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# Neuroimaging Highlight

Editors: William Hu, Mark Hudon

## Global Cerebral Anoxia

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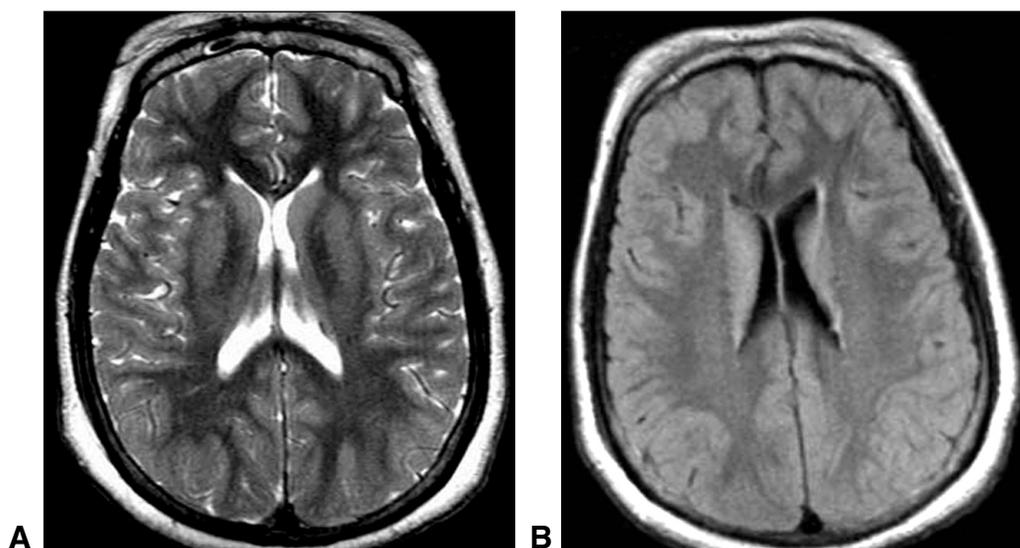
A 57-year-old, previously healthy tourist sustained closed-head trauma during a high-speed motor vehicle collision. She was found unconscious at the scene with a Glasgow coma scale score of 3. She suffered a multitude of injuries including frontal scalp lacerations and cervical cord injury. Upon arrival at the hospital, she suffered cardiopulmonary arrest of up to 12 minutes without a perfusing rhythm and was intubated.

The initial cerebral CT examination revealed no discernible abnormality. The following day, the MR showed subtle hyperintense T2 signal diffusely involving the cortical gray matter (Figure 1 A,B). This was much more evident on diffusion-weighted imaging (DWI) where the areas of restricted diffusion involved the cortex and predominated in the posterior cerebral hemispheres (Figure 2 A,B). MR angiography showed patent major intracranial vasculature and no evidence of neck arterial injury (Figure 3 A,B).

Three days following the initial insult, the follow-up CT study demonstrated loss of gray-white matter differentiation with generalized parenchymal edema, mild effacement of the basal cisterns and a relatively hyperdense cerebellum (Figure 4). This reflects a poor prognosis.<sup>1</sup> There was progressive clinical decline, but the patient was maintained on life support on compassionate grounds so that she could return to her homeland.

Global hypoxia results in cerebral injury primarily due to decreased perfusion. In this case, stagnation and anoxia are the two causative factors of global hypoxia. Other factors include: anemia, hypoglycemia and histotoxins.<sup>2</sup> What ultimately determines the degree of hypoxic brain damage is the severity and duration of the initial insult. Increased patient age, increased body temperature, elevated glucose level and decreased blood pH may also contribute to the magnitude of damage.<sup>3</sup>

The distribution of injury is based on the principle of selective



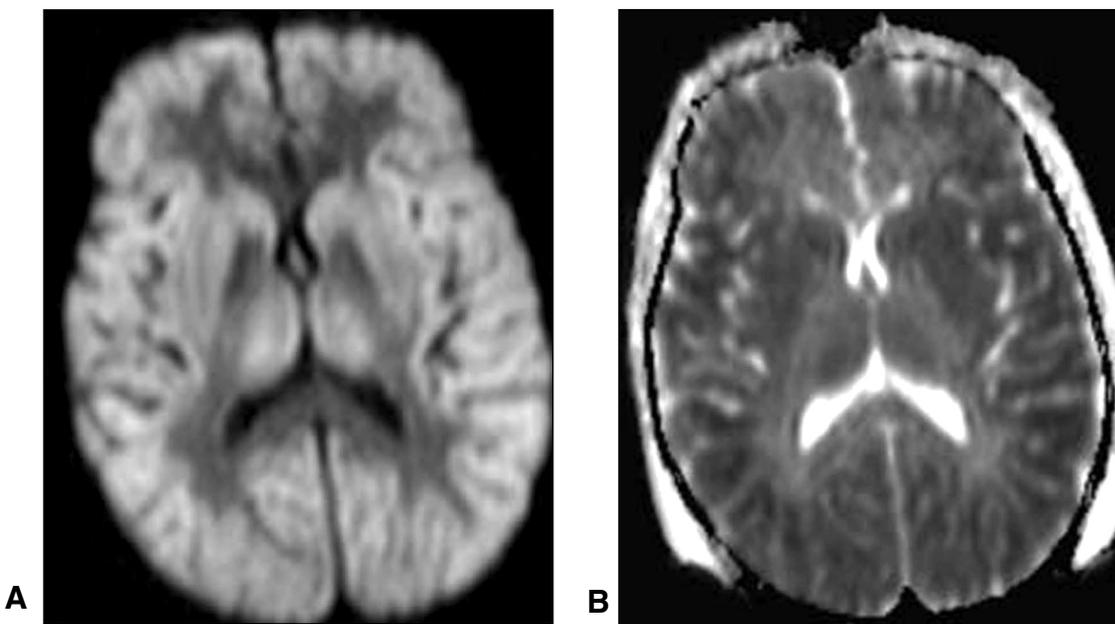
**Figure 1A:** MRI (Axial T2) and **Figure 1B:** MRI (Axial FLAIR) demonstrate mild cortical hyperintensity.

