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Causes of Acute Stroke:

A Patterned Approach

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Keywords

Stroke; Infarct; Ischemic; Hemorrhagic; Imaging; Cause

INTRODUCTION

Acute stroke is a leading cause of morbidity and mortality in the United States and worldwide, with more than 750,000 cases and 140,000 deaths in the United States each year.^{1,2} In the United States, patients who arrive at a hospital emergency department with acute stroke symptoms often undergo immediate computed tomography (CT) scanning, often before detailed clinical evaluation. Therefore, imaging has a critical role in early diagnosis of stroke. In addition to aiding in diagnosis of acute stroke, patterns of infarction on imaging can suggest a cause, which not only influences immediate management strategies, but also informs optimal secondary prevention therapies to prevent stroke recurrence. It is important to recognize these patterns to better guide image evaluation, recommend appropriate additional imaging as necessary, and ultimately arrive at the underlying cause. This article reviews imaging patterns and secondary findings that can assist in determining the cause of acute stroke.

ISCHEMIC STROKE

Ischemic strokes, which account for most infarcts, can be subdivided based on cause. The Trial of Org 10172 in Acute Stroke Treatment (TOAST) is a system developed to categorize subtypes of ischemic strokes and therefore guide proper management³ (Box 1).

Large Artery Atherosclerosis

Infarctions larger than 15 to 20 mm, involving the cortex, cerebellum, brainstem, and subcortical regions, are usually caused by large vessel disease arising from atherosclerosis of cervical or proximal intracranial vessels,^{3–5} comprising a major cause of acute stroke, ranging from 30% to 43%.⁵ Evaluation of the cervical and intracranial vasculature is critical when large vessel atherosclerosis is suspected as the cause of acute infarction. The mechanism of infarction secondary to atherosclerosis of the extracranial vasculature is a

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combination of low-flow states and artery-to-artery emboli, with the latter thought to be the greater contributing factor.⁶

Anterior circulation infarcts from carotid atherosclerosis most commonly involve the middle cerebral artery (MCA),⁵ resulting in a territorial infarction that involves the deep gray matter and cortex. Such infarcts typically involve the central portion of the brain, including the anterior parietal lobe, the posterior frontal lobe, and the superior temporal lobe (Fig. 1). The anterior cerebral artery (ACA) territory is frequently spared, because of an intact circle of Willis.⁷ Proximal intracranial vascular disease is also under the purview of large artery atherosclerosis. Multiple mechanisms are postulated regarding the pathogenesis of stroke in these cases, including artery-to-artery emboli, in situ thromboembolism, and hemodynamic impairment⁶ (Fig. 2).

Large vessel atherosclerosis affecting the vertebrobasilar system may also produce a specific set of imaging patterns. For example, in the setting of proximal or middle basilar artery occlusion, large infarctions are usually seen in the pons as well as the cerebellum, bilateral inferomedial temporal lobes, occipital lobes, and posterior thalami (Fig. 3). Distal basilar occlusions usually involve the midbrain and thalami, and this is one of the few instances in which bilateral infarctions are seen in the setting of large vessel atherosclerosis.⁸ Infarction of the lateral medulla should prompt radiologists to evaluate the distal vertebral artery, because this is often caused by occlusion of the posterior inferior cerebellar artery, clinically resulting in a lateral medullary syndrome.⁹

External border-zone infarcts are cortical infarcts that occur at the zones between the anterior, middle, and posterior cerebral artery (PCA) territories. Although the cause of these infarcts is not well understood, the prevailing theory is that they result from a combination of embolic phenomena and hypoperfusion.^{10,11} Hypoperfusion may be secondary to actual hemodynamic compromise from stenosis, or caused by these areas being less well perfused at baseline. This condition, in turn, can lead to impaired washout in the setting of an embolic event.¹⁰ In contrast, internal border-zone infarcts are more often caused by proximal stenoses or hemodynamic compromise, because the lenticulostriate vessels supplying these regions have the lowest perfusion pressure (Fig. 4).¹² Posterior circulation border-zone infarcts are less common and, when unilateral, are more often caused by an embolic source from the anterior circulation in the setting of a fetal-type PCA^{11,13} (Fig. 5).

