RadioGraphics

Sequence-specific MR Imaging Findings That Are Useful in Dating Ischemic Stroke[1]

TEACHING POINTS
See last page

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LEARNING OBJECTIVES
After completing this journal-based CME activity, participants will be able to:

- Explain how to determine the relative age of an ischemic stroke on the basis of MR imaging findings.
- Describe the importance and limitations of FLAIR MR imaging in dating acute ischemic stroke.
- Discuss the current time windows and guidelines for administering thrombolytic and neurointerventional therapies.

INVITED COMMENTARY
See discussion on this article by Yuh et al (pp 1297–1299).

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Patients may present to the hospital at various times after an ischemic stroke. Many present weeks after a neurologic deficit has occurred, as is often the case with elderly patients and those in a nursing home. The ability to determine the age of an ischemic stroke provides useful clinical information for the patient, his or her family, and the medical team. Many times, perfusion imaging is not performed, and pulse sequence–specific magnetic resonance (MR) imaging findings may help determine the age of the infarct. The findings seen at apparent diffusion coefficient mapping and diffusion-weighted, fluid-attenuated inversion recovery (FLAIR) and unenhanced and contrast material–enhanced T1- and T2-weighted gradient-echo and susceptibility-weighted MR imaging may help determine the relative age of a cerebral infarct. Strokes may be classified and dated as early hyperacute, late hyperacute, acute, subacute, or chronic. Recent data indicate that in many patients with restricted diffusion and no change on FLAIR images, it is more likely than was initially thought that the stroke is less than 6 hours old. The time window to administer intravenous tissue plasminogen activator is currently 4.5 hours from the time when the patient was last seen to be normal, and for anterior circulation strokes, the time window for administering intraarterial tissue plasminogen activator is 6 hours from when the patient was last seen to be normal. For this reason, accurate dating is important in patients with ischemic stroke.

Introduction

Patients may present to the hospital at various times after an ischemic stroke. Many present weeks after a neurologic deficit has occurred; unfortunately, this is often the case with elderly patients and those in a nursing home. In addition, about one out of seven strokes occurs during sleep, and the time from when the patient was last

Abbreviations: ADC = apparent diffusion coefficient, FDA = Food and Drug Administration, FLAIR = fluid-attenuated inversion recovery, tPA = tissue plasminogen activator

RadioGraphics 2012; 32:1285–1297 • **Published online** 10.1148/rg.325115760 • **Content Codes:** MR NR

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Table 1
Guide to Dating an Acute Ischemic Stroke on the Basis of MR Imaging Findings

Imaging Sequence	Early Hyperacute (0–6 hours)	Late Hyperacute (6–24 hours)	Acute (24 hours–1 week)
ADC mapping	Low signal intensity	Low signal intensity	Low signal intensity
Diffusion-weighted	High signal intensity	High signal intensity	High signal intensity
FLAIR	Variable signal intensity; usually high after 6 hours; ipsilateral arterial may be high at 0–2 hours	Usually high signal intensity	High signal intensity
T1-weighted	Isointensity	Usually low signal intensity after 16 hours	Low signal intensity; hyperintensity with cortical necrosis may be seen after 3–5 days
Contrast-enhanced T1-weighted	Arterial enhancement may occur after 0–2 hours; parenchymal cortical enhancement may occur after 2–4 hours in incomplete infarction	Arterial enhancement may occur; parenchymal cortical enhancement may be seen in incomplete infarction*; meningeal enhancement may occur	Arterial and meningeal enhancement may occur†; parenchymal enhancement may occur after 5–7 days in complete infarction
T2-weighted	Isointensity; may see loss of flow void in ipsilateral carotid artery at 0–2 hours in cases of large stroke	Variable; usually high after 8 hours	High signal intensity
Susceptibility-weighted or gradient-echo	May see hemorrhagic transformation within 0–12 hours (unlikely)	May see hemorrhagic transformation within 0–12 hours (unlikely)	Hemorrhagic transformation most likely within 48 hours; risk remains for up to 5 days

Note.—Susceptibility-weighted imaging may help differentiate hemorrhagic transformation from cortical necrosis, and diffusion-weighted imaging findings may be falsely negative in patients with hyperacute or acute posterior circulation or lacunar stroke. In the hyperacute stages, a large area of enhancement in the deep gray matter or basal ganglia may indicate impending hemorrhagic transformation.
*Increased parenchymal cortical enhancement should end around 48 hours after stroke.
†Arterial and meningeal enhancement should end around 1 week after stroke.

seen to be normal is unknown (1). The ability to determine the age of an ischemic stroke provides useful clinical information for the patient, his or her family, and the medical team caring for the patient. Many times, perfusion imaging is not performed, and sequence-specific magnetic resonance (MR) imaging findings may help determine the age of the infarct. Strokes may be classified and dated thus: early hyperacute, a stroke that is 0–6 hours old; late hyperacute, a stroke that is 6–24 hours old; acute, 24 hours to 7 days; subacute, 1–3 weeks; and chronic, more than 3 weeks old (Tables 1, 2). In this article, useful findings on apparent diffusion coefficient (ADC) maps and MR images obtained with diffusion-weighted, fluid-attenuated inversion-recovery (FLAIR), T2-weighted, T1-weighted, and gadolinium-based contrast material–enhanced T1-weighted sequences that help classify stroke are discussed with a brief review of current thrombolytic and neurointerventional therapies and their time windows (Fig 1).

Imaging Appearances

ADC Maps and Diffusion-weighted Imaging

ADC maps may depict darkening within minutes of stroke onset and are more sensitive than diffusion-weighted sequences that are performed after a stroke, which demonstrate hyperintensity.

