Section 1 Brain

Chapter 1

Cerebrovascular diseases

Yang Tang, Xinli Du, Sugoto Mukherjee, and Max Wintermark

Case 1.1

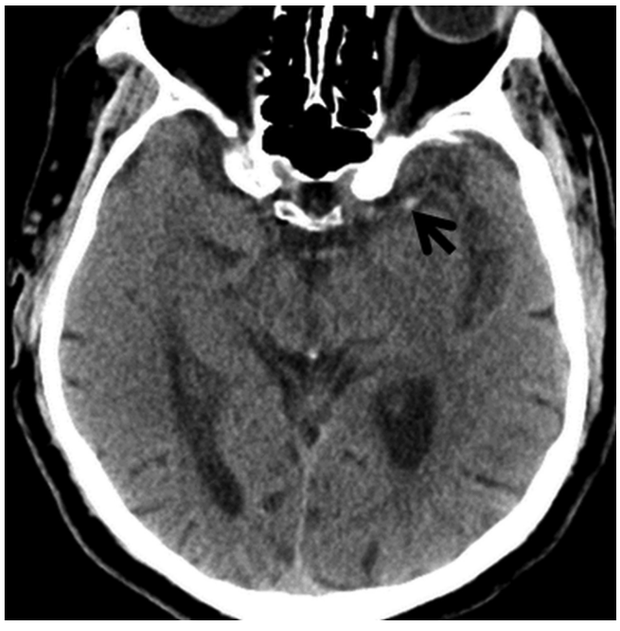

Figure 1.1.1 (patient 1)

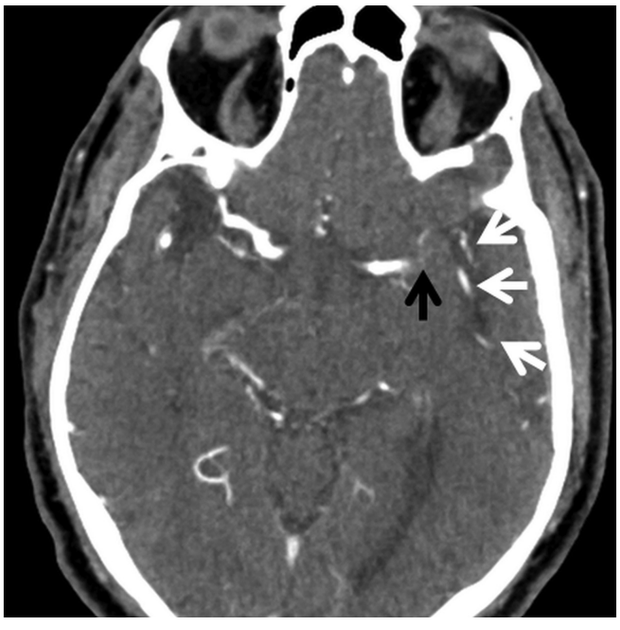

Figure 1.1.2 (patient 1)

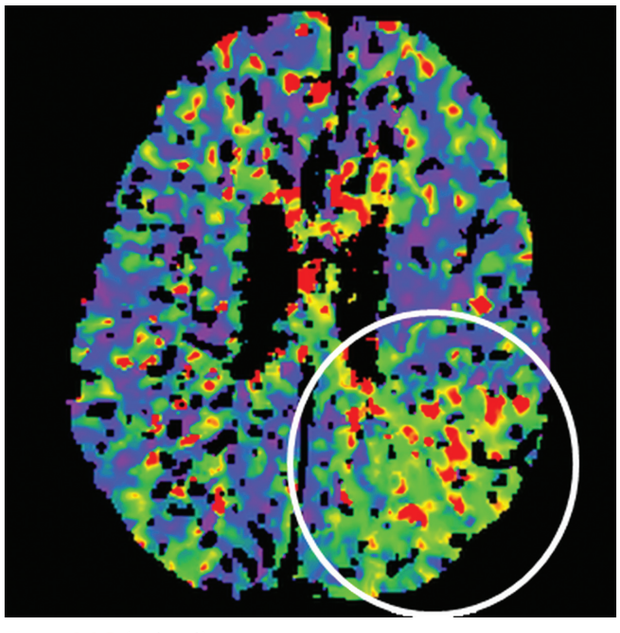

Figure 1.1.3 (patient 1)

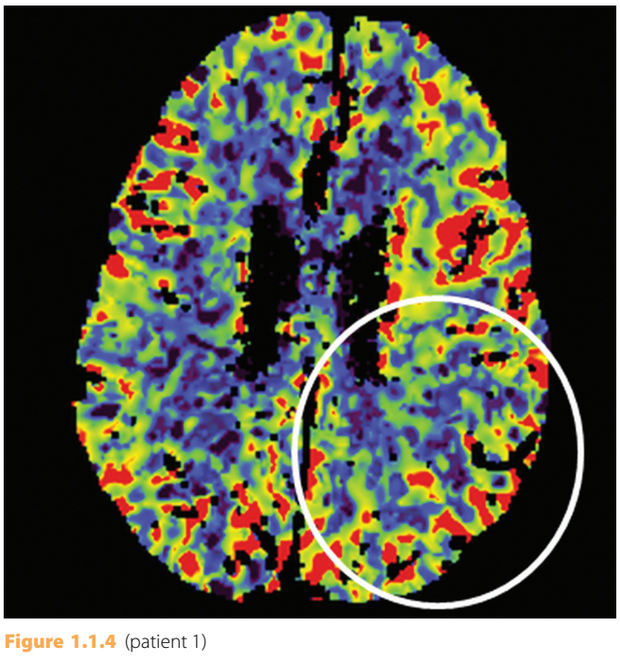

Figure 1.1.4 (patient 1)