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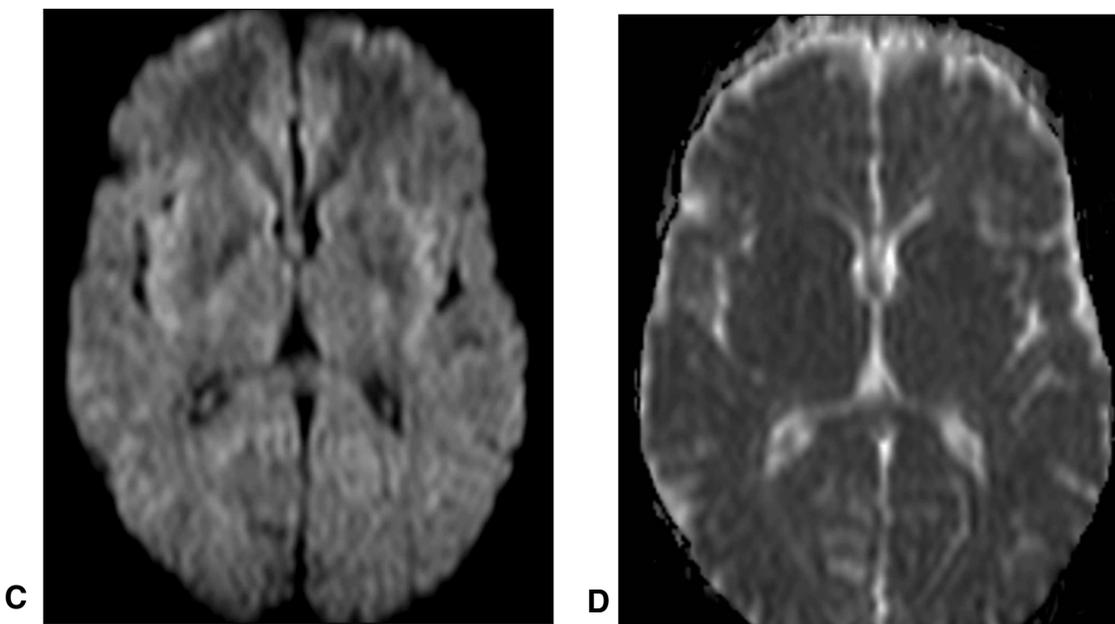
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**Figure 2A:** MRI (DWI) and **Figure 2B:** MRI (ADC) demonstrate restricted diffusion involving the cortical gray matter, basal ganglia, and thalami.



**Figure 2C and D:** Normal DWI and ADC of another patient for comparison.

vulnerability of cell types. There is preferential involvement of the gray matter with generalized ischemia/ anoxia early on since gray matter possesses a higher metabolic rate, a specific autoregulatory response, and receptors for excitatory amino acids that are presumed to be responsible for neuronal necrosis. White matter injury in the late subacute stage is thought to result secondary to Wallerian degeneration from neuronal injury,

although early hypoxic leukoencephalopathy has been described.<sup>4</sup>

Irreversible neuronal injury tends to occur following greater than four to six minutes of global ischemia.<sup>1</sup> Although early CT findings include diffuse sulcal effacement, hypodense cortical gray matter with loss of gray-white matter differentiation, hypodense basal ganglia, and hypodense watershed gray matter



A

**Figure 3A:** MRA of the circle of willis shows no abnormalities.



B

**Figure 3B:** MRA neck arteries shows no evidence of injury.



A



B

**Figure 4A and 4B:** Axial CTs demonstrate decreased cortical gray matter and white matter density with loss of gray-white interface and effacement of the basal cisterns. Hypodensity of the basal ganglia, thalami, and brainstem are also seen. The cerebellum appears to have relatively increased density.

regions, these may not be recognized in the acute stages. Conventional MRI findings may also be quite subtle and DWI may actually be most sensitive for evaluating global cerebral anoxia effects and determining the age of insult.<sup>5</sup> Cytotoxic edema results in restricted diffusion and hyperintense signal changes. In the acute period (< 24 hours), hyperintense DWI signal (hypointense ADC) is seen in the cortex, basal ganglia, and cerebellum. In the early subacute period (24 hours – 13 days), these findings become more evident on conventional sequences. In the late subacute period, there are diffuse hyperintense white matter changes. Although prognosis is generally poor, diffusion-weighted imaging may be beneficial in establishing the diagnosis and prognosis, as well as aiding management.

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