Table 2
Guide to Dating a Subacute or Chronic Ischemic Stroke on the Basis of MR Imaging Findings

Imaging Sequence	Subacute (1–3 weeks)	Chronic (>3 weeks)
ADC mapping	Low signal intensity for 7–10 days; may pseudonormalize at 10–15 days; then high signal intensity	High signal intensity
Diffusion-weighted	High signal intensity for 10–14 days; then iso- or hypointensity; hyperintensity if T2 shine-through is seen	Variable signal intensity; may be isointense; hyperintense in the presence of T2 shine-through; hypointense in the presence of cystic encephalomalacia
FLAIR	High signal intensity	Low signal intensity in the presence of gliosis and cystic encephalomalacia
T1-weighted	Low signal intensity; hyperintensity with cortical necrosis most common after 2 weeks	Low signal intensity; hyperintensity with cortical necrosis may be seen*
Contrast-enhanced T1-weighted	Parenchymal enhancement may occur in complete infarction, usually for 1–8 weeks	Parenchymal enhancement may occur for 8 weeks–4 months†
T2-weighted	High signal intensity; fogging may be seen at 2–3 weeks	High signal intensity
Susceptibility-weighted or gradient-echo	Hemorrhagic transformation uncommon after 1 week	Microbleeding and hemorrhagic transformation (new incidence unlikely)

Note.—Susceptibility-weighted imaging may help differentiate hemorrhagic transformation from cortical necrosis, and diffusion-weighted imaging findings may be falsely negative in patients with hyperacute or acute posterior circulation or lacunar stroke.
*Cortical necrosis usually resolves by 3 months after stroke and rarely persists for more than a year.
†If parenchymal enhancement persists for more than 8 weeks, other causes should be considered.

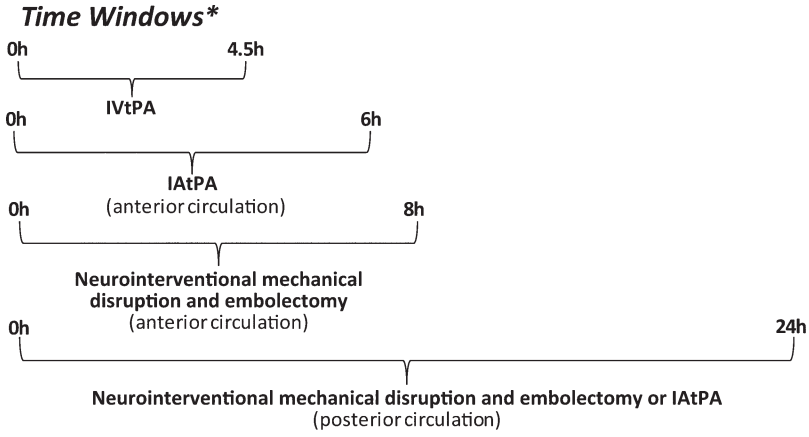

Figure 1. Chart shows the time windows for administering intravenous tissue plasminogen activator *(IVtPA)*; intra-arterial tPA *(IAtPA)*; neurointerventional mechanical disruption and embolectomy; and neurointerventional mechanical disruption and embolectomy or intraarterial tPA in patients with ischemic stroke. *DWI* = diffusion-weighted imaging, *PWI* = perfusion-weighted imaging.

It should be noted that ADC maps and diffusion-weighted images may not have reliably positive results within the first 2–4 hours after the onset of stroke symptoms. Darkening on ADC maps distinguishes stroke from "T2 shine-through," a later finding that occurs after infarction and appears bright on both diffusion-weighted images and ADC maps. Signal intensity on ADC maps is said to be lowest 2–3 days after stroke and persists for about 7–10 days. A good rule of thumb is that if the signal intensity on ADC maps is low, the stroke is less than 1 week old (2–4). Signal intensity is lowest on diffusion-