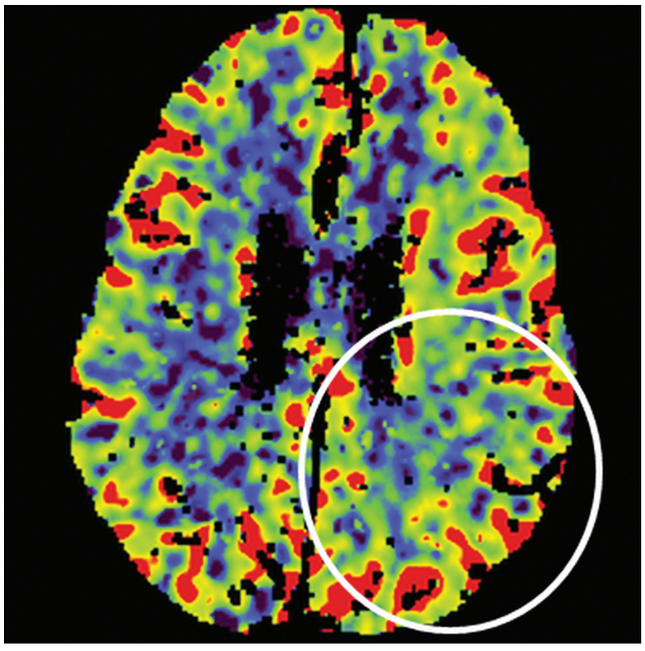

Figure 1.1.5 (patient 1)

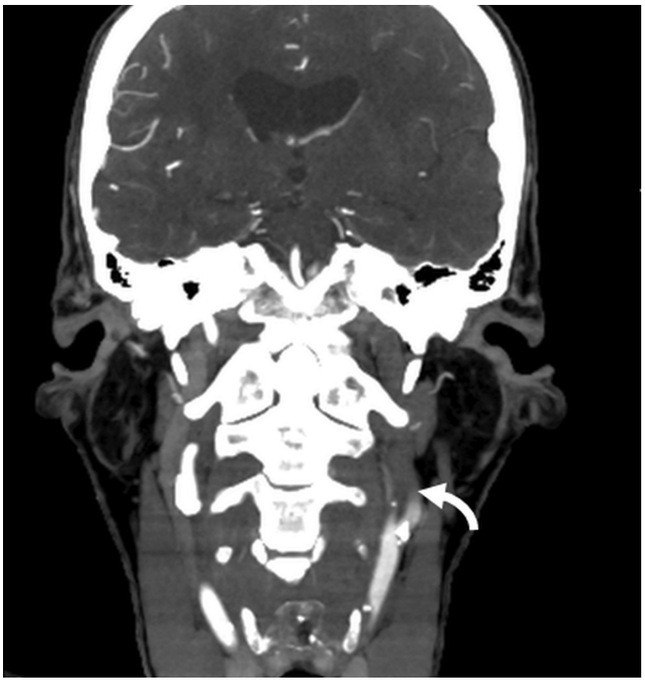

Figure 1.1.6 (patient 2)

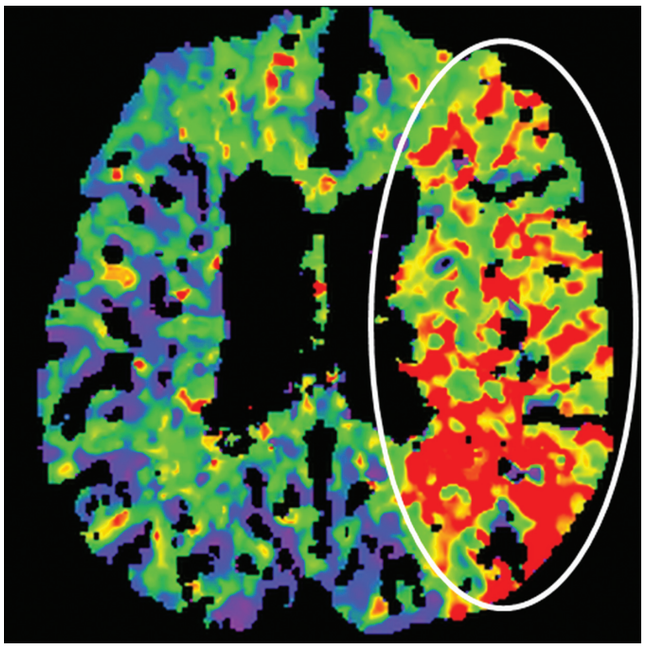

Figure 1.1.7 (patient 2)

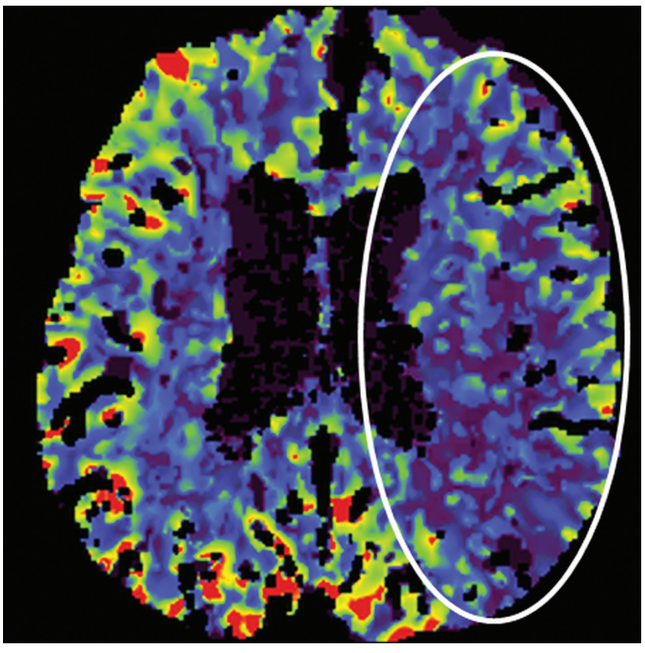

Figure 1.1.8 (patient 2)

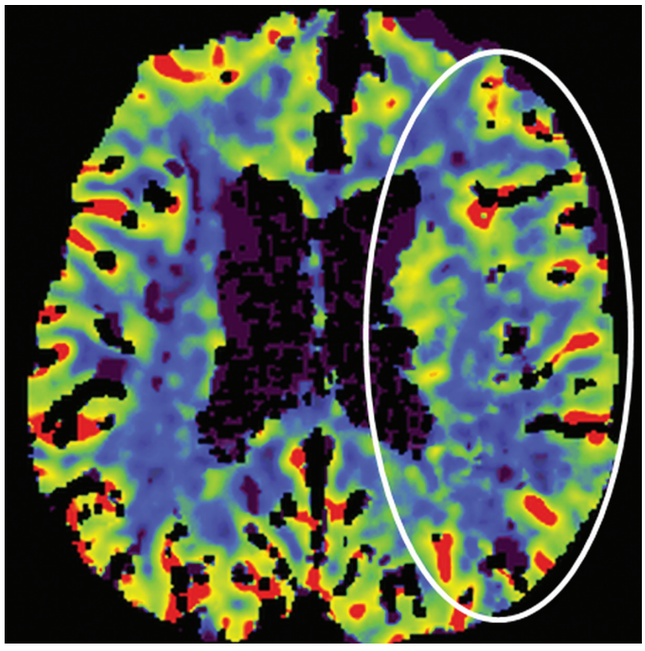

Figure 1.1.9 (patient 2)

