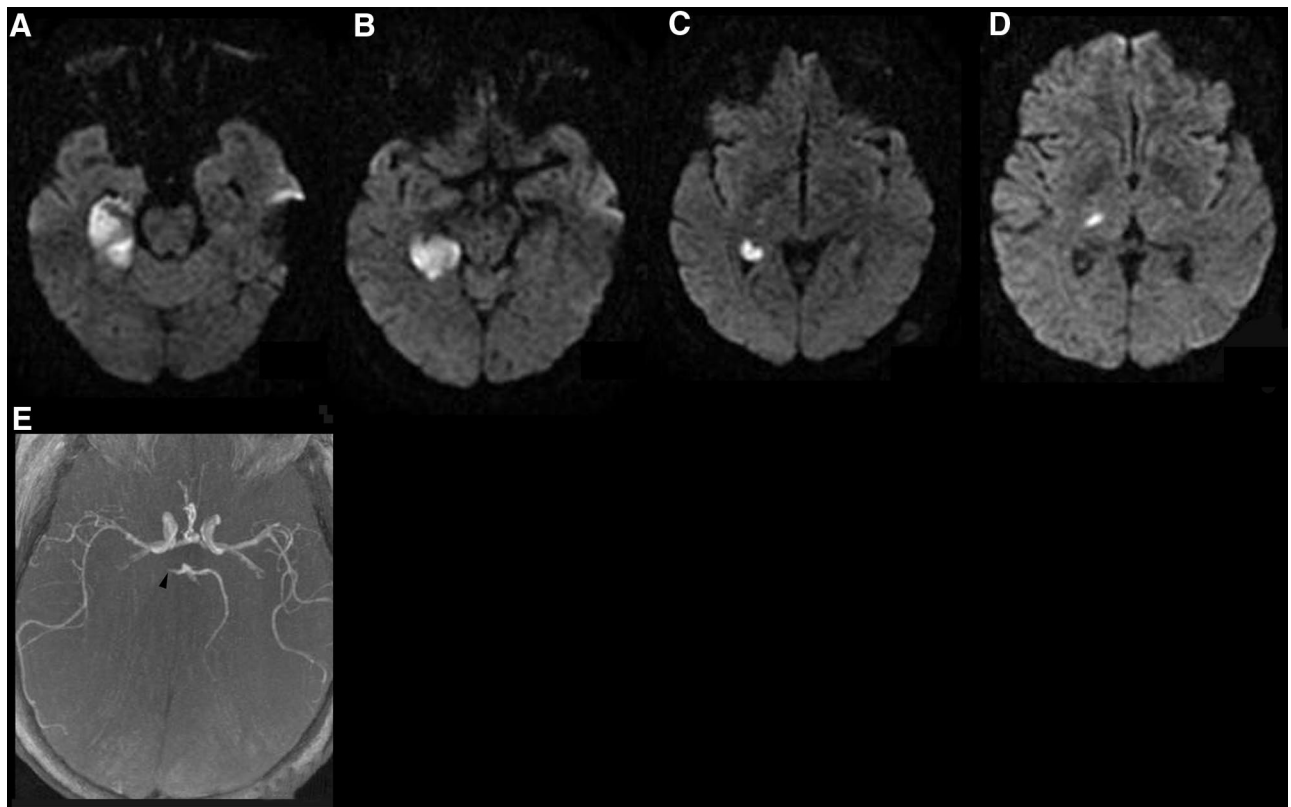


FIGURE 14. Right PCA infarction.

TABLE 2. Neuroimaging Features of PCA Infarction

CT*		
CT	Lesion morphology and location, <i>dense vessel sign (basilar and PCA)</i>	(Cases 3, 5, 8, and 13)
CTA	<i>Vessel anatomy, variants, occlusion, recanalization, dissection, plaque morphology, and wall calcification</i>	(Cases 3 and 13)
CTP	Evidence of infarct before CT visualization and <i>evidence of mismatch</i>	(Cases 13)
MR		
DWI	<i>Lesion morphology, location, extent, and age</i>	(Cases 1, 2, 5, 6, 8–10, 12, and 14)
MRA	Vessel anatomy, variants, occlusion, recanalization, and dissection, (<i>contrast enhanced more competitive with CTA</i>)	(Cases 2, 4, 7, 8, 13, and 14)
FLAIR	<i>Lesion morphology, location, extent, and age</i>	(Cases 10 and 12)
ADC	<i>Lesion age</i>	(Cases 10 and 12)
Enhanced	<i>Differential diagnosis of neoplasm, abscess, granulomatous disease, and others</i>	(Case 11)
Perfusion	<i>Evidence of mismatch</i>	