Teaching Point

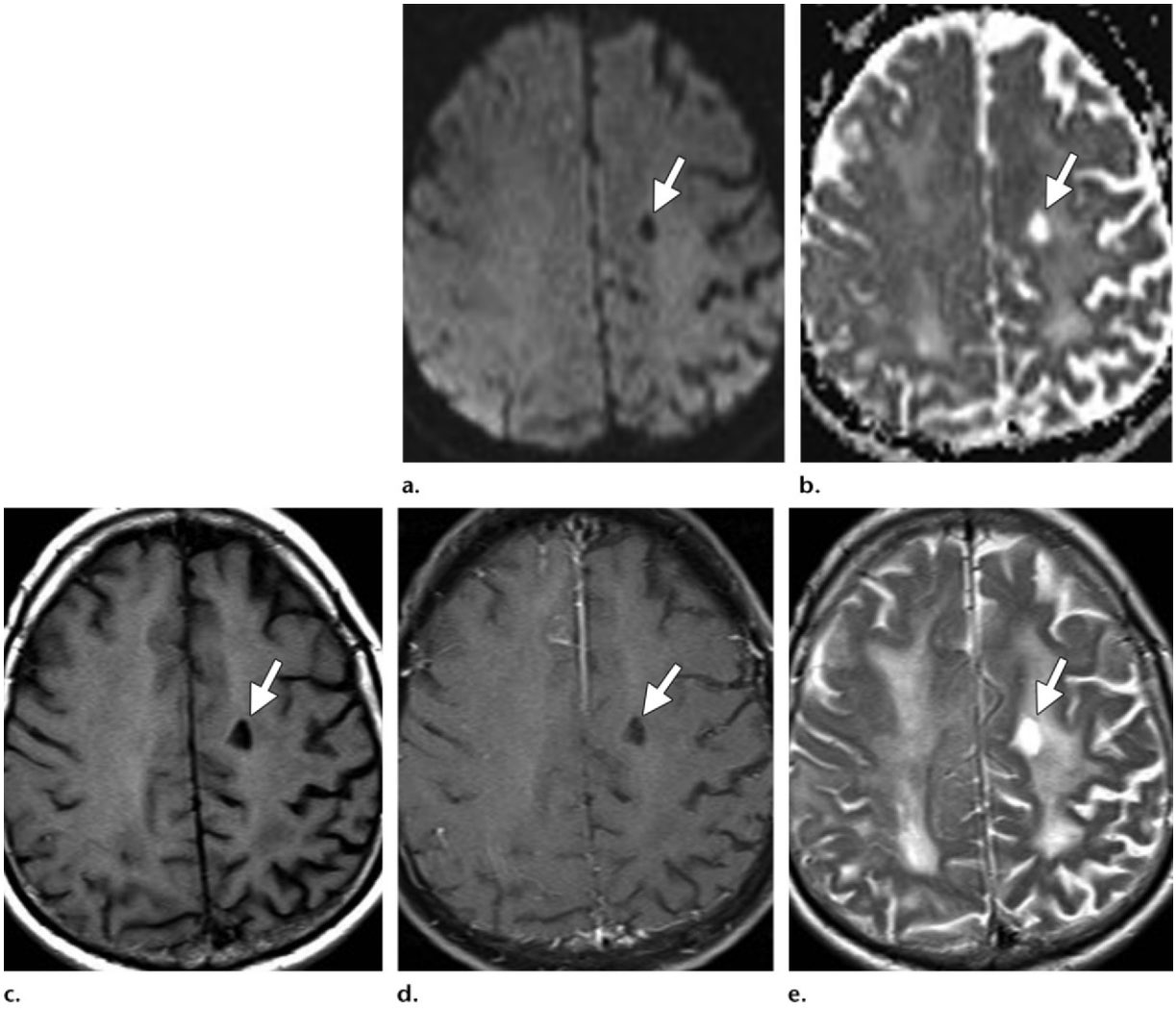

Figure 2. Chronic lacunar stroke in an 82-year-old man with diabetes, hypertension, and altered mental status. **(a)** Diffusion-weighted MR image shows an area of low signal intensity in the left centrum semiovale (arrow). **(b)** ADC map shows an area of high signal intensity in the left centrum semiovale (arrow). **(c)** Unenhanced T1-weighted MR image shows an area of low signal intensity in the left centrum semiovale (arrow). **(d)** Gadolinium-based contrast-enhanced T1-weighted MR image shows an area of contrast enhancement in the left centrum semiovale (arrow). **(e)** T2-weighted MR image shows an area of high signal intensity in the left centrum semiovale (arrow). These findings are consistent with a chronic lacunar stroke with resultant cystic encephalomalacia (cf Table 2).

weighted images 3–4 days after infarction and persists for about 10–14 days, longer than that seen at ADC mapping (2–4). In clinical practice, the sensitivity of diffusion-weighted imaging for depicting ischemic changes is inconsistent for the first 6 hours after stroke; in these situations, perfusion-weighted imaging is often necessary to depict such changes (5–7). In addition, there are several reports of documented stroke with no change in signal intensity on diffusion-weighted images within the first 24 hours, particularly in the posterior vertebrobasilar system and brainstem and in patients with lacunar stroke (8–12). On ADC maps, "pseudonormalization" may occur 1–2 weeks after stroke onset, but signal intensity remains high on T2-weighted

images, and it may be slightly high on diffusion-weighted images (13). At diffusion-weighted imaging, signal intensity usually normalizes early in the chronic phase and becomes low after cystic encephalomalacia occurs (Fig 2).

FLAIR Imaging

Teaching Point

According to clinical experience and the literature, signal intensity on FLAIR images varies after stroke (14–16). However, both clinical experience and most of the literature indicate that in most patients with ischemic stroke, findings on FLAIR images are positive 6–12 hours after onset of symptoms (14). For some neurointerventionists, the presence of restricted diffusion with negative findings at FLAIR imaging alone has been enough to initiate treatment. Furthermore, recent studies report that when diffusion-weighted imaging findings are positive and FLAIR imaging findings are negative, there is a strong likelihood that the stroke is less than 6 hours old (17,18). In a study by Thomalla et al (18) of 120 consecutive patients with stroke, it was reported that when restricted diffusion was present and FLAIR imaging findings were negative, specificity (93%) and positive predictive value (94%) were high that the stroke was less than 3 hours old. In another study by Aoki et al (17) of 333 consecutive patients with stroke that excluded lacunar and vertebrobasilar system infarcts, when restricted diffusion was present and FLAIR imaging findings were negative, the positive predictive value was 77% that the stroke was less than 3 hours old, 96% that it was less than 4.5 hours old, and 100% that it was less than 6 hours old. These findings suggest that when FLAIR imaging findings are negative, the stroke is likely less than 6 hours old. However, it is important to remember how much signal intensity can vary at FLAIR imaging: Recently, one patient at our institution did not demonstrate positive FLAIR imaging findings until 24 hours after changes were seen at diffusion-weighted imaging and ADC mapping.