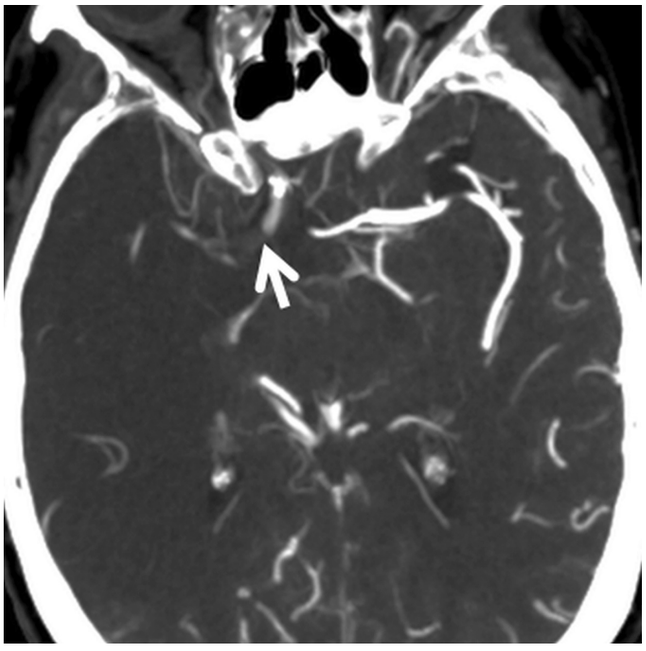

Figure 1.1.10 (patient 3)

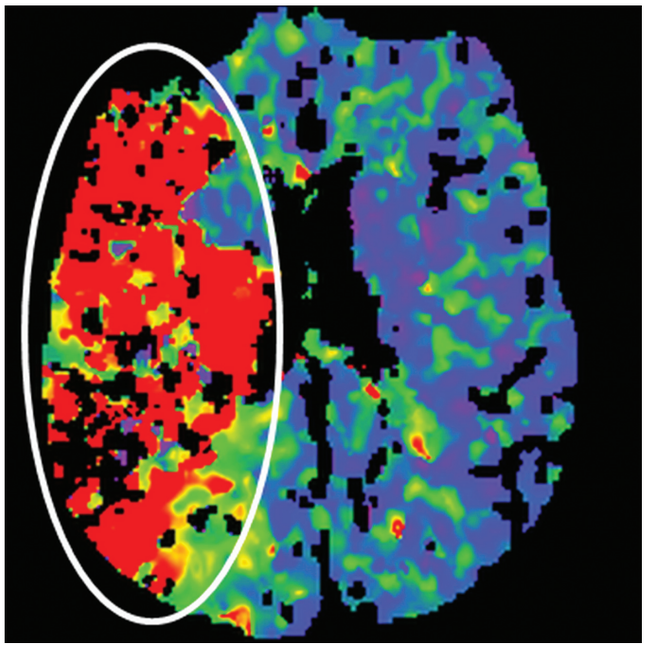

Figure 1.1.11 (patient 3)

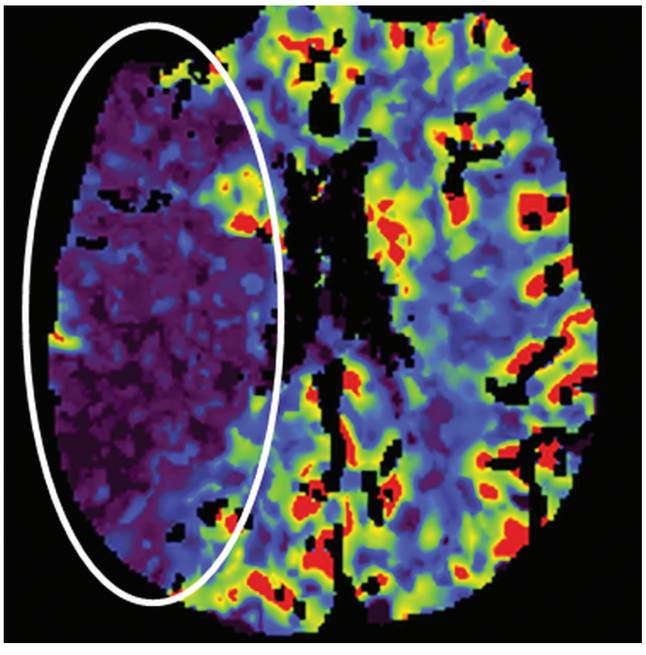

Figure 1.1.12 (patient 3)

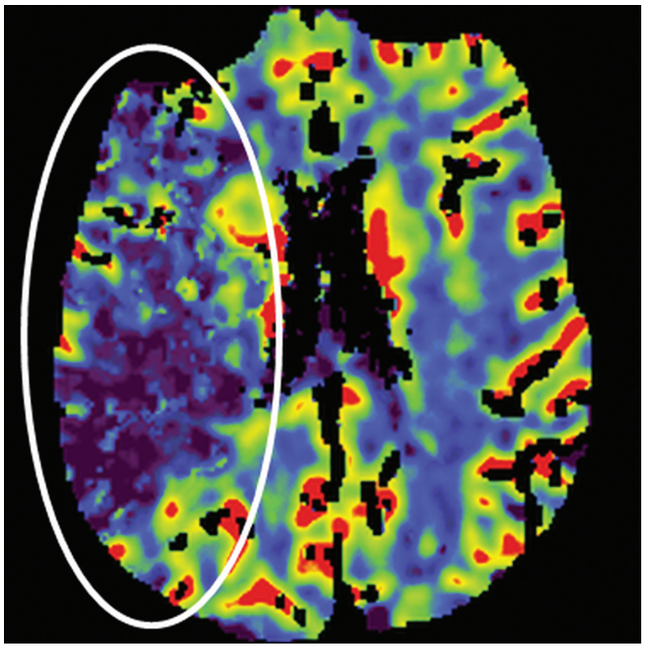

Figure 1.1.13 (patient 3)

History

Patient 1: 55-year-old man presents with acute onset of right-sided weakness.

