Imaging Sciences Interesting Cases

CASE 48

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CLINICAL PRESENTATION: A 14-month-old female presents with right-sided weakness after sustaining a posterior head injury. There was questionable loss of consciousness.

IMAGING FINDINGS: A non-contrast CT of the head was unremarkable. Due to the persistent symptoms, a head MRI and MRA were obtained to evaluate for occult mass or vascular abnormality. The MRI revealed an area of high signal within the left basal ganglia, external capsule, and subcortical parietal white matter on diffusion weighted imaging (**Fig. 1A**) with corresponding low signal on the area diffusion coefficient (ADC) map (**Fig. 1B**) compatible with restricted diffusion. The area exhibits low signal on T1- and high signal on FLAIR and T2-weighted images (**Figs. 2A and B**, respectively). There is no abnormal enhancement on post-contrast sequences (**Fig. 3**). Head MRA revealed patent anterior, middle, and posterior vasculature without malformation.

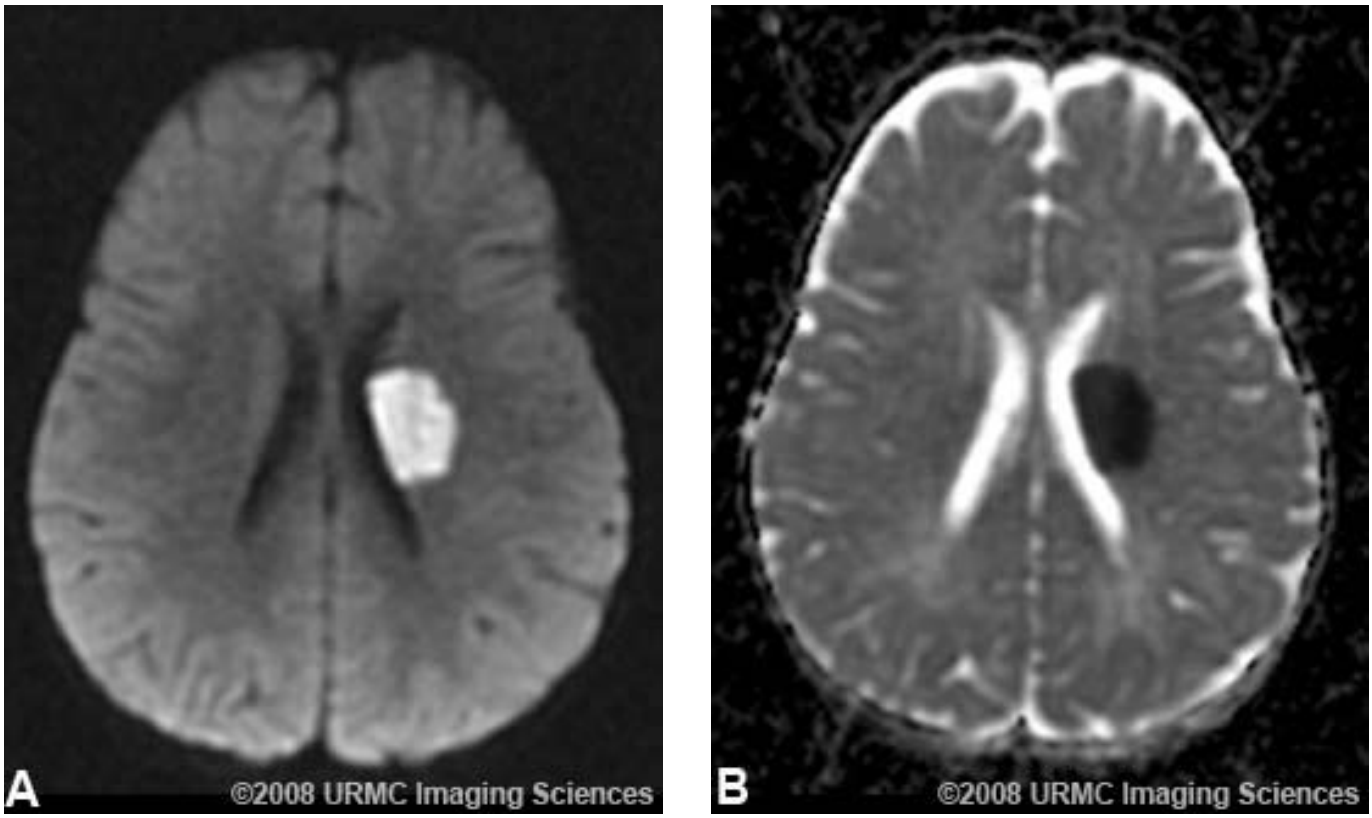

Figure 1: (**A**) Axial DWI demonstrates high signal within the left corona radiata and body of the caudate nucleus. (**B**) Low signal is seen in the same region on the corresponding ADC map indicating restricted diffusion.