When any of the imaging patterns discussed earlier are encountered, it is critical to evaluate the vasculature for large vessel atherosclerosis as the cause of acute stroke. Such evaluation may be performed with CT angiography (CTA), magnetic resonance (MR) angiography (MRA), or digital subtraction angiography (DSA). In addition, using MR imaging sequences beyond diffusion-weighted imaging may shed further light on infarct cause. For example, T2 fluid-attenuated inversion recovery (FLAIR) hyperintensity within intracranial vasculature may suggest slow flow caused by more proximal vascular disease (Fig. 6A). A serpiginous hypointense structure seen on susceptibility-weighted images may indicate thromboembolus in a distal vessel (Fig. 6B). A hyperdense MCA may be seen on initial noncontrast CT (Fig. 6C).

Cardioembolic

Cardioembolic sources account for 20% to 31% of acute ischemic infarctions.^{5,14} Such infarcts most often present on MR imaging as multiple foci of restricted diffusion involving multiple vascular territories, which are often bilateral¹⁵ (Fig. 7). When this pattern is identified, it is critical to consider further evaluation of the heart, including additional diagnostic imaging. There are several causes of cardioembolic disease, including atrial fibrillation, myocardial infarction with left ventricular thrombus, and infective or inflammatory endocarditis. In addition, emboli may originate from aortic arch disorder, which can be missed on routine transthoracic echo; such cases can lead to bilateral infarctions as well.¹⁶

When isolated ACA infarctions are seen, a cardioembolic source should be considered, although there is debate as to the most common cause (Fig. 8A). Some studies have shown that the cause is more often cardioembolic,^{17,18} whereas others have shown it is more often secondary to large vessel atherosclerosis of the ACA itself, the latter being more common in Asian populations.^{19,20} Isolated ACA infarctions are less common than MCA infarctions.^{5,7,17} When infarctions involve both the ACA and MCA territories, the cause is also often cardioembolic. For example, an acute myocardial infarction in a patient with atrial fibrillation may lead to distal emboli and decreased cardiac output⁷ (Fig. 8B).

Small Artery Occlusion (Lacunar Infarction)

Small artery occlusions can be surmised as the cause of infarcts less than 20 mm, without evidence of other disorders, such as vascular disease, vasospasm, or a cardioembolic source.⁴ These occlusions account for 10% to 23% of acute strokes.⁵ Risk factors include diabetes and hypertension. Lacunar infarcts are seen most often in the basal ganglia, internal capsule, corona radiata, and brainstem (Fig. 9). PCA infarcts are more often lacunar in cause, and often present with typical clinical syndromes.²¹ Exclusion of proximal vascular disease is critical to the diagnosis. Secondary findings on imaging that may suggest small vessel disease as the cause of infarction include presence of chronic lacunar infarcts, cerebral microbleeds, white matter ischemic disease, prominent perivascular spaces, and cerebral atrophy.²²

Unusual Causes

Approximately 2% to 11% of ischemic strokes have unusual causes.^{5,23} Although uncommon, it is important to consider atypical causes once common causes have been excluded, because this has important implications for management. Vasculopathies represent a large portion of unusual causes of stroke, in addition to other causes, such as hypercoagulable states, hematologic disorders, right-to-left vascular shunts, and arterial dissections. Although many of these disease processes look similar, certain patterns of vascular involvement and secondary findings on imaging may help elucidate the underlying cause.

Vasculopathies

Vasculopathies involving proximal vessels often result in territorial infarcts of perforating vessels supplying deep white matter and the basal ganglia. A component of subarachnoid

hemorrhage may also be seen.²⁴ Extensive collaterals are present in the setting of moyamoya and sickle cell disease. Moyamoya disease can present in both children and adults, usually as recurrent large territorial infarctions. In children, infarctions are often ischemic, whereas in adults they are more often hemorrhagic. Vascular imaging is critical and shows stenosis or occlusion of the distal internal carotid artery (ICA) and its proximal branches, with sparing of the posterior circulation.^{7,25,26} The most salient imaging feature is the extensive collateral vascularity, best seen on vascular imaging (Fig. 10).