Patient 2: 43-year-old man with recent cardiac surgery presents with sudden right-sided weakness and aphasia.

Patient 3: 62-year-old woman with history of atrial fibrillation develops sudden left-sided weakness.

Findings

Patient 1

Figure 1.1.1 NECT of the head shows a focal hyperdensity in the distal M1 segment of left MCA (arrow), suggestive of an intraluminal thrombus. No large infarction is identified.

Figure 1.1.2 Axial CTA of the head confirms an intraluminal thrombus with acute cutoff of distal M1 segment (black arrow) but good flow reconstitution in the M2 branches (white arrows).

Figures 1.1.3–1.1.5 CT perfusion images show mildly prolonged time to drain (TTD) (Fig. 1.1.3), preserved cerebral blood flow (CBF) (Fig. 1.1.4), and preserved cerebral blood volume (CBV) (Fig. 1.1.5) in the left parietal/temporal region (circle) of the posterior left MCA distribution, consistent with benign oligemia. No large ischemic infarct or tissue at risk is identified. Patient received intravenous tPA and had significant clinical improvement. Follow-up head CT in 24 hours (not shown) did not show large territory infarction or hemorrhage.

Patient 2

Figure 1.1.6 Coronal CTA of the head and neck shows abrupt occlusion of the proximal left ICA (curved white arrow) due to an intraluminal thrombus. Diminished flow is observed in the left cerebral hemisphere compared to the right.

Figures 1.1.7–1.1.9 CT perfusion images show moderately prolonged TTD (Fig. 1.1.7), decreased CBF (Fig. 1.1.8), and preserved CBV (Fig. 1.1.9) in the entire left MCA territory (circle), consistent with tissue at ischemic risk. This patient underwent emergent mechanical thrombectomy and had good clinical outcome.

Patient 3

Figure 1.1.10 Axial CTA of the head shows an intraluminal thrombus in the distal supraclinoid right ICA with abrupt cutoff (white arrow). The entire right MCA territory shows markedly diminished vascularity.

Figures 1.1.11–1.1.13 CT perfusion images show markedly prolonged TTD (Fig. 1.1.11), decreased CBF (Fig. 1.1.12), and CBV (Fig. 1.1.13) in the entire right MCA territory (circle), consistent with core infarction. This patient died in a few days from massive cerebral edema leading to herniation.

Diagnosis

Patient 1: left MCA thrombus with benign oligemia in the left MCA territory.

Patient 2: left ICA thrombus with tissue at ischemic risk in the left MCA territory.

Patient 3: right ICA thrombus with large right MCA infarction.

Differential diagnosis

- None.

Key points

- There are three major ischemic stroke subtypes: large-artery atherosclerotic infarctions, cardioembolic infarctions, and lacunar infarctions.
- The concepts of core, penumbra, and benign oligemia:
  - The infarcted tissue with irreversible cell death is defined as the "core."
  - Functionally impaired yet still viable and salvageable tissue surrounding the core is commonly termed "penumbra" or "tissue at risk." The penumbra will progress to infarction unless timely reperfusion occurs either spontaneously or as a result of treatment, and its fate largely depends on the severity/duration of ischemia and the availability of collateral circulation.
  - Further away from the ischemic core, there is tissue with "benign oligemia," which is hypoperfused yet functionally intact. This tissue will survive even if reperfusion does not occur. It is important to distinguish benign oligemia from true penumbra to avoid the overestimation of tissue at risk.
- Acute stroke imaging:
  - NECT is the initial modality, to exclude intracranial hemorrhage and other etiologies that may simulate acute stroke, such as mass or hydrocephalus. Although not sensitive, CT signs of early ischemia include hyperdense vessels, loss of gray–white differentiation, parenchymal hypodensity, and gyral swelling.

Hypodensity over one-third of MCA territory is commonly considered a contraindication for thrombolysis. Alberta Stroke Program Early CT Score (ASPECTS) has been developed to standardize the detection and reporting of the extent of early ischemia using a 10-point scale.

- CTA is the modality of choice to evaluate the extracranial/intracranial vascular anatomy and detect the site of vascular occlusion. In addition, it can provide valuable information on the status of collateral circulation, which is a major factor determining the rate of core expansion and the patient's outcome.
- Currently, there is considerable debate regarding the utility of penumbra imaging beyond initial NECT and CTA. A recent study found no benefit of penumbra imaging in selecting patients who would differentially benefit from endovascular therapy. Regardless, advanced imaging with either CT perfusion or MRI/MR perfusion has been a focus of intense research investigation and has been adopted by many centers as part of their stroke imaging protocol.
- MRI with diffusion-weighted imaging is the standard of infarct core characterization. An infarct volume of less than 70 ml has been suggested as a threshold for selecting patients for endovascular treatment, as patients with higher infarct volumes have unfavorable outcomes regardless of treatment. MR perfusion performed concurrently can help estimate the size of penumbra. However, the limited availability of MRI scanners and individual safety contraindications significantly restrict its use in the emergent setting.
- CT perfusion is an attractive alternative because of its wider availability, fewer contraindications, and potential for quantitative assessment. It should be noted that there is a critical need for standardization, as interoperator, intraoperator, and intervendor software differences can greatly influence the results, and the published parameter thresholds are not easily transferrable between institutions.
- In our institution, CT perfusion studies are interpreted with the following general guidelines, as illustrated in the cases and Table 1.1.1. Cerebral blood volume (CBV): the marker for core infarct. Cerebral blood flow (CBF): the marker for penumbra. Time to drain (TTD) and mean transit time (MTT): the markers for oligemia or hypoperfusion.

Table 1.1.1 Interpretation of CT perfusion in ischemic stroke

	MTT/TTD	CBF	CBV
Benign oligemia	↑	normal	normal or ↑
Penumbra	↑↑	↓	Normal or ↓
Core	↑↑↑	↓↓	↓↓

CBF, cerebral blood flow; CBV, cerebral blood volume; MTT, mean transit time; TTD, time to drain.
↑, mildly elevated; ↑↑, moderately elevated; ↑↑↑, markedly elevated.