It has been demonstrated that infarcts with a signal intensity ratio (defined as the intralesional signal intensity divided by that in the normal contralateral side) of less than 1.37 on FLAIR images are less than 36 hours old (2,4). Interestingly, in several patients with acute stroke and false-negative findings at diffusion-weighted imaging, FLAIR imaging findings were positive (8,11). However, if no changes indicative of acute stroke are seen at diffusion-weighted imaging or ADC mapping, another cause for the patient's symptoms (other than acute stroke) should be sought (3). Signal intensity remains high at FLAIR imaging into the chronic phase of infarction and is low with cystic encephalomalacia. Arterial hyperintensity may be seen at FLAIR imaging early in stroke, within 0–2 hours after onset of symptoms.

T2-weighted Imaging

High signal intensity is not usually seen at T2-weighted imaging until at least 8 hours after the initial ischemic insult. It persists into the chronic phase and usually maximizes in the subacute phase (3,19). It should be noted that, after stroke onset, the time at which T2-weighted imaging findings become positive varies; at our institution, we have found that it is likely longer than 8 hours. The time at which T2-weighted imaging findings become positive depends on the echo train length of the fast spin echo.

"Fogging" may be seen at MR imaging around 1–4 weeks after stroke, with a peak at 2–3 weeks. It appears as an area of isointensity relative to the brain and is thought to result from infiltration of inflammatory cells into infarcted tissue (20,21). With larger strokes, loss of the normal flow void may be seen in the ipsilateral carotid artery at T2-weighted imaging within the first 2 hours after onset of symptoms.

T1-weighted Imaging

Low signal intensity is not usually seen at T1-weighted imaging until 16 hours after onset of stroke and persists into the chronic phase (3,19). An area of serpiginous cortical high signal intensity may be seen in patients with cortical laminar or pseudolaminar necrosis from 3–5 days after infarction but is most commonly seen after about 2 weeks.

The pattern of contrast enhancement may help determine the age of the stroke. In ischemic stroke, enhancement may be arterial, meningeal, or parenchymal. Arterial enhancement, dubbed the "intravascular enhancement" sign, usually occurs first and may be seen as early as 0–2 hours after onset of stroke (Fig 3d). It fades about 1 week after stroke, around the time parenchymal enhancement begins, and after complete infarction

