

ILLUSTRATIVE TEACHING CASES

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Delayed Cerebral Abscess Formation After Posterior Cerebral Artery Stroke in a Patient With Opioid Use Disorder

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CASE PRESENTATION

A 33-year-old right-handed man with opioid use disorder presented with right arm weakness and confusion. He was febrile, tachycardic, and somnolent but able to open his eyes to repeated verbal stimulation. There was minimal spontaneous speech output. Inattention limited full mental status testing. Other pertinent features of the examination included a right homonymous hemianopia, left gaze preference, right nasolabial fold flattening, and right hemiparesis. National Institutes of Health stroke scale score was 15. Head computed tomography (CT) demonstrated a hypodensity in the left temporal and occipital lobes. CT angiography revealed an abrupt cutoff of the left posterior cerebral artery (PCA) at the bifurcation of the parieto-occipital and calcarine arteries, consistent with a distal occlusion of the PCA. Given these imaging findings (completed infarction on CT, distal occlusion on CT angiography), and unknown last known well, he was not a candidate for reperfusion therapy.

He was started on empirical, broad-spectrum antibiotics out of concern for infective endocarditis (IE). Transthoracic echocardiogram revealed a moderately sized mobile mass attached to the aortic valve suspicious for a vegetation, supporting the diagnosis of IE based on the Duke criteria.¹ Serial blood cultures were positive for methicillin-susceptible *Staphylococcus aureus*. A large mediastinal abscess required multiple debridements. The patient was transitioned to nafcillin given multiple positive cultures with the same organism from both the blood and manubrium.

The patient underwent magnetic resonance imaging (MRI) of the brain 2 days later (Figure 1), confirming a large left PCA territory infarct. Given the extent of the infarct—involving both the thalamus and medial temporal lobes—it was hypothesized that a septic thrombus had initially occluded the proximal PCA (causing the large left PCA infarct) and subsequently embolized distally (as seen on vessel imaging).

On hospital day 11, the patient reported a worsening, frontal headache, and repeat MRI was obtained. This showed a large, peripherally enhancing region within the territory of the left PCA stroke (not shown). There was edema around the thalamus and 7 mm of midline shift.

Over the course of the next 10 days, he became gradually more somnolent. He responded to questions with brief, often single-word answers. He was unable to repeat simple phrases. Repeat MRI (Figure 2) showed marked progression in the degree of edema around the left thalamus as well as increased effacement of the ambient cistern (Figure 2C). There remained a thick rind of peripheral enhancement in the left PCA distribution (Figure 2D) as well as persistent diffusion restriction (Figure 2A and 2B).

The patient was brought to the operating room for a posterior temporal craniotomy out of concern for an evolving brain abscess. A large amount of purulent material was evacuated from an abscess cavity within the left PCA territory. Two days later, he was brought back to the operating room for stereotactic aspiration of a deeper collection within the left thalamus, yielding both purulent and necrotic brain tissue. Cultures from both the temporal