- Treatment: the utility of intravenous tPA thrombolysis within the first 4.5 hours of stroke onset has been well established, while the risk of intracranial hemorrhage associated with intravenous tPA beyond the 4.5-hour window outweighs the benefit. Endovascular treatment using intra-arterial thrombolysis or mechanical embolectomy beyond the 4.5-hour window is still investigational.

Further reading

Kidwell CS, Jahan R, Gornbein J, Alger JR, Nenov V, Ajani Z, *et al.* A trial of imaging selection and endovascular treatment for ischemic stroke. *N Engl J Med* 2013; 368(10): 914–923.

Kucinski T, Naumann D, Knab R, Schoder V, Wegener S, Fiehler J, *et al.* Tissue at risk is overestimated in perfusion-weighted imaging: MR imaging in acute stroke patients without vessel recanalization. *AJNR Am J Neuroradiol* 2005; 26(4): 815–819.

Pexman JH, Barber PA, Hill MD, Sevick RJ, Demchuk AM, Hudon ME, *et al.* Use of the Alberta Stroke Program Early CT Score (ASPECTS) for assessing CT scans in patients with acute stroke. *AJNR Am J Neuroradiol* 2001; 22(8): 1534–1542.

Souza LC, Yoo AJ, Chaudhry ZA, Payabvash S, Kemmling A, Schaefer PW, *et al.* Malignant CTA collateral profile is highly specific for large admission DWI infarct core and poor outcome in acute stroke. *AJNR Am J Neuroradiol* 2012; 33(7): 1331–1336.

Yoo AJ, Verduzco LA, Schaefer PW, Hirsch JA, Rabinov JD, Gonzalez RG. MRI-based selection for intra-arterial stroke therapy: value of pretreatment diffusion-weighted imaging lesion volume in selecting patients with acute stroke who will benefit from early recanalization. *Stroke* 2009; 40(6): 2046–2054.

Zhu G, Michel P, Zhang W, Wintermark M. Standardization of stroke perfusion CT for reperfusion therapy. *Transl Stroke Res* 2012; 3(2): 221–227.

