

# **Original Articles**

# **Multimodality Comparison of Neuroimaging** in Pediatric Traumatic Brain Injury

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Traumatic brain injury is a common cause of death and disability in children; early neuroimaging has assumed an increasingly important role in evaluating the extent and severity of injury. Several imaging methods were assessed in a study of 40 children with traumatic brain injury: computed tomography (CT), T<sub>2</sub>-weighted magnetic resonance imaging (MRI), fluidattenuated inversion recovery (FLAIR) MRI, and susceptibility-weighted imaging (SWI) MRI to determine which were most valuable in predicting 6-12 month outcomes as classified by the Pediatric Cerebral Performance Category Scale score. Patients were subdivided into three groups: (1) normal, (2) mild disability, and (3) moderate/severe disability/persistent vegetative state. T2, FLAIR, and SWI showed no significant difference in lesion volume between normal and mild outcome groups, but did indicate significant differences between normal and poor and between mild and poor outcome groups. Computed tomography revealed no significant differences in lesion volume between any groups. The findings suggest that T<sub>2</sub>, FLAIR, and SWI MRI sequences provide a more accurate assessment of injury severity and detection of outcome-influencing lesions than does CT in pediatric traumatic brain injury patients. Although CT was inconsistent at lesion detection/outcome prediction, it remains an essential part of the acute traumatic brain injury work-up to assess the need for neurosurgic intervention. © 2007 by Elsevier Inc. All rights reserved.

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#### Introduction

Traumatic brain injury is a major cause of morbidity and mortality in children and is responsible annually for more than 500,000 emergency room visits, approximately 95,000 hospital admissions, 7000 deaths, and 29,000 permanent disabilities. The majority of children with traumatic brain injury present with minor head trauma and a single, significant clinical sign or symptom indicating underlying neuroimaging identifiable pathology has not been shown [1]. Predicting long-term outcome after traumatic brain injury remains unreliable, and efforts to do so have generated much controversy. Because few overt clinical signs have been demonstrated to correlate with the underlying neuropathology, clinicians increasingly rely on radiologic evidence for the assessment of the severity and extent of neuronal injury. Because decisions about patient management often hinge on such diagnostic information, it is important to obtain the most accurate depiction possible of neuronal damage following traumatic brain injury.

In the acute trauma work-up of traumatic brain injury patients, computed tomography is important for the rapid detection of extra-axial hemorrhage (e.g., subdural or epidural hematomas), acute hydrocephalus, fractures, or other intracranial lesions that may require acute neurosurgical intervention [1-5]. Magnetic resonance imaging appears to be very sensitive for intraparenchymal lesion detection in traumatic brain injury patients, but frequently is not easily obtainable acutely after injury [2,6-7]. Magnetic resonance imaging sensitivity is understood to be superior to computed tomography for intracranial evaluation. However, despite the newer technological improvements for both modalities, there have been no reports that have directly compared the two modalities as to their relative sensitivity and specificity in detecting injury and

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in predicting outcome in children. Initial studies performed in the 1990s compared computed tomography to magnetic resonance imaging in adults [2-4,6-11].

Each of the magnetic resonance imaging sequences used in the present study has unique detection capabilities. T<sub>2</sub> and fluid-attenuated inversion recovery (FLAIR) are common sequences in magnetic resonance imaging evaluation of traumatic brain injury patients. T2 is a more standard technique, generally able to detect a wide range of intraparenchymal lesions including edema, infarction, demyelination, moderate to large hemorrhages, and other parenchymal lesions [12]. FLAIR, a more recent technique, may be more sensitive in detecting some lesions within the parenchyma, including edema, particularly near the cortex or ventricles [13-15]. It is unknown whether there is any significance of intraparenchymal edema leading to more adverse outcomes compared with hemorrhage. In recent studies, we have demonstrated that a new gradient echo magnetic resonance imaging technique known as susceptibility-weighted imaging is also very useful in detecting hemorrhagic lesions associated with diffuse axonal injury [16,17]. This sequence, which utilizes the paramagnetic properties of deoxyhemoglobin, is more sensitive for detection of extravascular blood products [18].

The objective of the present study was to determine which of these methods of detection of pediatric traumatic brain injury was most accurate at predicting outcome.

#### **Patients and Methods**

#### Patient Selection and Data Collection

We studied 40 children and adolescents with traumatic brain injury admitted to Loma Linda University Children's Hospital from March 2001 to September 2002. This was an inception cohort of patients with traumatic brain injury without sufficient neurologic recovery in the first day who warranted magnetic resonance imaging during hospitalization for additional neurologic assessment of their injury. We excluded children with (1) previous head injuries; (2) pre-existing neurological disorders; (3) central nervous system malformations; (4) developmental disability; (5) psychiatric illness; (6) missile injuries; (7) suspected nonaccidental trauma; (8) age less than 1 month; or (9) poor-quality magnetic resonance imaging studies. These patients were part of a larger ongoing study examining magnetic resonance imaging for outcome prediction after various forms of acute central nervous system insult, approved by our institutional review board.