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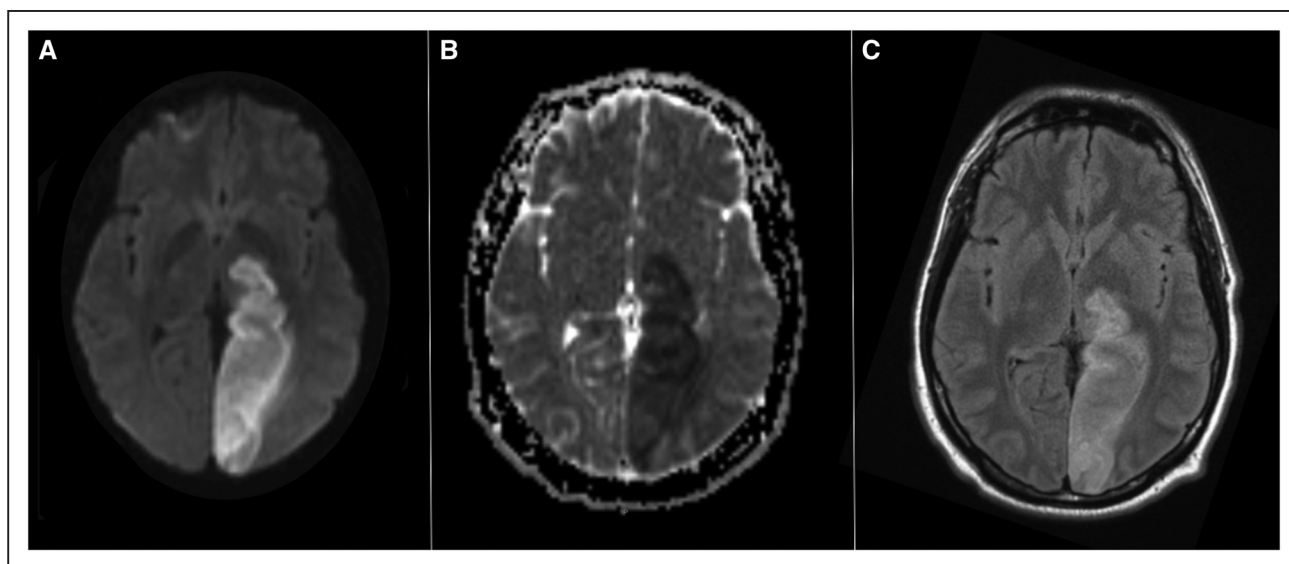


Figure 1. Initial magnetic resonance imaging (MRI) brain obtained on hospital day 2.

MRI without contrast obtained on hospital day 2 shows a large left posterior cerebral artery territory infarct with diffusion-weighted imaging hyperintensity (**A**) and associated apparent diffusion coefficient hypointensity (**B**). There was minimal edema seen on fluid-attenuated inversion recovery (**C**).

and thalamic abscesses were sterile. Cardiac surgery on the infected valve was not performed given his recent stroke and normal biventricular systolic function.

Over the next few days, he became more alert and interactive. The right homonymous hemianopia persisted throughout the remainder of his hospitalization, though

