An Unusual Pattern of White Matter Diffusion Restriction in Pediatric Patients

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Purpose
To describe a novel pattern of hemispheric white matter diffusion abnormality in pediatric patients following hypoxic and toxic insults of varying causes. Even though the mechanism underlying the development of this unusual pattern of white matter diffusion restriction is unknown, delayed demyelination is suspected as the common pathologic event.

Materials & Methods
Cases of isolated cerebral white matter diffusion restriction on MR imaging in infants and children following anoxic or toxic insults of varying etiologies were collected from the radiology databank of a pediatric hospital between the years 1999 and 2002. Hospital admission and discharge records, as well as MR imaging and proton MR spectroscopy studies were reviewed. Specific clinical information of interest included birth history, toxic exposure, metabolic derangement, cause and severity of the hypoxic insult and neurologic condition immediately following the event. Relevant laboratory data, including electroencephalogram (EEG) results, erythrocyte sedimentation rate, electrolytes, arterial blood gas values, and arylsulfatase A activity were reviewed, when available. Also, hospital and clinic records 1 month to 2 years following the hypoxic or toxic exposure were reviewed to determine neurologic outcome following hospital discharge.

Results
Ten patients, ranging in age from newborn to 15 years, were identified, all of whom experienced hypoxic insults of varying etiologies, including seizure, intraoperative respiratory failure, near drowning, strangulation, toluene exposure, and perinatal ischemic encephalopathy. In all but one instance, there was a delayed hypoxic insult with subsequent profound clinical deterioration with widespread white matter signal abnormalities, including marked alterations of the white matter apparent diffusion coefficients. Analysis of laboratory results revealed respiratory and metabolic acidoses in eight of the 10 cases with arterial pH values ranging from 6.85 and 7.37. The five cases in which an EEG was obtained were normal. No significant electrolyte abnormality was identified, and the two cases in which arylsulfatase levels were tested were within normal
limits. Follow-up of all patients included in our study revealed severe residual neurologic deficits. No definitive cause of this unusual clinical and radiologic outcome was elucidated for nine of the 10 pediatric patients identified. In one case, exposure to toluene was documented, and believed to be a cause of the white matter diffusion restriction.

**Conclusion**

Significant neurologic deterioration with profound cerebral white matter diffusion restriction as seen on MR imaging is an uncommon observation following hypoxic and toxic insults. The etiology underlying the development of this condition generally is not known, but proposed causes include demyelination and oligodendrocyte injury. This includes a mechanism of widespread demyelination described by Weinberger et al. (1), in which there is a proposed association with a pseudodeficiency of arylsulfatase A. Also, in one of our cases, inhalational exposure to toluene was documented. The mechanism of this white matter abnormality is believed to be a result of demyelination with deposition of iron (2). However, the mechanism of the white matter diffusion restriction in the majority of our patients remains unknown. Further investigations will be necessary to determine if these varying causes of diffusion restriction abnormalities are caused by similiar biochemical processes.

**References**


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