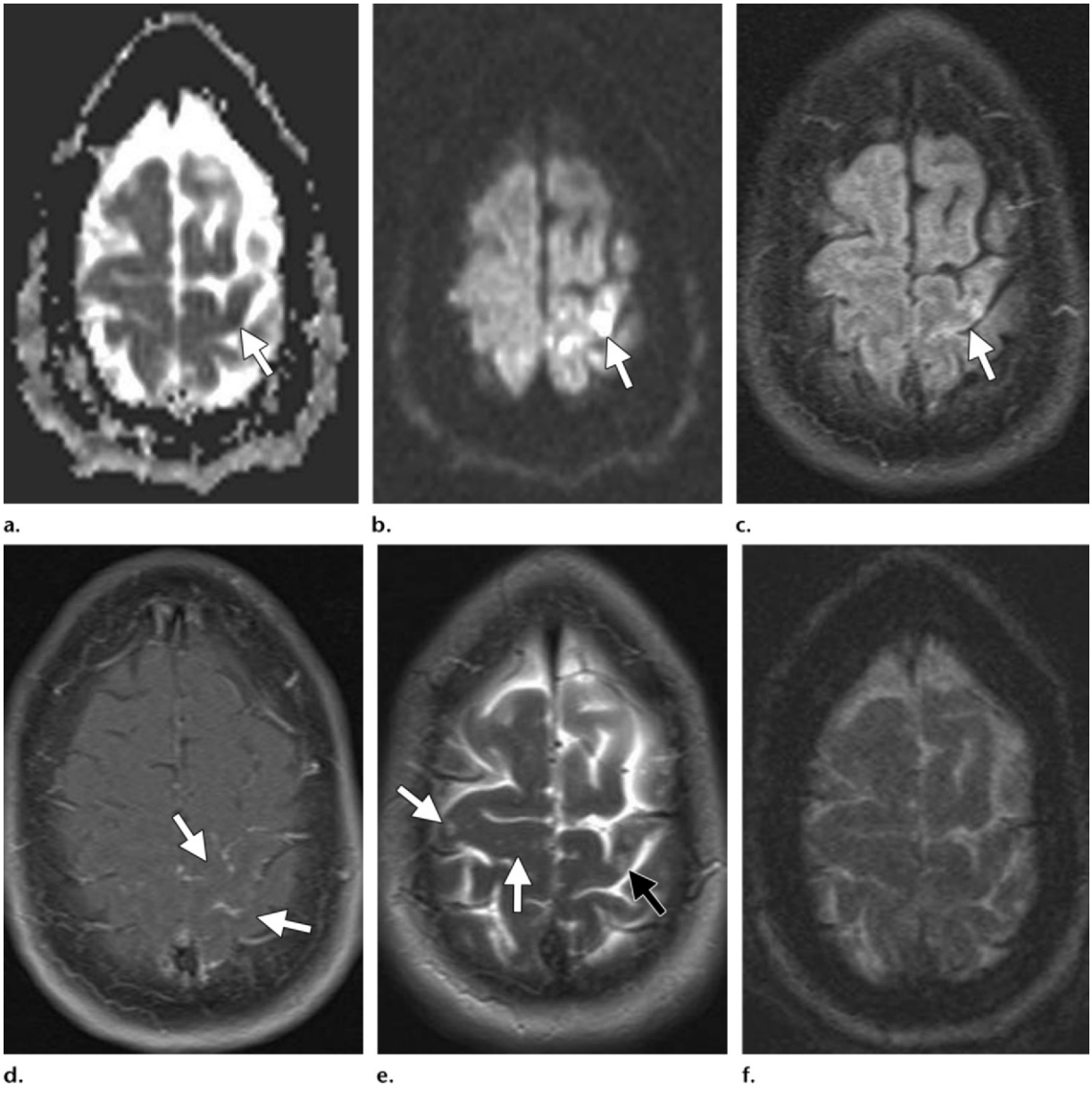

Figure 3. Early hyperacute stroke in a 49-year-old woman with right lower extremity weakness and rigidity. **(a, b)** ADC map **(a)** and diffusion-weighted MR image **(b)** show an area of restricted diffusion in the left motor cortex (arrow). **(c)** FLAIR image shows a corresponding area of slightly high signal intensity (arrow). **(d)** Gadolinium-based contrast-enhanced T1-weighted MR image shows arterial enhancement (arrows). No parenchymal enhancement is seen. **(e)** T2-weighted MR image shows an area of high signal intensity in the left motor cortex (black arrow), a finding indicative of a stroke that occurred more than 6 hours earlier (late hyperacute). Other scattered nonspecific subcortical areas of high signal intensity are also seen (white arrows), confounding the finding of late hyperacute stroke. **(f)** No hemorrhagic transformation is seen at susceptibility-weighted MR imaging. Because of the presence of early arterial enhancement and only slightly high signal intensity at FLAIR imaging, the stroke is likely less than 6 hours old (early hyperacute). In fact, this patient presented to the emergency department within 3 hours of the onset of symptoms.

(18,22). Arterial enhancement occurs in about 50% of patients with ischemic stroke and is most commonly seen 3 days after onset of symptoms; however, arterial enhancement is not specific to stroke (22–26).

Meningeal enhancement is the rarest type of enhancement. It occurs within the first week after onset of stroke, usually 2–6 days, with a peak on days 1–3. It usually occurs only after a large infarct in the adjacent meninges and is thought to be secondary to reactive hyperemia. Similar to arterial enhancement, meningeal enhancement usually resolves within the first week after stroke (18).

Parenchymal enhancement may be further subdivided into early and late enhancement.