Case 1.2

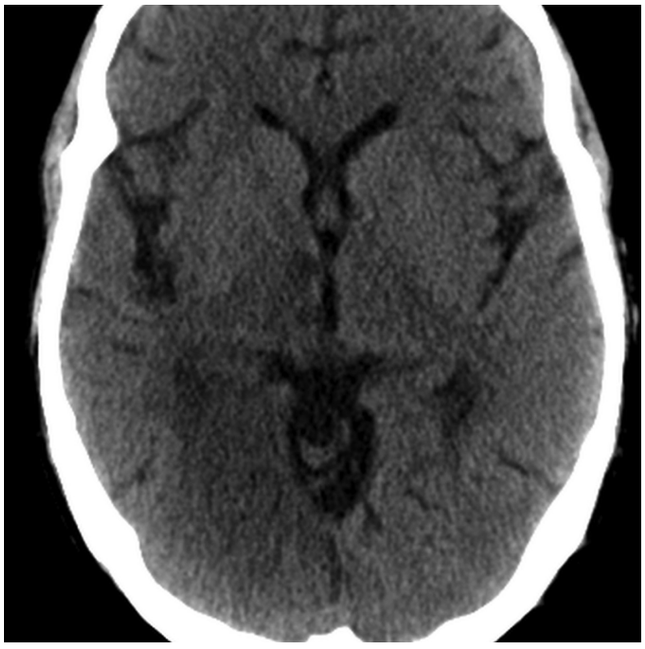

Figure 1.2.1

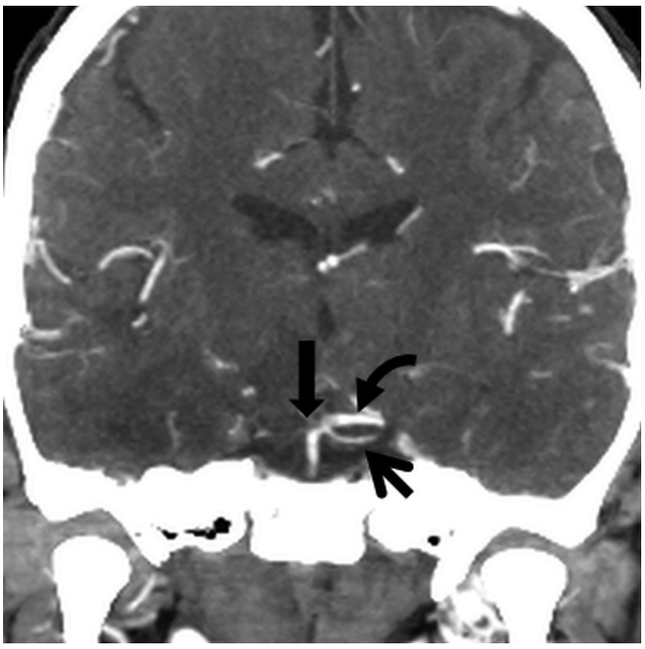

Figure 1.2.2

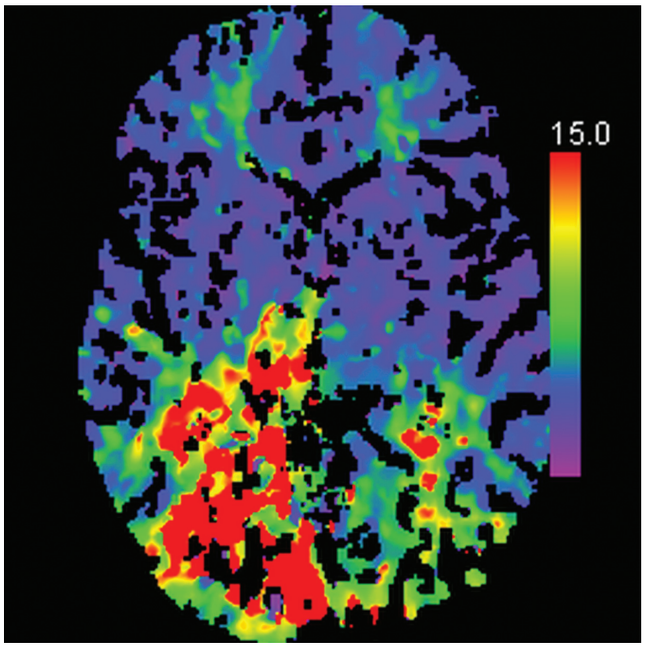

Figure 1.2.3

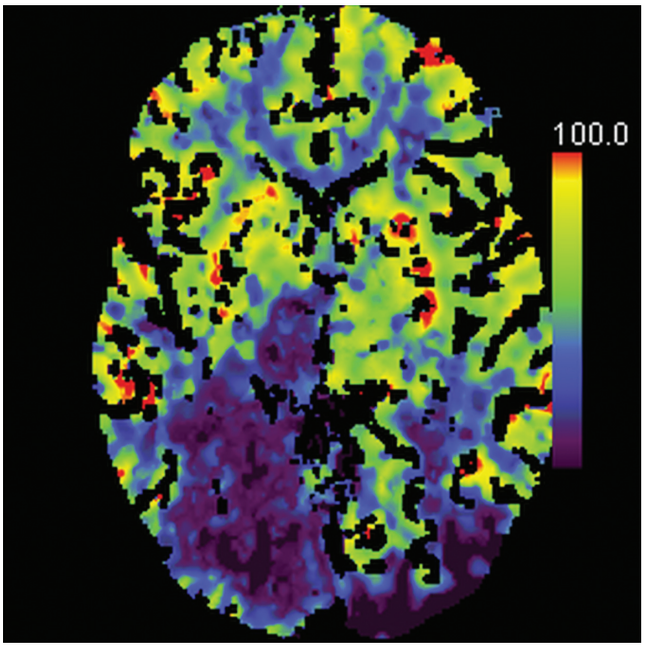

Figure 1.2.4

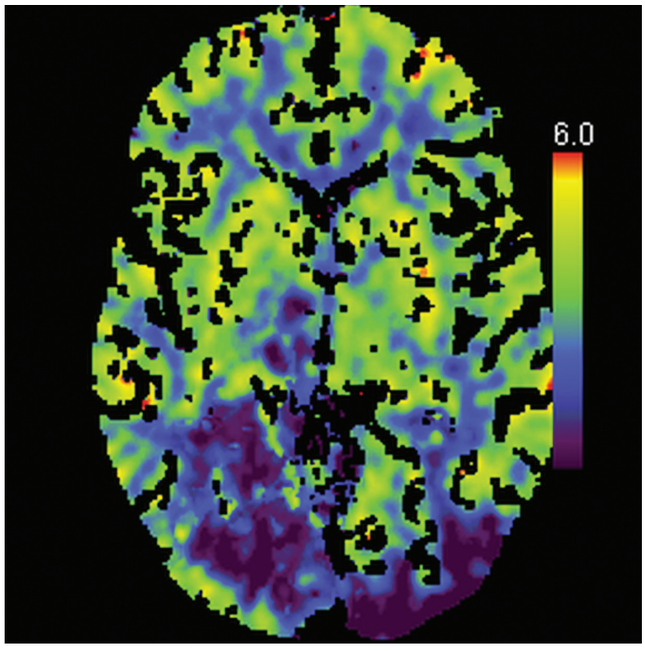

Figure 1.2.5

History

50-year-old man with history of atrial fibrillation is found unresponsive for an unknown duration.

Findings

Figure 1.2.1 NECT shows areas of low attenuation in the right thalamus and occipital lobe, consistent with acute infarction.

Figure 1.2.2 Coronal CTA of the head shows a filling defect in the distal basilar artery (block arrow), consistent with a thrombus. There is occlusion of the origins of the right posterior cerebral artery (PCA) and superior cerebellar artery (SCA). The proximal left PCA (curved arrow) and SCA (arrow) are patent.

Figures 1.2.3–1.2.5 CT perfusion images demonstrate prolonged TTD (Fig. 1.2.3), decreased CBF (Fig. 1.2.4), and decreased CBV (Fig. 1.2.5) in the right thalamus and bilateral occipital lobes, right greater than left, consistent with ischemic infarction in the bilateral PCA distributions. Note that the left PCA infarction is not yet obvious on NECT. Additional images (not shown) also revealed infarctions of superior cerebellum in the SCA distribution.

Diagnosis

Distal basilar thrombosis.

Differential diagnosis

- The diagnosis is usually straightforward, with characteristic vascular distribution and filling defects in the basilar top on CTA/MRA. Occasionally, if only thalami are involved, the following entities should be considered:
  - Artery of Percheron infarction.
  - Venous thrombosis of internal cerebral veins.
  - Hypoxic–ischemic encephalopathy.
  - Wernicke's encephalopathy.

Key points

- "Top of basilar" syndrome refers to thromboembolic occlusion of the distal basilar artery and its terminal branches including posterior cerebral arteries and superior cerebellar arteries, leading to ischemic infarction of the above vascular territories, including the thalami, occipital lobes, rostral midbrain, and superior cerebellum.
- Clinically, patients present with acute visual, oculomotor, and behavioral symptoms, while motor symptoms may not be obvious.
- Imaging findings:
  - CT: may be normal in the early stage and show hypodensity/edema if infarction occurs. Hyperdense embolus/thrombus in the distal basilar artery may be seen in some cases.
  - CTA/MRA/DSA: thrombus/embolus the distal basilar artery. It is also important to assess the patency of PCA and SCA.
  - CT or MRI perfusion: may add further information on the status of affected vascular territories (infarction versus viable tissue at ischemic risk), and select patients for endovascular intervention.
  - MRI: diffusion restriction if infarction occurs.
- It is associated with poor prognosis, frequently leading to death or "locked in" syndrome.
- Treatment: endovascular recanalization is the only treatment that may improve clinical outcomes, and it must be performed early.

Further reading

Barkhof F, Valk J. "Top of the basilar" syndrome: a comparison of clinical and MR findings. *Neuroradiology* 1988; 30(4): 293–298.

Davis SM, Donnan GA. Basilar artery thrombosis: recanalization is the key. *Stroke* 2006; 37(9): 2440.