Basilar meningitis, as in the case of tuberculosis, can lead to arterial spasm and vasoconstriction of proximal vessels, resulting in infarction. Additional findings that may suggest this cause include involvement of the posterior circulation, unlike moyamoya, and involvement of perforating vessels, leading to basal ganglia and thalamic infarcts.²⁷ Secondary findings of communicating hydrocephalus, ventriculitis, and vasospasm further support this as a cause (Fig. 11). Acute septic meningitis from *Streptococcus pneumoniae*, *Neisseria meningitidis*, *Haemophilus influenzae*, and *Staphylococcus aureus* have been reported to cause infarction as well²⁸ (Fig. 12). Vasospasm may be seen on vascular imaging.²⁹

Vasculopathies involving second-order and third-order arteries often result in cortical infarctions in distal vascular territories, typically without deep tissue involvement. These infarcts are also likely to be hemorrhagic. Contrast-enhanced MR angiography may show vessel wall enhancement. Causes include primary angiitis of the CNS (PACNS), granulomatosis with polyangiitis, polyarteritis nodosa, and sarcoid.²⁴ Infectious causes, such as syphilis and herpes zoster, may also affect second-order and third-order vessels. Herpes zoster occurs with reactivation of the latent virus, and is often indistinguishable from PACNS on imaging. It often presents with multiple infarcts, often involving the basal ganglia and subcortical white matter. Angiography may reveal segmental stenosis, thrombosis, and beading of the proximal MCA and ACA.²⁶ Subarachnoid hemorrhage may be seen^{30,31} (Fig. 13).

Small vessel vasculopathies, such as Sjögren, collagen vascular disease, human immunodeficiency virus encephalitis, and radiation vasculopathy typically appear normal on angiography. Infarctions may be seen in deep gray structures, white matter, and subcortical regions. In contrast with multiple sclerosis, the white matter lesions seen with small vessel vasculopathy often parallel the ventricles. These lesions often present at an earlier age and show rapid progression.²⁴

Systemic lupus erythematosus (SLE) is a collagen vascular disease that can result in small vessel vasculopathy (Fig. 14). Infarction pattern in lupus is nonspecific. More often than not, infarction is caused by secondary causes of the disease rather than vasculitis, such as cardiac valvular disease (Libman-Sacks endocarditis), hypercoagulability, atherosclerosis from hypertension and steroid treatment, and venous thrombosis. Secondary findings to look for include intracranial hemorrhage, and cerebral atrophy, with normal vascular imaging.³²

Neoplastic vasculopathy is a result of vascular invasion secondary to neoplasm; affected patients may present with acute infarct. Vessel occlusion may be seen on angiography.

Lymphoma is usually the cause, with the most common cause being non-Hodgkin. For example, diffuse large B-cell lymphoma (DLBCL) may present with acute infarcts and infarctlike lesions, which are diffusion restricting on MR imaging and show signal abnormality on T2-weighted sequences. Infarcts may involve the corpus callosum and periventricular white matter, and may be in a watershed distribution. In the case of DLBCL, small vessels are often involved, so angiography may be normal. Secondary findings include meningeal enhancement and central pontine hyperintensity, which may be secondary to venous congestion in the setting of vascular occlusion by tumor cells^{33,34} (Fig. 15). Less common causes of neoplastic vasculopathy include multiple myeloma, T-cell leukemia, and hairy cell leukemia.

There are also hereditary causes of acute infarction. Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) causes focal and confluent subcortical white matter infarcts predominantly involving the superior frontal lobes, external capsule, and anterior temporal lobes. Lacunar infarcts and microbleeds may also be seen³⁵ (Fig. 16).