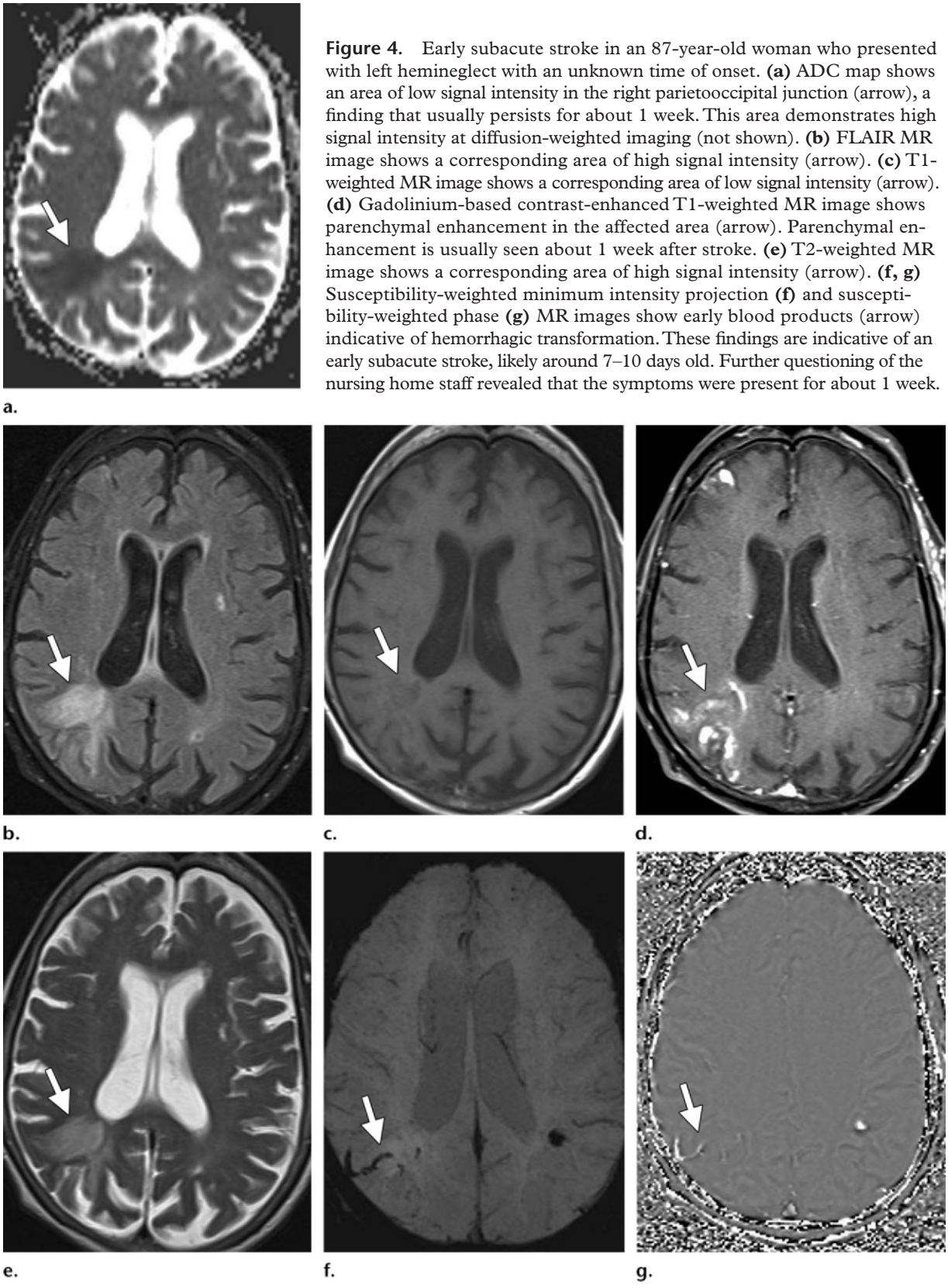

Figure 4. Early subacute stroke in an 87-year-old woman who presented with left hemineglect with an unknown time of onset. **(a)** ADC map shows an area of low signal intensity in the right parietooccipital junction (arrow), a finding that usually persists for about 1 week. This area demonstrates high signal intensity at diffusion-weighted imaging (not shown). **(b)** FLAIR MR image shows a corresponding area of high signal intensity (arrow). **(c)** T1-weighted MR image shows a corresponding area of low signal intensity (arrow). **(d)** Gadolinium-based contrast-enhanced T1-weighted MR image shows parenchymal enhancement in the affected area (arrow). Parenchymal enhancement is usually seen about 1 week after stroke. **(e)** T2-weighted MR image shows a corresponding area of high signal intensity (arrow). **(f, g)** Susceptibility-weighted minimum intensity projection **(f)** and susceptibility-weighted phase **(g)** MR images show early blood products (arrow) indicative of hemorrhagic transformation. These findings are indicative of an early subacute stroke, likely around 7–10 days old. Further questioning of the nursing home staff revealed that the symptoms were present for about 1 week.

In addition, there are two subtypes of early parenchymal enhancement. It commonly begins 5–7 days after complete infarction, around the time arterial and meningeal enhancement fades, although it may be seen earlier (Figs 4e, 5f). In most infarcts, parenchymal enhancement is seen between 1 week and 2 months after stroke; most

