**Case Report**

A 79-year-old white woman presented with a 4-day history of fever and decreased mental status. A CT scan showed a 3 x 2-cm rounded area of low attenuation in the left temporo-parietal region. This lesion appeared to be contiguous with a second 0.9-cm area of low attenuation that was slightly more anterior and adjacent to the left splenium of the corpus callosum. Marked vasogenic edema extended into the high left parietal lobe, with mass effect on the left lateral ventricle. This abnormal attenuation extended to the left lateral ventricle, which was filled with soft-tissue attenuation consistent with debris. Mild subfalcine herniation (1–2 mm) was evident.

T2-weighted MR images showed a 3 x 2 x 2-cm, multiloculated, centrally hypointense mass lesion with a peripheral rim of hypointensity and surrounding edema in the left occipitoparietotemporal region. Anteriorly, the lesion abutted the atrium and occipital horn of the left lateral ventricle. A fluid-fluid level (hypointense relative to CSF) was evident, filling 90% of the left lateral ventricle. Also noted was a dilute pus-CSF level in the dependent portion of the atrium of the right lateral ventricle. On contrast material-enhanced T1-weighted images, the lesion exhibited peripheral ring enhancement. In addition, linear enhancement was present in the left lateral ventricular margins, but the material that filled the ventricle was not enhancing (Fig 1B). Diffusion-weighted images showed marked hyperintensity in the center of the lesion, ventricular margins, bottom of the right lateral ventricle atrium (dilute pus), and 90% of the left lateral ventricular volume (corresponding to the pus that filled the ventricle) (Fig 1A and D). Corresponding ADC maps revealed prominent hypointensity in the center of the lesion. However, the hypointense signal intensity varied along the ventricular margins, at the bottom of the right lateral ventricle atrium (dilute pus), and in the purulent material in the left ventricle (Fig 2B and D). The MR imaging findings were consistent with a complicated intracerebral abscess that had ruptured into the ventricles.

The abscess was drained by placing a large-bore catheter. Twenty milliliters of tenacious and foul-smelling pus was drained. Cultures of pus grew *Streptococcus anginosus* (including *Streptococcus intermedius*, *Streptococcus constellatus*, *Streptococcus milleri*), gram-positive cocci, and *Actinomyces* organisms in a background of acute inflammation and necrotic cellular debris. Two days after the procedure, the patient had a communicating hydrocephalus, for which a right intraventricular catheter was placed. Despite these interventions, the patient died 2 days later.

**Discussion**

Brain abscesses represent organized foci of suppuration within the parenchyma. Clinical diagnosis is challenging, because the signs and symptoms often are nonspecific and overlap those of other intracranial mass lesions. Headache is the most frequent initial symptom of intracranial abscess. Other pre-
senting symptoms, roughly in order of descending frequency, include drowsiness and confusion; focal or generalized seizures; and focal motor, sensory, or speech disorders. While fevers are characteristic during the invasive phase of cerebral abscesses, the temperature may normalize as the abscess becomes encapsulated. Therefore, the diagnosis is best established by using radiologic modalities (2). The evolution of abscess is characterized by four stages: early cerebritis, late cerebritis, early capsule formation, and late capsule formation. Most patients receive this diagnosis when the abscess is in the stage of late cerebritis or mature formation. The period that is required for the formation of a mature abscess varies, ranging from 2 weeks to several months. Morbidity due to a brain abscess generally results from brain
herniation due to mass effect or, less commonly, rupture of the abscess cavity into the ventricular system, as in this case with acute hydroceholus. Edema surrounding a brain abscess may exceed the volume of the abscess itself, producing marked mass effect. In the event of abscess rupture and subsequent communication with the ventricular system, ependymitis develops, and enhancement of the ventricular lining is present, in addition to the characteristic ring enhancement around the abscess cavity. Such development heralds a poor prognosis. The clinical picture, along with laboratory findings, may be helpful in narrowing the diagnosis, but radiologic examination has become invaluable for confirmation. Usually, drainage of the abscess is necessary by any means, so that appropriate antibiotics can be administered to treat the cultured organisms. Lumbar puncture in the setting of cerebral abscess is potentially detrimental, particularly when intracranial pressure is elevated, in which case diagnosis based on CT or MR imaging findings becomes crucial (2, 3).