Case 1.3

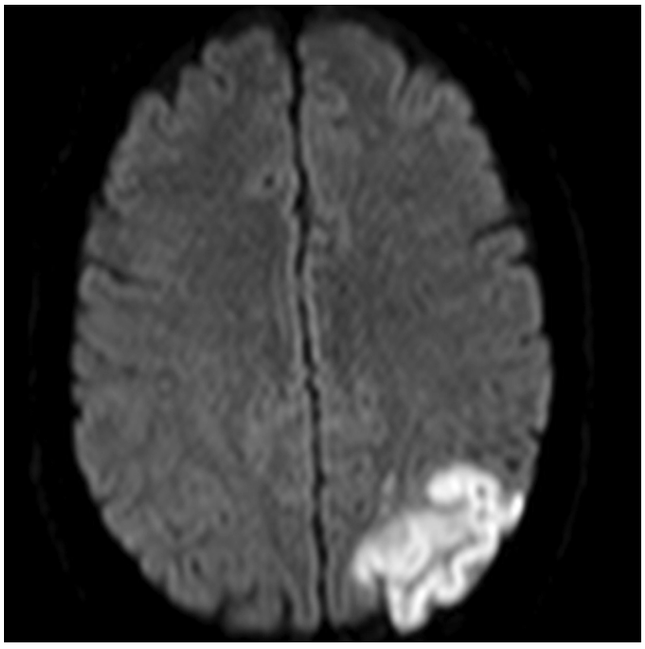

Figure 1.3.1

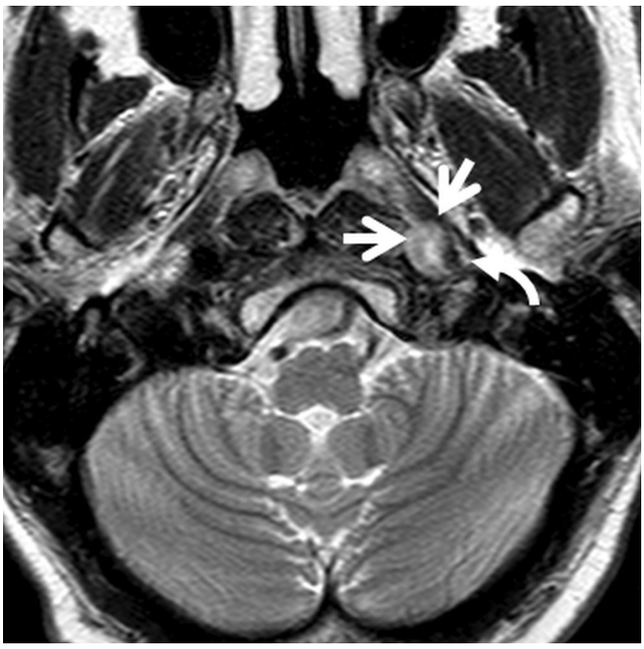

Figure 1.3.2

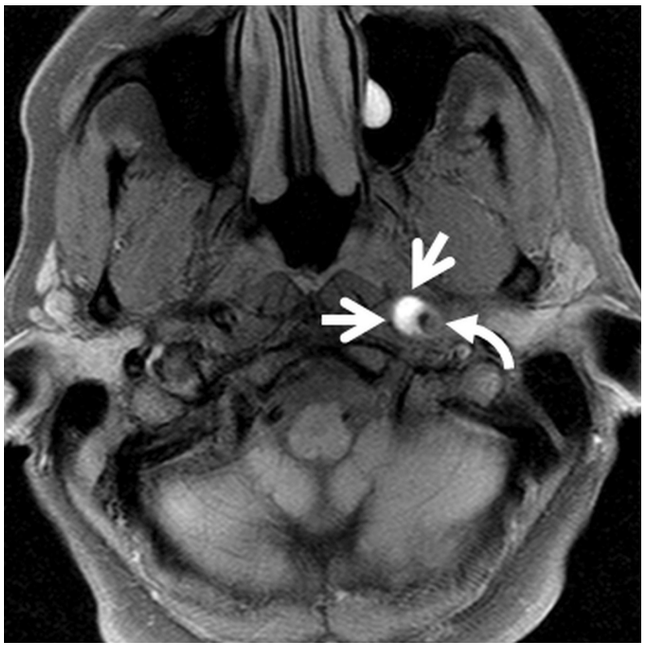

Figure 1.3.3

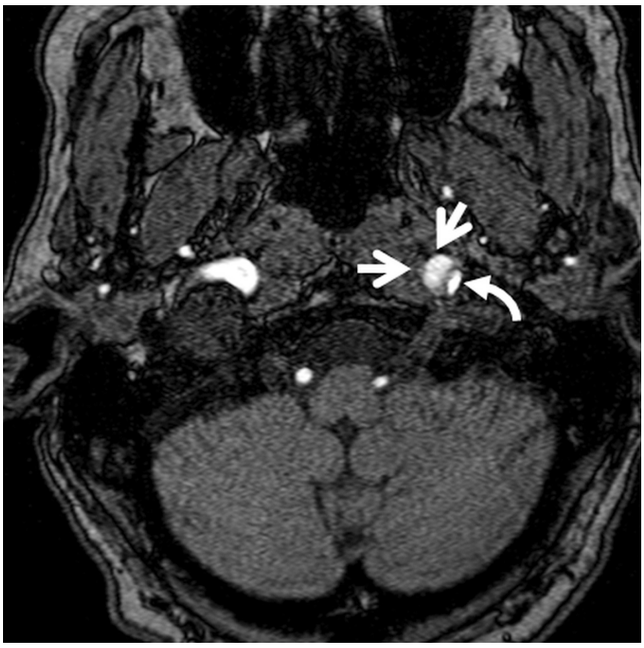

Figure 1.3.4

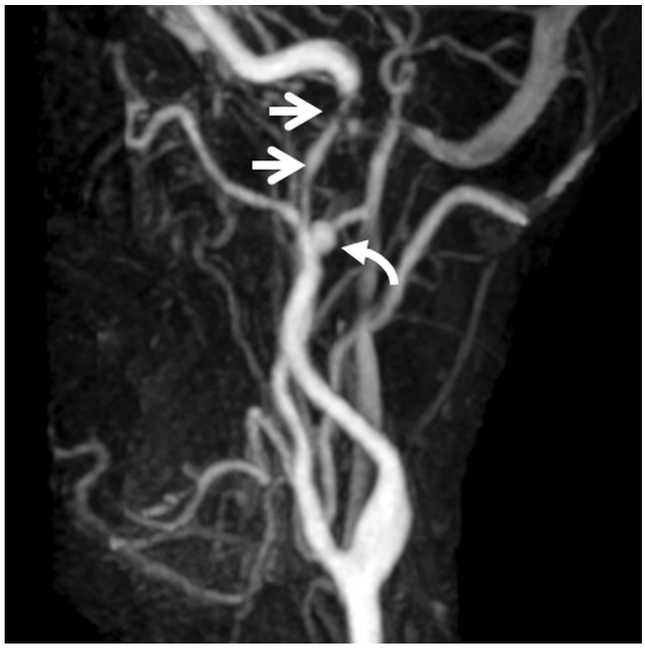

Figure 1.3.5

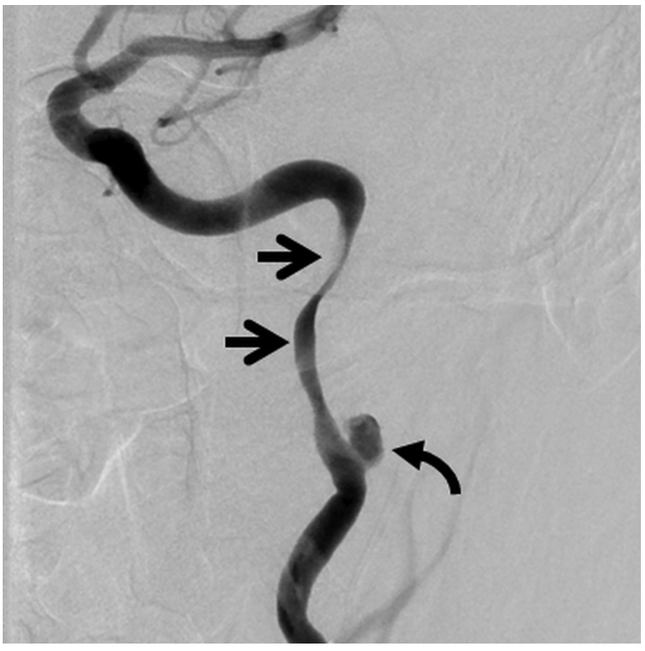

Figure 1.3.6

History

44-year-old man presents with sudden right-arm weakness and speech difficulty.

Findings

Figure 1.3.1 Axial DWI shows diffusion restriction in left parietal lobe, consistent with acute ischemic infarction.

Figures 1.3.2, 1.3.3 Axial T2 (Fig. 1.3.2) and axial T1 with fat saturation (Fig. 1.3.3) demonstrate a crescent of T2 and T1 hyperintensity (arrows) surrounding a diminished left ICA flow void (curved arrow) at the skull base, consistent with dissection with an intramural hematoma.

Figure 1.3.4 TOF MRA source image again demonstrates the eccentric intramural hematoma (white arrow), which is hyperintense compared to adjacent muscles but less so than the flow-related enhancement of residual left ICA lumen (curved arrow).

Figure 1.3.5 TOF MRA MIP image shows a long segment of tapered narrowing (arrows) of distal left ICA with an associated pseudoaneurysm (curved arrow).

Figure 1.3.6 DSA of left ICA prior to carotid stenting confirms the luminal narrowing (arrows) and pseudoaneurysm (curved arrow).

Diagnosis

Spontaneous extracranial carotid dissection.

Differential diagnosis

- Arterial thrombosis: once an artery is thombosed, it is difficult to determine whether underlying dissection is present.
- Atherosclerosis: commonly at carotid bifurcation or of vertebral origin. In comparison, carotid artery dissection typically involves the postbulbar segment at the skull base, and vertebral dissection tends to occur at the C1–2 and C6–7 levels or at the intradural segment. A long segment of tapered narrowing favors dissections, whereas focal narrowing with associated calcification is typical for atherosclerosis. Other findings such as intimal flap or pseudoaneurysm are seen only with dissection.
- Fibromuscular dysplasia: typically beaded appearance. However, arterial dissection is a common complication of fibromuscular dysplasia.

Key points