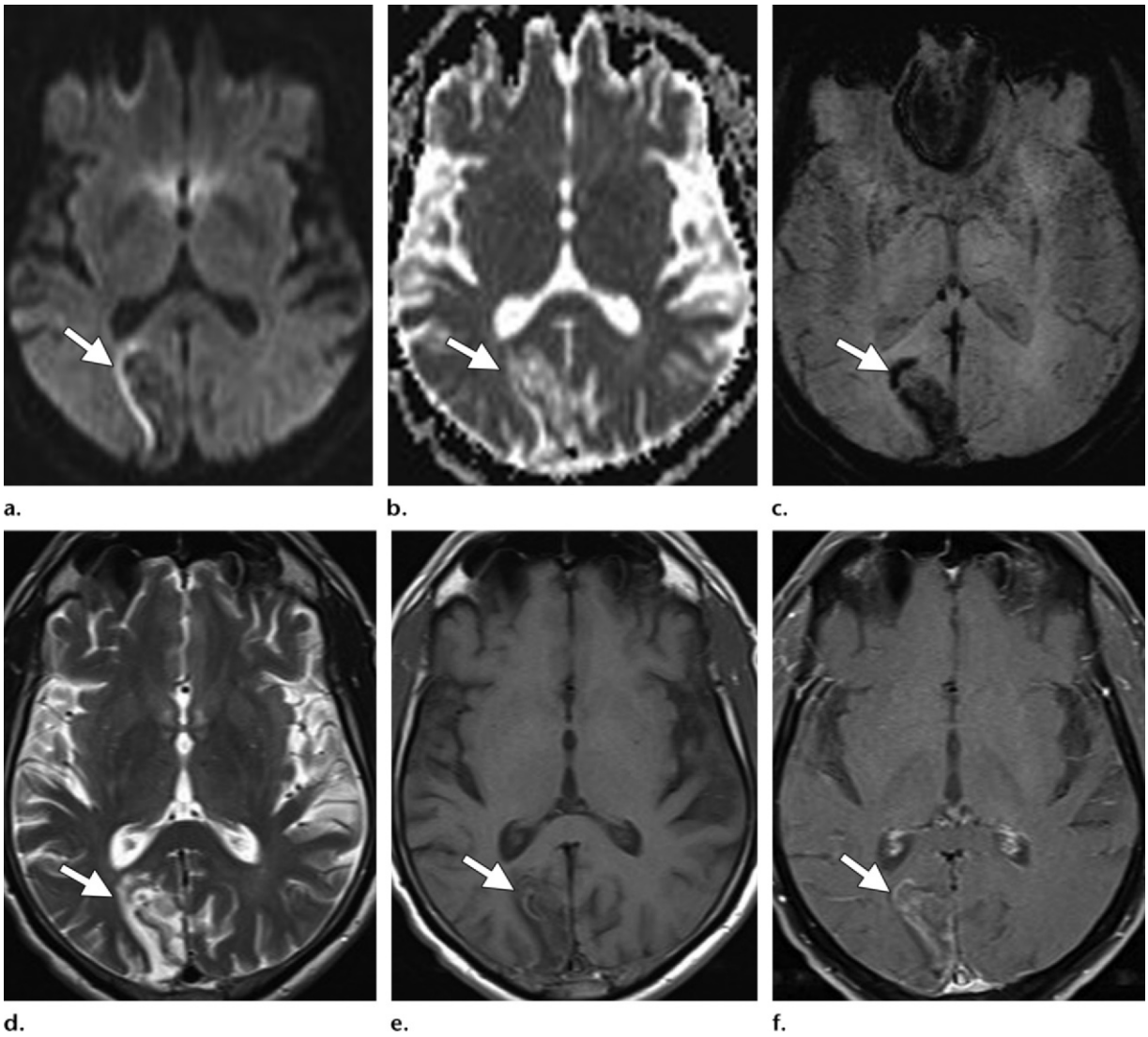

Figure 5. Chronic stroke in a 67-year-old man with a history of head and neck cancer. MR imaging was performed to further evaluate an area of hypoattenuation in the right occipital lobe at recent fused positron emission tomography/computed tomography (PET/CT). **(a)** Diffusion-weighted MR image shows an area of low signal intensity in the right occipital lobe (arrow) with a peripheral rim of high signal intensity, a finding that may be due to T2 shine-through. **(b)** ADC map shows a corresponding area of high signal intensity (arrow). **(c)** Susceptibility-weighted MR image shows hemorrhagic products (arrow) in the right occipital lobe. **(d)** T2-weighted MR image shows an area of high signal intensity in the right occipital lobe (arrow). **(e)** T1-weighted MR image shows a corresponding area of low signal intensity (arrow). **(f)** Contrast-enhanced T1-weighted MR image shows a corresponding area of parenchymal enhancement (arrow). These findings are indicative of a chronic stroke that is likely 3 weeks to 2 months old. At further questioning, the patient reported experiencing recent left visual field defects, but he could not remember exactly when they started.

infarcts do not enhance after this time, although parenchymal enhancement may be seen as much as 4 months after infarction (19,22–24,27,28).

Teaching Point

If parenchymal enhancement persists longer than 8–12 weeks, a diagnosis other than ischemic stroke should be sought (3). In cortical infarction, parenchymal enhancement may be gyriform, and in the basal ganglia and brainstem it may be generalized or ringlike. Recently, lacunar infarcts were found to enhance more

intensely than cortical infarcts, and watershed infarcts may enhance earlier than thromboembolic infarcts (29).

In incomplete infarction, a separate entity, parenchymal cortical enhancement, may be seen earlier, about 2–4 hours after the ischemic insult. Incomplete infarction is defined as selective loss of cortical neurons, with survival of glia and vascular structures after moderate ischemia (30). In incomplete infarction, enhancement is often very intense and disappears by 24–48 hours after the ischemic event. It is thought to result from either iatrogenic vessel occlusion or a cerebral embolus with very early reperfusion. Incomplete infarction is associated with a good prognosis (18,19,24,31).

Teaching Point

Several studies have reported that the presence of early parenchymal enhancement within 6 hours of stroke is associated with a higher risk for clinically significant hemorrhagic transformation, particularly when it is seen in the deep gray matter or basal ganglia (32–34). It should be noted that the aforementioned enhancement patterns (arterial, meningeal, and parenchymal) overlap and that they may be seen in a "transition phase," which may occur around 4–6 days after stroke (18).

Gradient-echo and Susceptibility-weighted Imaging

Hemorrhagic Transformation.—Gradient-echo and susceptibility-weighted sequences are the most sensitive sequences for depicting hemorrhagic transformation in patients with ischemic stroke, particularly susceptibility-weighted imaging, which is routinely performed in all patients with stroke at our institution. Hemorrhagic transformation demonstrates a spectrum of findings ranging from small petechial areas of microbleeding to large parenchymal hematoma. Several studies reported that microbleeding is present in one-half to the majority of patients with ischemic stroke and is seen around 48 hours after onset of symptoms (3). These areas of bleeding are thought to be secondary to diapedesis of red blood cells across a leaky and damaged blood-brain barrier. A recent study reported that one-half of patients who present with microbleeding develop more areas of microbleeding within the next 5 years (35). These areas of microbleeding are not associated with a worse outcome, and guidelines state that the presence of fewer than five areas of microbleeding on initial MR images does not contraindicate thrombolysis because they are not associated with increased adverse outcomes (7,36).

Parenchymal hematoma is a rarer type of hemorrhagic transformation that results from vessel wall rupture caused by high reperfusion pressure. It is more common with cardioembolic events, is associated with hyperglycemia, most commonly occurs in the basal ganglia, and confers a much worse prognosis (37,38).

Teaching Point

Hemorrhagic transformation is rare in the first 12 hours after stroke onset (the hyperacute stage), particularly within the first 6 hours. When it occurs, it is usually within the first 24–48 hours and, in almost all cases, is present 4–5 days after stroke (3,34,36,39). Late hemorrhagic transformation is less common but may occur 1 week after stroke.

Cortical and Pseudolaminar Necrosis.—Cortical laminar and pseudolaminar necrosis cause serpiginous cortical T1 shortening, which is not caused by calcium or hemoglobin products; rather, it presumably results from some other unknown substance or paramagnetic material, possibly lipid-laden macrophages (40–44). High cortical signal intensity may be seen on T1-weighted images 3–5 days after stroke, and in many cases it is seen about 2 weeks after stroke. Thereafter, it increases in intensity and fades after about 3 months but, in some cases, it may persist for more than a year (43–45). In patients with suspected cortical laminar necrosis, susceptibility-weighted imaging may help differentiate it from hemorrhagic transformation (42,46,47).

Current Thrombolytic and Neurointerventional Techniques

As reported in the European Cooperative Acute Stroke Study III (ECASS III), the window for administering an intravenous tissue plasminogen activator (tPA) was recently extended to 4.5 hours after onset of stroke symptoms (7,48). Alteplase, an intravenous tPA, is the only thrombolytic therapy approved by the United States Food and Drug Administration (FDA). It should be noted that the following sections are guidelines and that treatment plans are decided

by the medical team. For example, none of these interventions are approved to treat stroke in children, although this appears to be changing, and guidelines are emerging (49,50). The following interventions may be combined as the medical team sees fit to optimize patient care. In addition, many physicians believe that off-label uses of devices may be the new standard of care and that no time window is absolute (51,52).