Diffusion-weighted imaging has been shown to be useful for the evaluation of a myriad of disease processes, including cerebral infarction, tumors, demyelinating disease, and vertebral compression fractures (4). It provides unique information about the molecular diffusion properties of water in healthy and diseased brain parenchyma. Ebisu and colleagues (1) reported on a case of cerebral abscess in which the diffusion-weighted images demonstrated hyperintensity in the abscess cavity (in vivo) as well as in the aspirated abscess fluid (in vitro); both of these findings corresponded to a low ADC value.

The ADC in the extracellular space is dictated by a tortuosity factor that represents the length of the path of a molecule traversing in a porous medium that approximates the brain tissue (5). In the normal condition, water molecules easily traverse through the extracellular space by means of random brownian motion. In the setting of an organized abscess, the movement of water molecules is influenced by the content of the abscess, which consists of variable amounts of inflammatory cells, microorganisms, and proteins (1, 4, 6–8). All of these components are large macromolecules. Martin and colleagues (9) showed that the ADC value is inversely proportional to the protein concentration. As a result, the macromolecules retarded diffusion of water molecules within an abscess cavity. In addition, water molecules in an abscess are bound to carboxyl, hydroxyl, and amino groups on the surfaces, further limiting their translational movement (4). These factors most likely explain the imaging features of an abscess cavity (high signal intensity on diffusion-weighted images and low ADC values); they are similar to other processes producing restricted water diffusion.

Recently, Ramsay and colleagues (10) reported on a cerebral abscess and subdural empyema on diffusion-weighted images obtained without ADC maps. Both empyema and brain abscess revealed the same level of increased signal intensity. In our case in which a cerebral abscess ruptured into the left lateral ventricle, the abscess core had hyperintensity on diffusion-weighted images with corresponding ADC hypointensity; these results confirmed the findings reported by other groups. Of note, although the intraventricular pus had the same degree of hyperintensity on diffusion-weighted images as that of the abscess core, it had variable signal intensity on the corresponding ADC map (intermediate to hypointense signal with an appearance that was never as dark as that in the abscess cavity). A layer of fluid in the dependent portion of the atrium of the right lateral ventricle had ADC values that ranged from very high to intermediate and low. This finding was most likely related to a gradient of concentrated macromolecules (ie, proteinacious material), cells and cellular debris, and microorganisms within pus that were diluted by CSF to a greater extent and, thus, were better depicted in the right ventricle. It indicated that the ADC map, which depicted regional variation in ADC values, was more sensitive than diffusion-weighted imaging in showing subtle change in the content or concentration of pus in the abscess. Therefore, ADC maps are important for extracting quantitative information that is lost on diffusion-weighted images. The uniformly intense brightness of both intracavitary and intraventricular pus on diffusion-weighted images probably was related to the combination of opposing effects of restricted water diffusion and T2 shine-through.

Another interesting finding in this case was that linear signal intensity changes (hyperintensity on diffusion-weighted images and hypointensity on ADC maps) were identified along the left lateral ventricular wall and the left part of septum pellucidum, despite the absence of a true abscess formation in these regions. The cause for this finding is unclear, although a possible explanation is restriction of water movement along the ventricular wall as a result of edema. Analogous findings are depicted on diffusion-weighted images of multiple sclerosis plaques; a low ADC value is found in the very early lesion in which marked inflammation is present without substantial demyelination (11). This finding is postulated to be compatible with shifts of intracellular water protons and charges that alter membrane permeability. Furthermore, an influx of inflammatory cells and macromolecules destroys water diffusion (11). Enhancement in the ventricular lining and corresponding low ADC values in the setting of ependymitis can be explained with a similar mechanism. Given these findings, one might predict that high signal intensity on the diffusion-weighted image and a low ADC value also are observed in early and late cerebritis, even before pus is evident. Intraparenchymal edema, however, may negate this effect.

**Conclusion**

Diffusion-weighted imaging is useful in distinguishing a brain abscess from a necrotic or cystic tumor because of the specific signal intensity profile of an
abscess that shows restricted water movement. ADC maps can further depict subtle variation in the nature and content of pus within the abscess cavity, ventricle, and extraaxial space; therefore, they may be useful in the characterization of an abscess for therapeutic planning and follow-up. Finally, ependymitis may be hyperintense on diffusion-weighted images and may have a low ADC value, for which the explanation is restricted water movement as a result of an inflammatory reaction.

References