Dissection

Another unusual cause of acute stroke is arterial dissection. This condition often occurs in young patients presenting with focal deficit and headache or neck pain, with a possible inciting event, often trauma. Infarction usually has an embolic cause rather than hypoperfusion.^{36,37} Vascular imaging is critical to evaluate for a dissection flap and stenosis or occlusion secondary to dissection. However, in the absence of vascular imaging, a distal cervical ICA dissection may be seen on routine brain MR imaging, so it is important to evaluate the carotid arteries at the skull base for any signs of dissection (Fig. 17).

Stroke of Undetermined Cause

This category is invoked when multiple causes or no causes of stroke are identified. Causes that may be missed on conventional cardiac imaging, cardiac work-up, or vascular imaging should be considered as potential causes of cryptogenic stroke. Such entities include patent foramen ovale (Fig. 18), aortic arch atherosclerosis, paroxysmal atrial fibrillation,³ or nonstenosing atherosclerosis of the carotid arteries.^{38,39}

HEMORRHAGIC STROKE

Hemorrhagic strokes comprise approximately 5% to 21% of acute strokes.¹⁴ One of the most common causes of hemorrhagic strokes is hypertension.⁴⁰ Hypertensive strokes occur in typical locations, including the basal ganglia, thalamus, pons, and cerebellum (Fig. 19).

Vascular malformations are another cause of hemorrhagic strokes. Arteriovenous malformations (AVMs) should be considered in younger patients who present with acute hemorrhagic strokes, because hemorrhage is the most common presentation.⁴¹ AVMs are characterized by abnormal arteriovenous connections with a vascular nidus, and absence of an intervening capillary bed. Vascular imaging with CTA, MRA, or DSA is critical in making the diagnosis (Fig. 20).

Venous infarction should be considered when the location of the infarct does not correspond with an arterial vascular distribution, or spans more than 1 arterial distribution (Fig. 21). These patients may initially present with vasogenic edema on T2 FLAIR sequences, which, if not addressed, usually progresses to infarction, which is often hemorrhagic. Secondary findings to search for include presence of a hyperdense venous sinus or cortical vein on noncontrast CT, or absence of flow-related signal void in venous structures on MR imaging. Further evaluation with MR venography or CT venography is often performed for confirmation. Hypercoagulable states are the most important contributing factor in adults.^{41,42}

Cerebral amyloid angiopathy is another cause of hemorrhagic infarction to be considered in older patients who present with multiple, recurrent cortical and subcortical hemorrhagic strokes. Susceptibility-weighted imaging is key in making this diagnosis, because multiple foci of parenchymal signal hypointensity are seen in a cortical and subcortical distribution on MR imaging (Fig. 22). Vascular imaging is usually normal.⁴³

SUMMARY

Determining the cause of an acute stroke is critical, because this guides patient management. Failure to accurately identify the cause for stroke impairs the clinician's ability to deliver optimal, immediate, acute care and limits the precision with which longer-term therapies can be initiated to prevent stroke recurrence. Patterns on imaging, such as vascular territories affected, vessels involved, and secondary findings, can provide clues to the underlying cause. It is critical to understand these imaging patterns to suggest a cause or recommend further imaging to elucidate the underlying cause. Once large vessel, small vessel, and cardioembolic sources have been excluded, unusual causes should be considered.

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Box 1

Trial of Org 10172 in acute stroke treatment classification

- Large artery atherosclerosis (embolus or thrombosis)
- Cardioembolic
- Small vessel occlusion (lacune)
- Stroke of other determined cause, or unusual cause
- Stroke of undetermined cause
 - Two or more causes identified
 - Negative evaluation
 - Incomplete evaluation

The TOAST classification was developed to categorize causes of acute ischemic stroke. It is a useful way to categorize stroke in order to guide management decisions.

KEY POINTS

- Emergent imaging is performed on a high proportion of patients presenting with acute stroke symptoms; as such, imaging plays an important role in early diagnosis and management.
- Determining the cause of an acute stroke is critical for clinicians to initiate appropriate therapy.
- Imaging patterns of acute stroke can suggest the cause, and therefore aid in appropriate management.
- Once large vessel atherosclerosis, small vessel disease, and cardioembolic sources have been excluded, unusual causes should be considered.

Fig. 1.