Italics indicate the preferred imaging modality (both italicized if no clear advantage).
*Indicates current preferred modality for acute stroke evaluation.⁹

strate not only changes of infarction before conventional CT but also provide information on salvageable brain, which represents a difference or “mismatch” between the infarct core and surrounding ischemic penumbra, and is thus helpful in deciding on thrombolytic therapy^{18,19} (case 13). A decrease in cerebral blood flow and prolongation of mean transit time vis-à-vis a lesser decrease in cerebral blood volume defines mismatch (case 13) and correlates with the potential efficacy of thrombolytic therapy.²⁰

A principle feature of DWI in stroke is its ability to detect infarction early, frequently within an hour of symptom onset and define the location and extent of infarction more accurately than CT (cases 1, 2, and 6). Despite these early changes with DWI, the CT triple study is favored (see above) as the modality of choice in evaluating acute stroke patients for thrombolytic therapy. Also, appreciation of lesion morphology with CT and more so with DWI can be helpful, such that the circular appearance frequently seen with thalamic infarction compared with the wedge shaped infarction of the cerebral hemisphere, a result of vertical versus horizontal vessel orientation, may avoid misdiagnosis of neoplasm, particularly with an isolated lesion (case 5). Further, utilization of DWI, FLAIR, and acquired diffusion coefficient MR sequences, can determine the acute, subacute, or chronic nature of the infarction (case 10). A patient diagnosed initially by CT as bilateral PCA infarction subsequently had DWI, acquired diffusion coefficient, and FLAIR images that demonstrated an unsuspected recent right MCA infarction whereas the PCA infarctions seen on CT were chronic (case 12). This case illustrates how determining infarct age can be helpful and also show how PCA infarction may be asymptomatic. Given their common origin from the basilar artery, infarction of the PCA occasionally is bilateral and may be associated with a syndrome of denial of blindness (Anton syndrome) or visual attention deficits with normal visual fields, optic ataxia and ocular apraxia (Balint syndrome) and lethargy if both thalami are involved (case 9).

At present, the use of thrombolytic and other interventional procedures for stroke involve the carotid, MCA, and

vertebrobasilar arteries where the potential neurologic disability justifies the cost and risk of the procedure.^{21,22} Although the same use guidelines and applications apply for PCA infarction,⁸ thrombolytic therapy, both IV and IA and other interventional procedures, are less well established, in part because the neurologic deficits may be more limited. Regarding isolated visual field defect from acute PCA infarction, currently there are no specific recommendations. However, considering the potential disability the consensus is to treat with IV tPA, weighing factors as patient age, comorbidities, and lifestyle. Hemiparesis, however, may be a prominent accompaniment of PCA infarction^{23,24} with sufficient stroke deficit to warrant IA thrombolytic or other interventional therapy. Interventional options should be considered specifically when hemiparesis, particularly in association with a visual field defect, is present in the context of a fetal-type PCA, where the anterior circulation is the involved vascular territory. As safety and efficacy of interventional procedures of the posterior circulation for vertebrobasilar artery disease become established, so to more distal intervention for the PCA territory infarction is a consideration, as recently reported with IA thrombolysis.²⁵

In a fetal-type PCA, the carotid system may be the main blood supply of the PCA. In this circumstance, MRA visualization of the carotid artery including the bifurcation in the neck and its contribution to the PCA can help correlate clinical features. Two patients with fetal-type PCA, one of whom had significant carotid bifurcation disease (case 4), demonstrate the superiority of CTA compared with MRA in this setting, and the second with a PCOM polar artery infarction of the anterior thalamus (case 8) illustrates the value of MRA in depicting vessel anatomy. A third patient who presented clinically as an MCA territory infarction with a negative admission CT, on follow-up CT showed a PCA infarction in association with an ipsilateral internal carotid artery occlusion on Doppler study that suggested a possible fetal-type PCA. A subsequent DWI and MRA showed no variant circulation, but rather a PCA occlusion with infarction that clinically mimicked MCA territory infarction (case 7).