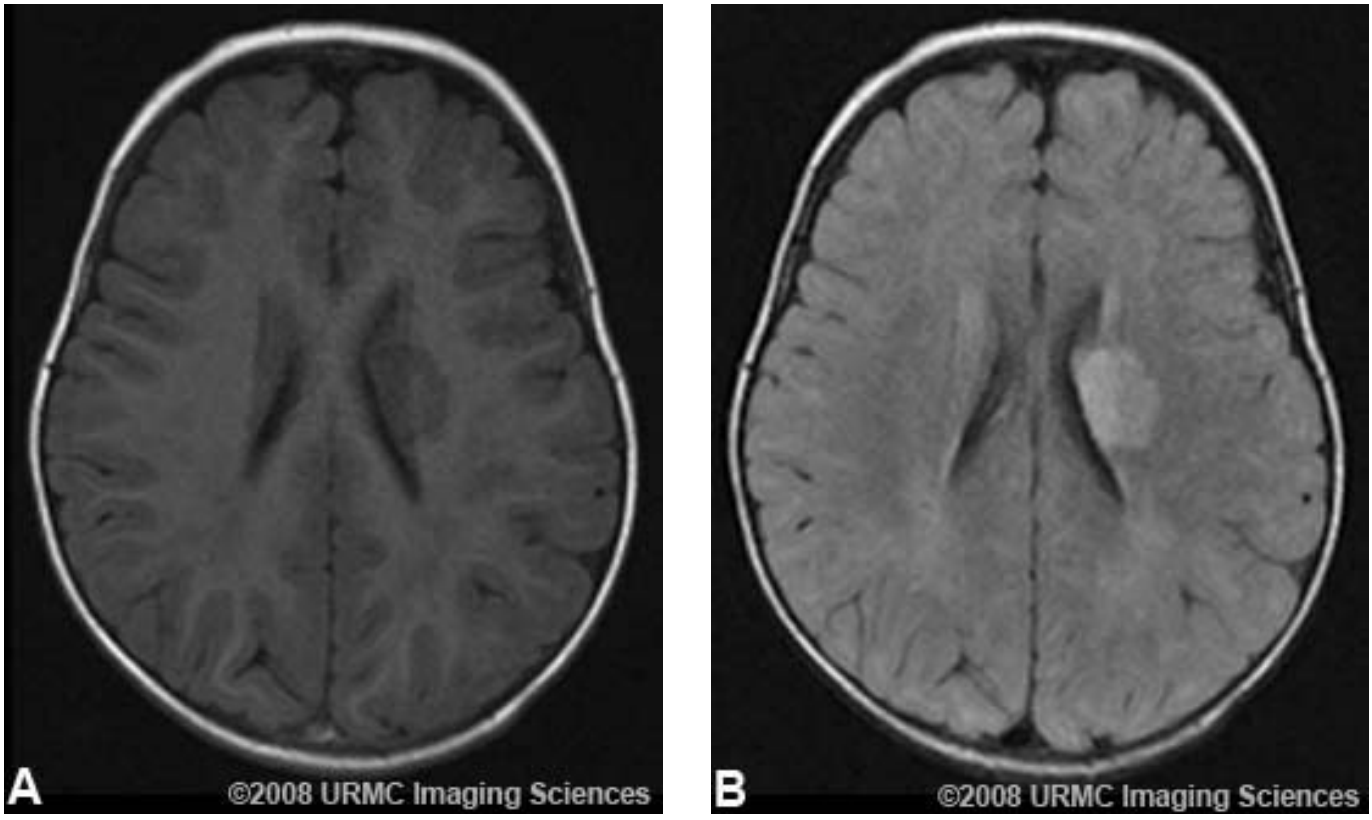

Figure 2: Axial images through the area of interest reveal well-demarcated low signal on T1-weighted (**A**) and high signal on T2-weighted FLAIR (**B**) sequences characteristic of an hyperacute infarct.