A 54-year-old man presenting with altered mental status. (A) Axial CT images show an acute left MCA territory infarction involving the left insular cortex, basal ganglia, posterior frontal, and anterior parietal lobes. (B) Axial images from CT angiography (computed tomography) of the head and neck show moderate to severe stenosis of the proximal left ICA (red arrow) and left MCA thrombus involving the distal M1 and proximal M2 segments (yellow arrow).

Fig. 2.

Anterior circulation infarctions. (A) A 96-year-old woman with history of aortic valve repair on Coumadin presenting with acute-onset left hemiparesis. Axial diffusion-weighted magnetic resonance (MR) shows infarctions involving both deep gray matter and cortex, typical of an MCA territory infarction. (B) Axial MR angiography (magnetic resonance) time of flight (TOF) shows stenosis of the right intracranial ICA (*red arrow*). (C) Three-dimensional (3D) maximum intensity projection (MIP) image shows occlusion of the M1 segment of the right MCA (*yellow arrow*), with infarction probably secondary to a combination of hemodynamic impairment and artery-to-artery emboli. (D) A 73-year-old woman with hypertension and hyperlipidemia presenting with acute right MCA syndrome. Axial diffusion-weighted image shows infarct involving the right frontal lobe and basal ganglia in an MCA distribution. (E) 3D MIP from MRA head shows luminal narrowing of the right M1/M2 junction of the MCA (*white arrow*).

Fig. 3. Basilar occlusion. (A) Axial diffusion-weighted images show bilateral cerebellar, right and left pontine, and right thalamic infarcts. (B) 3D MIP image from MRA shows severe stenosis of the V1/V2 segment of the right vertebral artery (*red arrow*). (C) Sagittal MIP image from CTA head shows near-complete occlusion of the distal basilar artery (*yellow arrow*), which extended into the proximal PCAs.

Fig. 4.

A 72-year-old man with history of transient ischemic attacks, hypertension, and diabetes, presenting with right arm numbness. (A) Axial diffusion-weighted image shows acute infarction in an internal border zone distribution in the left frontoparietal lobes. (B) 3D MIP from MRA of the neck shows severe stenosis of the proximal cervical ICA.

Fig. 5.

A 75-year-old man with history of type 2 diabetes mellitus, hypercholesterolemia, hypertension, presenting with word-finding difficulty and right facial droop. (A) Axial diffusion-weighted images show acute infarctions involving MCA/PCA border-zone territories. (B) 3D MIP from MRA head shows complete lack of flow in the left V4 vertebral artery, as well as multifocal stenoses of the left PCA, both of which likely contributed to the border zone infarction.

Fig. 6.

(A) A 33-year-old woman presenting with confusion and aphasia, found to have a left MCA territory infarction and nonocclusive left M2 thrombus. Axial T2 FLAIR shows increased signal within multiple left MCA branches (*yellow arrows*) secondary to slow flow from the proximal partial occlusion. (B) A 94-year-old woman presenting with speech difficulties and left facial droop, found to have right MCA territory infarction. Susceptibility-weighted sequences show a tubular area of blooming in the anterior sylvian fissure, consistent with thromboembolus (*white arrow*). (C) A 90-year-old woman with history of congestive heart failure, hypertension, hyperlipidemia, presenting with left hemiparesis and dysarthria. Initial axial noncontrast head CT shows a dense right MCA (*red arrow*).

Fig. 7. Multiple bilateral infarctions from cardioembolic source. (A) Axial diffusion-weighted MR images in a 36-year-old woman with systemic lupus erythematosus and Libman-Sacks endocarditis. (B) Axial T2 FLAIR images in a 38-year-old male intravenous drug abuser with bacterial endocarditis. (C) Axial diffusion-weighted MR images in a 79-year-old man with atrial fibrillation.

Fig. 8.