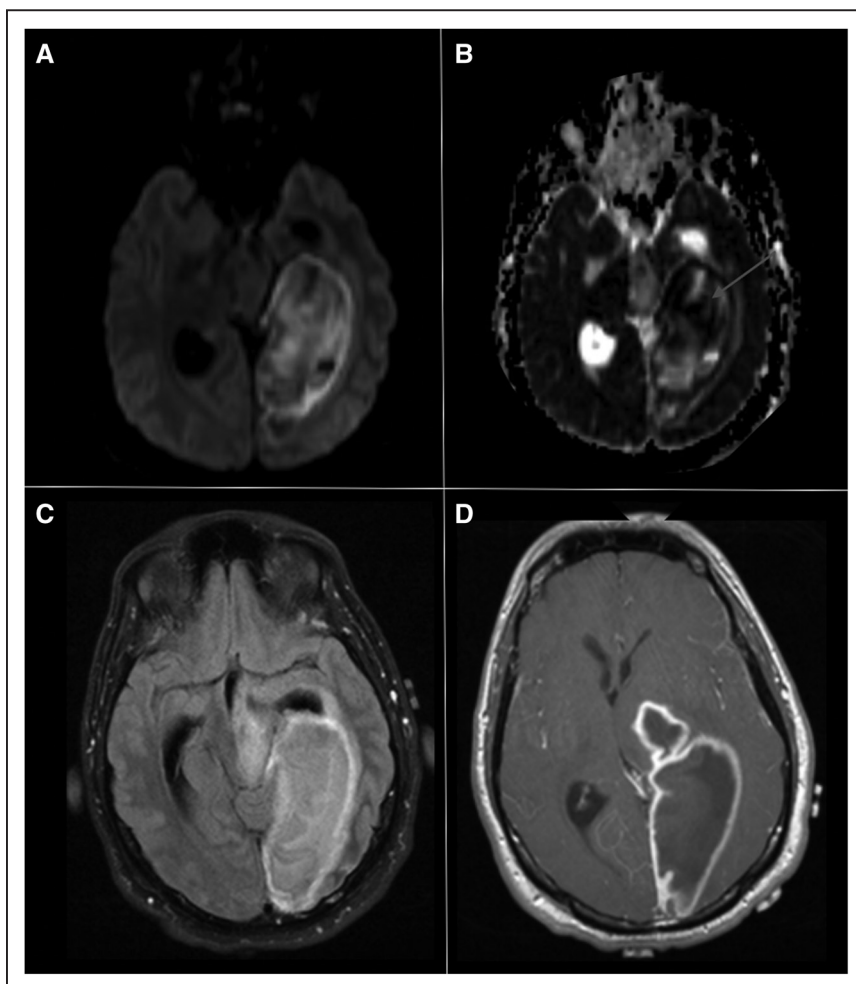


Figure 2. Repeat magnetic resonance imaging (MRI) brain obtained on hospital day 21.

Repeat MRI with contrast obtained on hospital day 21 demonstrated persistent diffusion-weighted imaging hyperintensity (**A**) and associated apparent diffusion coefficient hypointensity (**B**, arrow) with marked progression in the degree of edema around the left thalamus (**C**). There remained a thick rind of peripheral enhancement in the left posterior cerebral artery distribution on T1 postcontrast imaging (**D**). Precontrast T1 not shown.

his right hemiparesis improved. He was continued on nafcillin for an additional 4 weeks after his last neurosurgical procedure and discharged to a rehabilitation facility after 36 days in the hospital. Four months later, he was conversant, able to name, repeat, and comprehend. Writing and reading remained difficult. He was able to ambulate independently without assistance.

DISCUSSION

There are myriad neurological complications of IE including both embolic and hemorrhagic stroke, encephalopathy, mycotic aneurysm, and meningitis (Table). A recent retrospective analysis of >1300 patients with left-sided IE demonstrated that >25% of patients experienced at least one neurological complication, stroke being the most common.² Brain abscess, meanwhile, is relatively rare, affecting roughly 1% to 7% of patients with IE.³ This patient presented with an embolic stroke in the setting of methicillin-susceptible *Staphylococcus aureus*

aortic valve endocarditis. Though he received appropriate antibiotics early in his course, earlier diagnosis and surgical management of the brain abscesses could have improved his outcome.

Radiographically, it can be challenging to distinguish between acute ischemic stroke and cerebral abscess on MRI as both may lead to restricted diffusion, edema, and enhancement with gadolinium. Yet, the evolution of these imaging findings over time can help differentiate between the two.

Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps have become instrumental in the rapid diagnosis and localization of stroke. The restricted diffusion of water molecules, detected by these sequences, is not unique to cerebral ischemia, however, and may result from any form of cellular injury that disrupts the flow of water such as active demyelination, recurrent seizure, tumor, and infection.

Acute ischemic stroke is characterized by DWI hyperintensity and ADC hypointensity. Although variable to some degree, the DWI signal generally becomes iso- to mildly hypointense within 14 days.⁴ Because of T2-signal shine-through, DWI hyperintensity may persist for several weeks in some instances and may not be a reliable indicator for estimating infarct age.⁵ Meanwhile, increasing hypocellularity of the infarcted brain tissue leads to increased diffusivity and thus ADC hyperintensity.

Although cerebral abscesses have early DWI hyperintensity, a key distinguishing feature from ischemic stroke is ADC hypointensity, which is thought to result from the hypercellular, highly viscous abscess cavity.⁴ In this patient, there was DWI hyperintensity and ADC hypointensity for more than 3 weeks after his initial stroke (Figure 2A and 2B). Persistent ADC hypointensity was thus a key distinguishing feature supporting abscess formation.