Catheter-directed Intraarterial Thrombolysis

Intraarterial thrombolysis may be considered in patients with an anterior circulation stroke that is 6 hours old or less, who are ineligible for intravenous thrombolysis, or in whom intravenous thrombolysis was unsuccessful (53). In patients with a stroke less than 3 hours old and large vessel occlusion with a considerable diffusion or perfusion mismatch, it was recently proposed that intraarterial thrombolysis be the first-line treatment, although this strategy is controversial (54,55). Intraarterial thrombolysis may be performed as much as 24 hours after a posterior circulation stroke, although there are no established guidelines (56). The Prolyse in Acute Cerebral Thromboembolism (PROACT) trials (phases I and II) demonstrated the safety and efficacy of prourokinase as an intraarterial thrombolytic when administered within the first 6 hours after onset of symptoms. Patients with proximal middle cerebral artery occlusion had the best response. Agents used for intraarterial thrombolysis include urokinase, prourokinase, streptokinase, alteplase, and reteplase. Intraarterial thrombolysis is associated with higher recanalization rates than intravenous thrombolysis, particularly in patients with proximal occlusion; however, because of concern over delays in administering intraarterial thrombolytic agents, intravenous thrombolysis is considered the first-line method. In addition, intraarterial thrombolysis may be performed in patients who recently underwent surgery, a contraindication for intravenous thrombolysis. The recanalization rates for a combination of intravenous tPA and microinfusion intraarterial tPA catheters with ultrasonographic activation (particularly the EKOS Primo microcatheter [EKOS Corp, Bothell, Wash]) are superior to those for combined intravenous and intraarterial therapy, as reported in the Interventional Management of Strokes II trial. The ongoing Interventional Management of Strokes III clinical trial aims to prove the efficacy of combined intravenous and intraarterial tPA, including the use of embolectomy and mechanical disruption devices, compared with intravenous tPA alone (53). No thrombolytic agent has yet received FDA approval for intraarterial administration.

Embolectomy and Mechanical Disruption Devices

Recommendations for embolectomy and mechanical disruption devices, such as the MERCI clot retriever (Concentric Medical, Mountain View, Calif) and Penumbra aspiration system (Penumbra, Alameda, Calif), are that they may be used as much as 8 hours after onset of stroke in those with an anterior circulation stroke and for whom intravenous thrombolysis was ineffective or is not an option and as much as 24 hours after onset of stroke in those with a posterior circulation stroke (53). These devices are approved by the FDA. As reported in the PROACT trial, the MERCI clot retriever has lower recanalization rates than prourokinase and was superior when combined with intraarterial thrombolysis. At our institution, the guideline of employing an embolectomy device less than 8 hours after an anterior circulation stroke has been exceeded by as much as 6 hours with successful results.

Intracranial Angioplasty and Stenting

Maximal medical therapy is the treatment of choice in patients with substantial cerebrovascular atherosclerotic lesions, as evidenced by the Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS) trial. In patients with more than 70% symptomatic atherosclerotic lesions in whom optimal medical therapy is ineffec-

tive, revascularization with angioplasty or stenting is a feasible option (53). Emergent stenting is becoming increasingly popular; for this use, only one system (Wingspan Stent System with Gateway PTA Balloon Catheter [Boston Scientific, San Leandro, Calif]) has been approved by the FDA.

Conclusions

MR imaging may help determine the age of an ischemic stroke, particularly in elderly patients or those in a nursing home. Findings on ADC maps and diffusion-weighted, FLAIR, and T1- and T2-weighted gradient-echo and susceptibility-weighted images, including contrast enhancement patterns, may help classify strokes as early hyperacute, late hyperacute, acute, subacute, or chronic and provide useful information for the medical team and the patient's family. Recent data indicate that in many patients with restricted diffusion and no change on FLAIR images, it is more likely than was initially thought that the stroke is less than 6 hours old. The time window to administer intravenous tPA is currently 4.5 hours from the time when the patient was last seen to be normal, and for anterior circulation strokes, the time window for administering intraarterial tPA is 6 hours from when the patient was last seen to be normal. Some neurointerventionists use a cutoff of 8 or 24 hours from when the patient was last seen to be normal, depending on the blood vessels involved, to determine whether to intervene (51,52). However, patients are always evaluated on a case-by-case basis, and if restricted diffusion is present and findings on FLAIR images are negative, a diffusion-perfusion mismatch is seen, or salvageable penumbra is present, an interventionist may choose to act.

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Sequence-specific MR Imaging Findings That Are Useful in Dating Ischemic Stroke

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RadioGraphics 2012; 32:1285–1297 • **Published online** 10.1148/rg.325115760 • **Content Codes:** MR NR

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A good rule of thumb is that if the signal intensity on ADC maps is low, the stroke is less than 1 week old (2–4).

Page 1289
However, both clinical experience and most of the literature indicate that in most patients with ischemic stroke, findings on FLAIR images are positive 6–12 hours after onset of symptoms (14). For some neurointerventionists, the presence of restricted diffusion with negative findings at FLAIR imaging alone has been enough to initiate treatment.

Page 1292
If parenchymal enhancement persists longer than 8–12 weeks, a diagnosis other than ischemic stroke should be sought (3).

Page 1293
Several studies have reported that the presence of early parenchymal enhancement within 6 hours of stroke is associated with a higher risk for clinically significant hemorrhagic transformation, particularly when it is seen in the deep gray matter or basal ganglia (32–34).

Page 1293
Hemorrhagic transformation is rare in the first 12 hours after stroke onset (the hyperacute stage), particularly within the first 6 hours. When it occurs, it is usually within the first 24–48 hours and, in almost all cases, is present 4–5 days after stroke (3,34,36,39).