(A) A 77-year-old woman with history of hypertension and advanced congestive heart failure with new-onset right hemiparesis. Axial diffusion-weighted MR image shows acute left ACA territory infarction likely secondary to cardioembolic source from advanced heart failure, because no vascular abnormality of the ACA was seen. (B) An 80-year-old man with history of prior stroke, atrial fibrillation, coronary artery disease, and hypertension presenting with new-onset left-sided weakness after collapse. Axial diffusion-weighted MR images reveal acute infarction in the right frontal cortex and right caudate head in a combined ACA/MCA distribution, likely cardioembolic in cause from known atrial fibrillation.

Fig. 9. Infarctions measuring less than 20 mm in the typical locations for small vessel infarctions, consistent with lacunar infarcts. (A) Axial diffusion-weighted MR shows acute infarct in the left thalamus in a hyperlipidemic smoker. (B) Axial diffusion-weighted MR shows acute infarct in the right internal capsule in a patient with multiple vascular risk factors. (C) Axial diffusion-weighted MR shows acute infarct in the right pons in a patient with hyperlipidemia and hypertension.

Fig. 10.

A 49-year-old man with no known past medical history presented with acute-onset right-sided hemiparesis and confusion. (A) Axial noncontrast CT of the head shows a large left thalamic hematoma (*arrow*) with intraventricular extension. (B) Axial and MIP images from follow-up CTA show stenosis of the left cavernous ICA (*arrow*) as well as multiple collateral vessels, consistent with moyamoya.

Fig. 11.

A 55-year-old man with nasopharyngeal carcinoma presenting with altered mental status and fever, found to have *Streptococcus intermedius* meningitis. (A) Axial diffusion-weighted MR images show left basal ganglia and internal capsule infarction (*star*). On further inspection, the patient is noted to have mild ventriculomegaly and layering foci of restricted diffusion in the atria of the lateral ventricles, consistent with ventriculitis (*yellow arrows*). Restricted diffusion is also present in the basilar cisterns, consistent with basilar meningitis (*red arrows*). (B) Axial TOF image from MRA head at initial presentation and CTA head 8 days later. At 8-day follow-up, there is increased narrowing in the supraclinoid ICA (*yellow arrows*) and basilar artery (*red arrows*) secondary to vasospasm from basilar meningitis.

Fig. 12.

A 27-year-old woman presenting with fever and acute visual loss after abscess drainage for lower extremity necrotizing fasciitis. (A) Axial diffusion-weighted MR image shows a small infarct in the right middle cerebellar peduncle (*circle*). (B) Secondary findings of sulcal restricted diffusion (*red arrow*) and leptomeningeal enhancement on axial T1 postcontrast MR (*yellow arrows*) suggest meningitis as the cause of infarction.

Fig. 13.

Herpes zoster vasculitis. A 65-year-old woman initially presented with sudden-onset left hand weakness and facial droop. (A) Axial diffusion-weighted MR shows a right parietal infarct (*star*). (B) The patient was also noted to have failure of suppression of sulcal cerebrospinal fluid on T2 FLAIR, which corresponded to right convexity subarachnoid hemorrhage on noncontrast CT (*yellow arrows*). Vascular imaging was normal on initial presentation. (C) The patient returned 8 months later with right hand clumsiness and was found to have a new left insular infarct on axial diffusion-weighted MR (*red arrow*). (D) Axial T1-weighted image shows intrinsic T1 hyperintensity, consistent with hemorrhage (*circle*). (E) 3D MIP from MRA at that time reveals focal high-grade stenosis of an M2 branch of the left MCA (*yellow arrow*). Lumbar puncture revealed herpes zoster infection.

Fig. 14.

SLE. A 36-year-old woman with history of SLE presents with change in mental status and palate numbness. (A) Axial diffusion-weighted MR image shows punctate acute right frontal cortical infarct (*red arrow*). (B) Secondary findings of chronic right frontal subcortical infarct (*yellow arrow*) and white matter lesions paralleling the ventricles (*oval*) on axial T2 FLAIR images are characteristic of lupus. (C) Additional chronic infarcts were seen in this young patient, including a chronic right cerebellar infarct on axial T2-weighted MR image (*red arrow*) and left caudate infarct on axial T2 FLAIR-weighted MR image (*yellow arrow*). (D) There is a normal appearance of the vessels on 3D MIP image from MRA, which is typical of SLE.