A neurosurgeon or neurologist examined all patients before magnetic resonance imaging was acquired. Clinical variables included age, initial Glasgow Coma Scale (GCS) score, initial mean arterial blood pressure, heart rate, arterial pH, hematocrit, serum glucose and sodium levels, and the number of days in coma, days on a ventilator, and days of hospitalization. Intracranial pressure and cerebral perfusion pressure could not be attained for all patients and therefore was excluded from our analysis.

Clinical variables and extent of lesion detection on magnetic resonance imaging and computed tomography were compared with outcomes obtained at 6 to 12 months after injury. Outcomes were determined without knowledge of imaging results using the Pediatric Cerebral Performance Category Scale (PCPCS) score, a six-point system modified from the Glasgow Outcome Scale score [19,20]. The six categories were

(1) normal: performs all age-appropriate activities; (2) mild disability: interacts at most age-appropriate activities, mild neurologic deficits; (3) moderate disability: performs age-appropriate activities of daily living, has significant cognitive impairment; (4) severe disability: conscious, dependent on others for daily support; (5) vegetative state; and (6) death. For outcome analysis, the six PCPCS scores were stratified into three categories: N, normal/no disability (n = 14); M, mild disability (n = 16); and P, moderate/severe disability/persistent vegetative state (n = 10). In this cohort, no patients died after study entry. Some categories were grouped for statistical analysis due to the small sample size.

### **Imaging**

A computed tomographic scan with standard and bone algorithms was obtained for all patients using a multidetector computed tomography scanner (General Electric, Milwaukee, WI). These were obtained in the acute setting of traumatic brain injury within 24 hours.

When medically stable, all children were transported to the magnetic resonance scanner and monitored by staff from the intensive care unit or transport team and the radiology personnel. Magnetic resonance imaging was obtained a mean of  $7\pm4$  days after injury. Studies were performed using a circularly polarized head coil in a conventional 1.5-T whole-body imaging system (Magnetom Vision; Siemens Medical Systems, Iselin, NJ) with a standard head coil.

Routine magnetic resonance imaging included axial dual spin-echo [proton density and T<sub>2</sub>-weighted] (TR = 2500 milliseconds; TE = 22/80 milliseconds; 5 mm thick), sagittal T<sub>1</sub>-weighted spin echo (TR/TE = 550/22 milliseconds; 5 mm thick), axial fluid attenuated inversion recovery (TR/TE = 9,000/110 milliseconds, TI = 2200 milliseconds, 5 mm thick), axial two-dimensional gradient recalled echo (FLASH, TR/TE = 500/18 milliseconds; flip angle = 15 degrees; bandwidth of 78 Hz/pixel, 4-5 mm thick), and axial three-dimensional FLASH susceptibility-weighted (TR/TE = 57/40 milliseconds, flip angle = 10 degrees; bandwidth of 78 Hz/pixel, 2 mm thick) sequences as described previously by Reichenbach et al. [18] and Tong et al. [16]. Diffusion-weighted imaging and magnetic resonance spectroscopy data were also obtained for this patient population (these are being analyzed separately). For the present study, only computed tomography, T<sub>2</sub>, FLAIR, and susceptibility-weighted imaging were analyzed.

Magnetic resonance imaging studies were reviewed on a clinical workstation (DS3000, Impax; Agfa, Teterboro, NJ) to determine study quality and then were downloaded for off-line analysis of parenchymal lesions. Observers were blinded to clinical variables and outcome. Lesions studied included parenchymal hemorrhages and edema, and were defined as predominantly hyperintense/hyperdense (FLAIR, T<sub>2</sub>, computed tomography) or hypointense (susceptibility-weighted imaging) parenchymal abnormalities that were not compatible with vascular, bony, or artifactual structures on conventional imaging. Known artifactual components from interventional procedure, ventricular shunt placement, or any other explainable phenomenon not associated with traumatic brain injury were not included. If there was doubt as to the origin of the lesions they were not included. Extra-axial or intraventricular hemorrhages and definite large vessel infarcts were excluded from the analysis.

Due to the variability in lesion shape, all images were analyzed using a computer software program (Image Pro Plus, Media Cybernetics Inc.) that semiautomatically counted and traced the outline of each lesion using threshold intensities assigned by one of the investigators (G.A.S., K.A.T., J.P.N., or C.J.W.). After predefined, observer-dependent adjustments, the program automatically counted and calculated the digital area of lesions in each image. The true area was determined by a conversion factor involving original pixel size calculated by field of view and matrix size. After converting digital area to actual size, the true area of each lesion was multiplied by the image slice thickness to determine the volume of each lesion. All lesions in each image were counted and summed, and the volume of all lesions was summed to obtain the global number and volume load of lesions per patient, respectively.

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