MRI Aids Diagnosis of New-Onset Afebrile Seizures

BY DIANA MAHONEY
New England Bureau

LOS ANGELES — Magnetic resonance imaging is useful for identifying the etiology of new-onset afebrile seizures in infancy and, when available, should be included as part of the standard diagnostic evaluation in this population, according to Dr. William S. Benko.

Of 103 MRIs performed in 144 infants presenting to the emergency department of Children’s National Medical Center in Washington between January 2001 and January 2005 with new-onset afebrile seizures (NOAS), 40% detected diagnostic abnormalities related to the seizures, Dr. Benko said in a presentation at the annual meeting of the Child Neurology Society.

By comparison, in 136 of the 144 infants who underwent CT, abnormalities were detected in 42% of the scans; however, 15% of these were deemed incidental findings.

Fifteen of the infants with normal CT scans had abnormal MRIs. “In the majority of these CT-negative, MRI-positive patients, the CT scans did not identify focal abnormalities,” said Dr. Benko of the National Institute of Neurological Disorders and Stroke in Bethesda, Md.

According to practice parameters published in 2006 by the Quality Standards Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society, the evaluation of a first nonfebrile seizure in a child should include EEG as a routine part of the diagnostic evaluation to predict the risk of recurrence and to classify the seizure type and epilepsy syndrome (Neurology 2000;55:616-23).

There was insufficient evidence for the routine use of other studies, such as lumbar puncture and neuroimaging, so these were deemed warranted only under specific circumstances and at a neurologist’s discretion, according to the parameters.

More studies with large, well-characterized samples were needed, according to the seizure department, 82% of the infants had experienced two or more seizures. All of the patients were evaluated by a child neurologist on the basis of the patient history, laboratory values, including complete blood count, electrolytes, urinalysis, and toxicology screen; 24-hour observation; electroencephalography; and CT, MRI, and/or lumbar puncture at the clinician’s discretion.

This MRI sagittal image shows a single subependymal lesion not seen on CT.

A proton density axial image shows tubers of tuberous sclerosis complex.

In this afebrile population, we found that CBC, urinalysis, and toxicology screens were not at all useful or contributory,” Dr. Benko said. Of 59 infants who were given lumbar punctures, 96% had normal results and 5% had evidence of pleocytosis. Treatable electrolyte abnormalities, including hypocalcemia, hyponatremia, and hypoglycemia were detected in five of the children. Electroencephalography showed abnormal results in 62% of patients, including focal abnormalities, hypsarrhythmia, and generalized spikes.

By comparison, neuroimaging results were particularly revealing, Dr. Benko said. With CT, congenital malformations were identified in 12% of the patients imaged, evidence of trauma was seen in 5%, and atrophy was evident in 3%.

Among the patients scanned by MRI, dysplasia was seen in 14%. These abnormalities included focal diffuse involvement, dysgeneses, heterotopias, Aicardi syndrome, and Dandy Walker malformation.

MRI also detected vascular events in 8% of patients. These included new and old cerebrovascular accidents, hemorrhagic, subdural hematoma, and Sturge-Weber syndrome.

The abnormalities noted on MRI but missed on CT included dysplasia, mesial temporal sclerosis, cerebrovascular accidents, and tuberous sclerosis, he said. “The yield of CT-negative, MRI-positive findings was high in patients with focal neurologic exam, neurodevelopmental delay, focal EEG, focal seizure, [and MRI evidence of right mesial temporal sclerosis].”

The decision to perform MRI was made by the treating neurologist, raising the possibility of a selection bias, Dr. Benko said.

In addition, the findings are limited by the fact that not all the patients who underwent CT or also underwent MRIs, concluded Dr. Benko, who conducted the investigation during his pediatric neurology fellowship at Children’s National Medical Center.

Pediatric Brain-Death Guidelines Often Ignored, Update Needed

BY ROBERT FINN
San Francisco Bureau

SAN FRANCISCO — Pediatric brain-death guidelines were followed to the letter in only 1 case of 142 that resulted in organ donation during a 5-year period in Southern California. Dr. Mudit Mathur reported at the annual congress of the Society of Critical Care Medicine.

“There is an urgent need for the update and revision of these criteria. … What we need are clear, consistent, uniform, and reliable guidelines in terms of brain-death diagnosis, declaration, documentation, and reporting,” said Dr. Mathur, a pediatric critical care specialist at Loma Linda University Children’s Hospital.

The guidelines, issued in 1987 by the American Academy of Pediatrics Task Force on Brain Death in Children, call for the evaluation of 14 clinical elements in determining that a child is brain dead (Arch. Neurol. 1987;44:587-8). (See sidebar.)

A review showed that the charts of the 142 children declared to be brain dead contained documentation of a median of 6 of the 14 elements considered crucial to establishing brain death. Involvement of a pediatric intensivist in the diagnosis did not result in more elements being recorded.

The AAP guidelines call for the documentation of 14 elements to be recorded in patient charts for brain death.

... What we need are clear, consistent, uniform, and reliable guidelines in terms of brain-death diagnosis, declaration, documentation, and reporting,” said Dr. Mathur, a pediatric critical care specialist at Loma Linda University Children’s Hospital.

Elements for Brain Death Declaration

In his study, Dr. Mathur and his colleagues examined charts of pediatric organ donors for documentation of the following 14 elements that should be considered before declaring a child to be brain dead, according to 1987 guidelines (Arch. Neurol. 1987;44:587-8):

- Documented etiology of coma
- Coexistence of coma and apnea
- Flaccid tone, no movements
- Absence of pupillary reflex
- Absence of corneal reflex
- Absence of gag reflex
- Absence of cough reflex
- Absence of eye movement with doll’s eye maneuver
- Absence of respiratory effort
- Absence of hypothermia
- Absence of hypotension
- Irreversibility of changes
- No history of drug or metabolic intoxication
- Absence of respiratory effort on apnea test.

Source: Dr. Mathur