Fig. 15.

Intravascular DLBCL. A 54-year-old man presenting with 3 weeks of ascending numbness and malaise, initially treated for Guillain-Barré syndrome. (A) Patient returned for ongoing symptoms, and axial diffusion-weighted MR image revealed multiple bilateral areas of acute infarction in a watershed distribution (*red arrows*). (B) After 2 months, axial diffusion-weighted and T2 FLAIR images show multiple additional infarcts and infarctlike lesions, with diffusion restriction and corresponding T2 hyperintensity (*ovals*). (C) Axial T1 postcontrast images revealed leptomeningeal enhancement (*yellow arrows*). (D) Axial T2 FLAIR shows central pontine T2 hyperintensity (*white arrow*).

Fig. 16.

CADASIL. A 38-year-old woman with known CADASIL presented with altered mental status. (A) Axial diffusion-weighted MR image shows a focus of diffusion restriction in the left temporal subcortical white matter consistent with acute infarct (*star*). (B) Axial T2 FLAIR images show confluent periventricular white matter hyperintensity involving the anterior temporal and frontal white matter, as well as the external capsule, in a pattern consistent with CADASIL (*red arrows*). (C) Axial susceptibility-weighted image shows nonspecific microhemorrhages (*yellow arrows*).

Fig. 17.

Cervical carotid dissection. A 40-year-old man with no past medical history presented with acute left facial droop and right jaw pain. (A) Axial diffusion-weighted image shows right-sided parietal and caudate head infarcts. (B) Axial T1-weighted sequence shows hyperintensity surrounding the right petrous ICA consistent with blood in the dissection flap (*yellow arrow*). (C) MRA neck performed for confirmation shows hyperintensity surrounding the proximal cervical ICA on T1 fat-saturated sequence (*red arrow*). (D) 3D MRA MIP image shows resultant long segment smooth stenosis extending from just distal to the bifurcation to the petrous ICA (*yellow arrows*).

Fig. 18.

A 61-year-old-woman with breast cancer and left upper extremity deep venous thrombosis. Axial diffusion-weighted images show multiple bilateral infarctions involving multiple vascular territories. Cause was presumed as secondary to undetected patent foramen ovale, because no large vessel or cardioembolic sources were identified.

Fig. 19.

Three different patients with systemic hypertension, presenting with hemorrhagic infarctions in characteristic locations. In all of these cases, vascular imaging revealed no abnormality. (A) Axial CT with hemorrhagic infarct in the pons. (B) Axial CT with hemorrhagic infarct in the right basal ganglia. (C) Axial T1-weighted MR image with hemorrhagic infarct in the left thalamus.

Fig. 20.

A 16-year-old boy with no significant past medical history presenting with acute-onset left-sided weakness, headache, and subsequent coma. (A) Axial noncontrast head CT revealed right basal ganglia and intraventricular hemorrhage causing a leftward midline shift. (B) Subsequent CT angiography of the head revealed an arteriovenous malformation with its nidus at the junction between the thalamus and posterior limb of the right internal capsule, supplied by a PCA branch with deep venous drainage.

Fig. 21.

A 53-year-old woman with history of breast cancer presented after being found down unresponsive. (A) Noncontrast head CT at time of presentation shows hemorrhagic infarctions of the bilateral basal ganglia and thalami. (B) Tubular hyperdensity is seen in the bilateral internal cerebral veins (*red arrows*), vein of Galen (*yellow arrows*), and straight sinus (*white arrow*), consistent with venous sinus thrombosis and venous infarction.

Fig. 22.

Cerebral amyloid angiopathy. A 65-year-old woman with recurrent transient neurologic symptoms of unclear cause and migraine with aura, presenting with acute-onset right face and right arm numbness and weakness. (A) Axial noncontrast head CT and axial T1-weighted MR image show a left high parietal intra-parenchymal hemorrhage. (B) Axial susceptibility-weighted images reveal multifocal subarachnoid and subcortical microhemorrhages. Brain biopsy was positive for β -amyloid plaques.