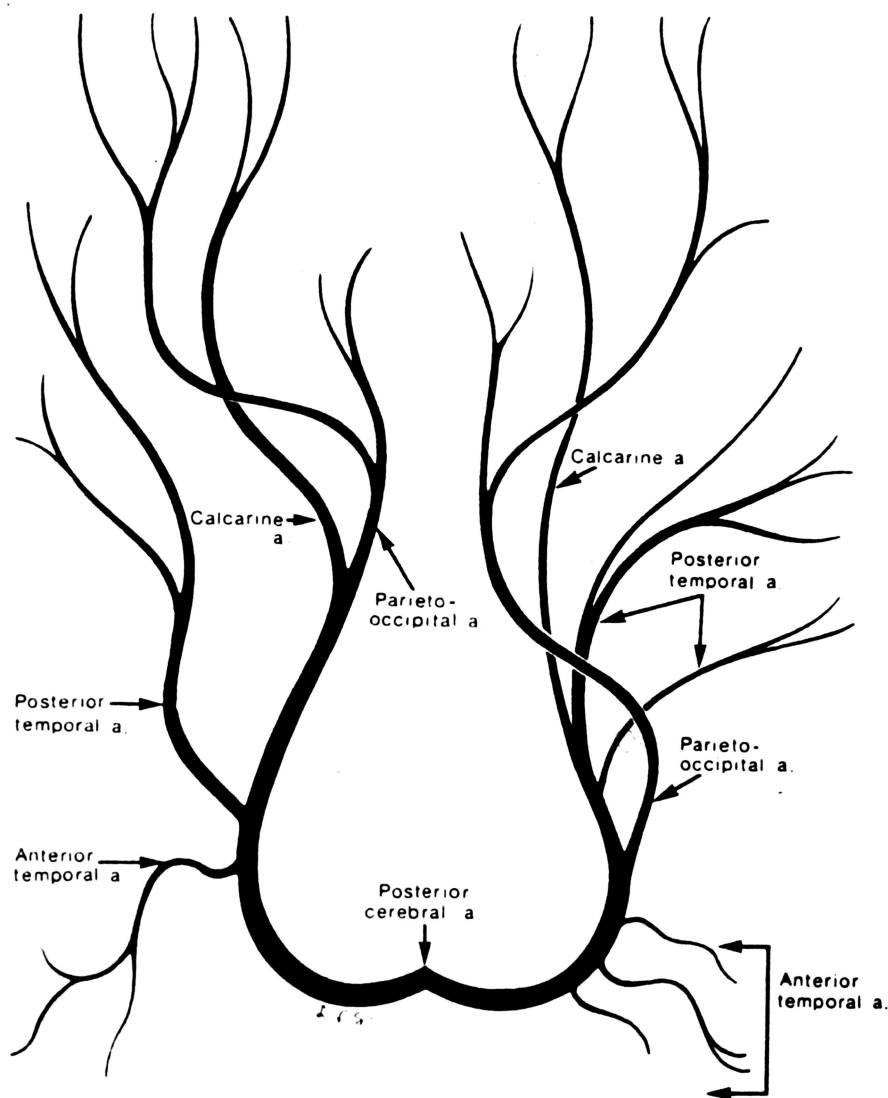


FIGURE 15. Cortical branches of PCA with common variations. Reprinted with permission from *Neuroradiology*.²⁶

On occasion, PCA infarction can mimic the clinical features of an MCA infarction particularly when hemiparesis is prominent, the result of peduncular²³ or more superficial or cortical PCA infarction.²⁴

Another unusual occurrence is isolated infarction of the anterior temporal artery (ATA) the most proximal cortical branch of the PCA (case 14). The ATA arises either from a single trunk or as multiple branches from the proximal ambient segment of the PCA and occasionally from the posterior temporal artery (Fig. 15). The artery extends laterally and anteriorly beneath the hippocampal gyrus to supply the inferior aspect of the anterior portion of the anterior temporal lobe.²⁶ The clinical deficits of our patient were most likely not related to the ATA infarct but due to the small right thalamic infarction involving the thalamostriate arteries.

Among conditions with a predilection for the PCA territory that can mimic infarction are infections such as progressive multifocal leukoencephalopathy and toxoplasmo-

Among conditions with a predilection for the posterior cerebral artery territory that can mimic infarction are progressive multifocal leukoencephalopathy and toxoplasmosis as well as reversible posterior leukoencephalopathy and thalamic infarction due to deep cerebral vein thrombosis.

sis as well as reversible posterior leukoencephalopathy and thalamic infarction due to deep cerebral vein thrombosis. Neoplasms both primary and metastatic may mimic PCA infarction and MR features, more than CT, are helpful in the

differential diagnosis by demonstrating vasogenic edema and contrast enhancement (case 11). Also, subacute infarction of the PCA may appear as an arterial venous malformation mimic on contrast enhanced CT.²⁷

ACKNOWLEDGMENT

The author wishes to acknowledge Kevin Hagan for technical assistance with image preparation.