- Dissection is a common cause of ischemic stroke in a young patient. It refers to separation of the arterial wall layers by intramural hematoma, which may result from intimal tear or rupture of the vasa vasorum. While subintimal dissection typically causes luminal stenosis/occlusion, subadventitial dissection may result in dissecting aneurysm and vessel rupture contained by thrombus, leading to pseudoaneurysm. Extracranial dissection is more common than intracranial dissection.
- Besides traumatic injury, many inciting factors such as sports, chiropractor manipulation, sneezing and coughing, etc. have been associated with spontaneous dissection. In addition, a number of patients have underlying arteriopathies such as fibrous dysplasia, Marfan syndrome, cystic medial necrosis, and type IV Ehlers–Danlos syndrome.

- Clinical presentations: ischemic stroke or TIA, due either to an embolic or to a hemodynamic phenomenon, is the most common presentation. Intracranial dissection may occasionally result in subarachnoid hemorrhage. Local symptoms such as headache and neck pain are common. Carotid dissection may result in Horner's syndrome due to oculosympathetic paresis. Other symptoms include tinnitus and cranial neuropathies, etc.
- Imaging:
  - DSA has been largely replaced by noninvasive CT or MRA. Angiographic findings include string sign, tapered stenosis/occlusion, flame-shaped occlusion, intimal flap, or pseudoaneurysm.
  - MRI/MRA is the modality of choice to assess the cerebral infarction and directly image the intramural hematoma, which eccentrically or circumferentially surrounds a normal or narrowed flow void (crescent or target sign), widens the external diameter of the artery, and is typically hyperintense on fat-saturated T1 and T2 in the subacute phase (in the acute phase, it can be isointense or slightly hyperintense on T1 and T2 sequences).
  - CTA has the same sensitivity as MRI/MRA, and a CTA source image can demonstrate hyperdense intramural hematoma.

Further reading

Provenzale JM. Dissection of the internal carotid and vertebral arteries: imaging features. *AJR Am J Roentgenol* 1995; 165(5): 1099–1104.

Redekop GJ. Extracranial carotid and vertebral artery dissection: a review. *Can J Neurol Sci* 2008; 35(2): 146–152.

Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. *N Engl J Med* 2001; 344(12): 898–906.