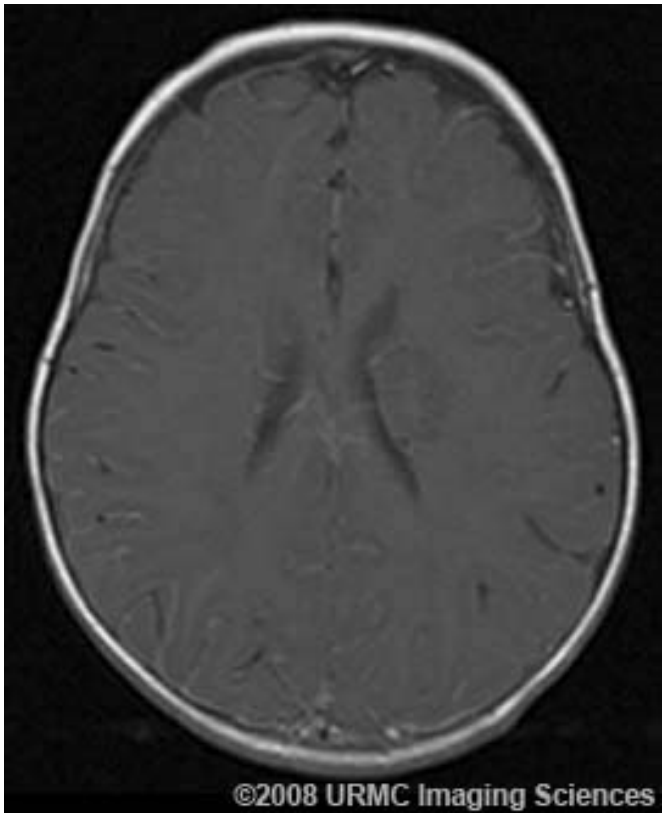

FIGURE 3: Axial T1 with fat suppression post contrast administration shows no enhancement of the affected area.

DIAGNOSIS: Subacute infarct in a left MCA distribution.

DISCUSSION: Cerebral infarction is the third leading cause of death behind heart disease and cancer and is the leading neurologic cause of long-term disability in the United States. Although there are multiple

causes, thromboembolic disease is the most prevalent. Most patients present with neurologic deficits, however the clinical findings are often nonspecific. The specificity of diagnosis is markedly improved with adjunct imaging. The early detection of cerebral infarction is important for identifying patients that may undergo targeted treatments such as thrombolysis or thrombectomy.

Although non-contrast computed tomography (NCCT) of the brain is excellent at detecting changes associated with acute infarction, up to 50% of patients with infarcts have no findings on initial CT imaging. MRI, particularly diffusion-weighted imaging (DWI), is highly sensitive for detecting ischemic injury associated with early infarction. On a cellular level, neuronal ischemia leads to failure of membrane ionic channels. This results in the intracellular accumulation of sodium ions followed by a net movement of water into the cell producing cytotoxic edema.; Diffusion of the intracellular water molecules out of the cell is restricted by the cell membranes.

In DWI, an initial gradient pulse disrupts the normal homogenous magnetic field causing the hydrogen molecules of water to spin, or precess. A second gradient pulse is applied with opposite magnitude resulting in spin rephrasing. Under normal circumstances, water molecules freely diffuse in the interval between pulses, resulting in a net reduction of spin rephrasing and decreased signal strength on DWI. However in areas of restricted diffusion, there is no net movement of protons resulting in near perfect spin rephrasing manifest by high signal on DWI.

Diffusion data can be presented as signal intensity (DWI) or as an image map of the apparent diffusion coefficient (ADC). The behavior of water molecules is not symmetric and may show uneven distribution of the ADC when measured in one direction. ADC values are therefore measured in several directions with the combined imaging data composed into an ADC map, producing a direction-insensitive measurement of diffusion. An area of low ADC corresponds to high signal intensity on DWI and represents restricted diffusion. DWI should always be evaluated in conjunction with ADC maps to distinguish true restriction (high on DWI and low on ADC) from lesions with "T2 shine-through" (high on both DWI and ADC).

The imaging findings on MRI in the setting of cerebral infarction depend upon the duration of ischemia. A common classification with associated findings is detailed below.

*Hyperacute (0-24 hrs): Within minutes, reduced proton motion is evident as high signal on DWI and reduced signal on the ADC map. By 2 hours, proton density/intermediate sequences may demonstrate loss of the normal arterial flow void in the occluded artery. Progressive cytotoxic and vasogenic edema results in low T1 and elevated T2 signal by 8 hours from the onset of ischemia.

*Acute (1-7 days): Progressive edema leads to persistent high T2 and low T1 signal. Mass effect on adjacent structures is usually present. The risk for hemorrhagic transformation is greatest in this period (24-48 hrs) due to reperfusion injury; blood products may alter characteristic T1 and T2 findings, however hemorrhage is readily detected on non-contrast CT.

*Subacute (7-21 days): Edema and associated mass effect slowly resolve. Areas of low T1 and high T2 signal may persist, but gradually diminish. The ADC map findings may reverse and become bright, reflecting T2 shine through on the DWI due to vasogenic edema.

*Chronic (>21 days): Edema completely resolves. Areas of irreversible ischemia undergo encephalomalacia with ex-vacuo phenomena—ventricular enlargement and widening of the cortical gyri and fissures. High T2-signal CSF fills the space previously occupied by brain parenchyma.

Perfusion-weighted MR imaging, although not performed in the case presented, may be obtained to identify areas of potentially reversible ischemia.In such instances, contrast enhanced perfusion images demonstrate a much larger area of low signal than the corresponding DWI ischemic core.The "penumbra" indicates potentially reversible ischemia and its presence may be a determining factor in targeted therapies to prevent irreversible damage.

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