REFERENCES

1. Caplan LR, Bogousslavsky J. Posterior cerebral artery syndromes. In: Ginsberg MD, Bogousslavsky J, eds. *Cerebrovascular Disease: Pathology, Diagnosis, and Management*. Vol. 2. Boston: Blackwell Science; 1998:1028.
2. Binder JR, Mohr JP. Posterior cerebral artery disease. In: Mohr JP, Choi DW, Grotta JC, et al. eds. *Stroke: Pathophysiology, Diagnosis, and Management*. 4th ed. New York: Churchill Livingstone; 2004:167.
3. Brandt T, Steinke W, Andreas T, et al. Posterior cerebral artery territory infarcts: clinical features, infarct topography, causes and outcome. *Cerebrovasc Dis*. 2000;10:170–182.
4. Tatu L, Moulin T, Bogousslavsky J, et al. Arterial territories of the human brain. *Neurology*. 1998;50:1699–1708.
5. Phan TG, Fong AC, Donnan G, et al. Digital map of posterior cerebral artery infarcts associated with posterior cerebral artery trunk and branch occlusion. *Stroke*. 2007;38:1805–1811.
6. Caplan LR. *Posterior Circulation Disease: Clinical Findings, Diagnosis, and Management*. Boston, MA: Butterworth-Heinemann; 1996.
7. Brandt T, Thie A, Caplan LR, et al. Infarkte in Versorgungsgebiet der A. cerebri posterior. *Nervenarzt*. 1995;66:267–274.
8. Hill MD. Posterior cerebral artery stroke. *e-medicine*. 2005.
9. Bowen BC, Truwit CL. Controversies: MRA vs CTA in the evolution of neurovascular disease. *Radiology*. In press.
10. Wahlgren N, Ahmed N, Davalos A, et al. Thrombolysis with alteplase for acute ischemic stroke & the Safe implementation of thrombolysis in stroke—monitoring study (SITS-MOST): an observational study. *Lancet*. 2007;369:275–282.
11. Adams HP Jr, Del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Stroke*. 2007;38:1655–1711.
12. Krings T, Noelchen D, Mull M, et al. The hyperdense posterior cerebral artery sign. *Stroke*. 2006;37:399–403.
13. Choi CG, Lee DH, Lee JH, et al. Detection of intracranial arteriosclerotic stenosis-occlusive disease with 3D time-of-flight magnetic resonance angiography with sensitivity encoding at 3T. *Am J Neuroradiol*. 2007;28:439–446.
14. Lev MH, Farkas J, Rodriguez VR, et al. CT angiography in the rapid triage of patients with hyperacute stroke to intraarterial thrombolysis: accuracy in the detection of large vessel thrombus. *J Comput Assist Tomogr*. 2001;25:520–528.
15. Caplan LR, Estol CJ, Massaro AR. Dissection of the posterior cerebral arteries. *Arch Neurol*. 2005;62:1138–1143.
16. Chalela JA, Kidwell CS, Nentwich LM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. *Lancet*. 2007;369:293–298.
17. Wintermark M, Meuli R, Browaeys P, et al. Comparison of CT perfusion on angiography and mri in selecting stroke patients for acute treatment. *Neurology*. 2007;68:694–697.
18. Parsons MW, Pepper EM, Chan V, et al. Perfusion computed tomography: prediction of final infarct extent and stroke outcome. *Ann Neurol*. 2005;58:672–679.
19. Parsons MW, Pepper EM, Bateman GA, et al. Identification of the penumbra and infarct core on hyperacute noncontrast and perfusion CT. *Neurology*. 2007;68:730–736.
20. Zaidat OO, Suarez JI, Sunshine JL, et al. Thrombolytic therapy of acute ischemic stroke: correlation of angiographic recanalization with clinical outcome. *AJNR Am J Neuroradiol*. 2005;26:880–884.
21. Smith WS, Sung G, Starkman S, et al. Safety and efficacy of mechanical embolectomy in acute ischemic stroke, results of the merci trial. *Stroke*. 2005;36:1432–1440.
22. Schellinger PD, Hacke W. Intra-arterial thrombolysis is the treatment of choice for basilar thrombosis: pro. *Stroke*. 2006;37:2436–2437.
23. Chambers BR, Brooder RJ, Donnan GA. Proximal posterior cerebral artery occlusion simulating middle cerebral artery occlusion. *Neurology*. 1991;41:385–390.
24. Maulaz AB, Bezerra DC, Bogousslavsky J. Posterior cerebral artery infarction from middle cerebral artery infarction. *Arch Neurol*. 2005;62:938–941.
25. Kostanian V, Cramer SC. Artery of percheron thrombolysis. *AJNR Am J Neuroradiol*. 2007;28:870–871.
26. Margolis MT, Newton TH, Hoyt WF. Cortical branches of the posterior cerebral artery. Anatomic-radiologic correlation. *Neuroradiology*. 1971;2:127–135.
27. Henrikson GC, Patel DV. Enhancing cerebral infarction simulating arteriovenous malformation on computed tomography. *J Comput Assist Tomogr*. 1985;9:502–506.