Understanding the development of cerebral edema over time may also aid in differentiating stroke-associated swelling from abscess. Edema following ischemic stroke involves both cytotoxic and vasogenic features. Cytotoxic edema occurs early, within minutes of stroke onset, as energy-dependent exchange pumps fail along the cell membrane and fluid shifts from the extracellular to intracellular space. As such, there is translocation of water from one compartment to another but minimal increase in total brain volume. Vasogenic edema, meanwhile, leads to a net increase in brain water content given endothelial dysfunction and breakdown of the blood-brain barrier. This begins several hours later and is thought of as the primary driver of associated mass effect and herniation.

Well-characterized in patients with large hemispheric ischemic stroke, malignant edema is associated with significant morbidity and mortality. Recent volumetric analyses have demonstrated that swelling is an important secondary complication of smaller strokes as well.⁶ Moreover, the degree of edema is an independent predictor of poor outcomes. Younger individuals, such as

Table. Neurological Complications of Infective Endocarditis

Neurological complication	Timing of onset	Relevant diagnostic studies to consider
Ischemic stroke and TIA	Hyperacute to acute	CTA head and neck
		MRI brain
		Transthoracic and/or transesophageal echocardiogram
Intracerebral hemorrhage	Hyperacute to acute	CTA head and neck
		MRI brain with contrast
		PTT, PT, INR
Seizure	Hyperacute to acute	Continuous EEG
		MRI brain
		Lumbar puncture
		Urine and serum toxicology screen
Encephalopathy	Acute to subacute	Continuous EEG
		MRI brain with contrast
		Blood cultures
		Basic laboratory studies
		Lumbar puncture
Encephalitis and meningitis	Acute to subacute	MRI brain with contrast
		Lumbar puncture
Headache	Acute to subacute	CT head
		MRI brain with contrast
		Lumbar puncture
Cerebral abscess	Subacute	CT
		MRI head with contrast
Mycotic aneurysm (unruptured)	Subacute to chronic	CTA head
		Conventional cerebral angiography

Hyperacute refers to onset of symptoms in seconds to minutes. Acute refers to hours to 1 to 2 d, subacute to a few days, and chronic to several days to weeks. CT indicates computed tomography; CTA indicates CT angiography; INR, international normalized ratio; MRI, magnetic resonance imaging; PT, prothrombin time; PTT, partial thromboplastin time; and TIA, transient ischemic attack.

Take Home Points

- Cerebral abscess is a rare, although important, complication of infective endocarditis.
- Poststroke edema has both cytotoxic and vasogenic components, with peak swelling 3 to 5 days after stroke onset.
- There has been a dramatic increase in the number of strokes related to infective endocarditis during the opioid epidemic.
- Consider alternative etiologies for progressive swelling and neurological decline occurring >7 days after ischemic stroke.
- Though antibiotics alone may be appropriate for initial management of a small cerebral abscess, surgical drainage is often required in patients with large abscesses and clinical deterioration.

this patient, are particularly vulnerable; possibly due to the lack of age-related brain atrophy.⁷

Although ischemic stroke is the most common cause of cytotoxic edema, vasogenic edema may result from any lesion that affects the permeability of the blood-brain barrier, such as tumor, encephalitis, and, importantly, brain abscess.⁸

While poststroke edema can vary in both onset and duration, peak swelling typically occurs 3 to 5 days after the stroke. Our patient underwent repeat imaging 11 and 21 days after admission. MRI at both time points demonstrated a peripherally enhancing lesion in the territory of the left PCA with progressive edema around the left thalamus and extension into the rostral midbrain, associated with worsening midline shift. These findings were thus inconsistent with poststroke swelling and more likely the result of delayed cerebral abscess formation with vasogenic edema. Only after failure of intravenous antibiotics and clinical deterioration was the patient brought to the operating room for surgical exploration and drainage confirming presence of the abscesses.

Over the past decade, there has been a dramatic increase in the number of strokes related to IE and opioid use.⁹ Intravenous opiate use, and the associated risk of infectious endocarditis, likely accounts for the rising incidence of stroke associated with the current opioid epidemic.¹⁰ It is important for neurologists to be aware of both stroke and abscess as important complications of IE. Understanding the clinical course and imaging features of these conditions over time can help distinguish between the two.

ARTICLE INFORMATION

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Disclosures

None.

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