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Original Article

Post-Traumatic Epilepsy in Children—Experience From a Tertiary Referral Center

Jun T. Park MD [a,b,*], **Harry T. Chugani MD** [b]

[a] *Division of Pediatric Epilepsy, Department of Pediatrics, Rainbow Babies & Children's Hospital, Case Western Reserve University School of Medicine, Cleveland, Ohio*
[b] *Divison of Pediatric Neurology, Department of Pediatrics, Children's Hospital of Michigan, Wayne State University School of Medicine, Detroit, Michigan*

ABSTRACT

BACKGROUND: Post-traumatic epilepsy after a traumatic brain injury occurs in 10%-20% of children. Unfortunately, a biomarker that could provide prognostic information about both post-traumatic epilepsy and cognitive development is lacking. In this first of a series of studies, we have reviewed and analyzed clinical variables in children following traumatic brain injury to understand the epidemiologic and clinical characteristics of post-traumatic epilepsy in our urban population. **METHODS:** We performed a retrospective electronic chart review of patients who had suffered traumatic brain injury and subsequently evaluated at Children's Hospital of Michigan from 2002 to 2012. Various epidemiologic and clinical variables were analyzed. **RESULTS:** Patients who had severe traumatic brain injury and post-traumatic epilepsy had an abnormal acute head computed tomography. These patients had increased number of different seizure types, increased risk of intractability of epilepsy, and were on multiple antiepileptic drugs. Hypomotor seizure was the most common seizure type in these patients. There was a high prevalence of patients who suffered nonaccidental trauma, all of whom had severe traumatic brain injury. **CONCLUSIONS:** This study demonstrates a need for biomarkers in children following traumatic brain injury to reliably evaluate the risk of post-traumatic epilepsy.

Keywords: post-traumatic epilepsy (PTE), nonaccidental trauma (NAT), seizure semiology, hypomotor seizures, epileptic spasms, traumatic brain injury (TBI), brain injury and seizures

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Introduction

Post-traumatic epilepsy (PTE) is a common cause of morbidity in children after a traumatic brain injury (TBI), occurring in 10%-20 % of children following severe TBI.[1-5] A diagnosis of PTE is reserved for patients who have had two or more unprovoked seizures following TBI.[6] Unfortunately, the pathogenesis of PTE remains poorly understood, and there are no biomarkers at this time to help predict reliably the risk of epilepsy and, thus, prevent PTE[7,8] and its comorbidities.

At the Children's Hospital of Michigan (Detroit), a level I pediatric trauma center, we have been applying advanced neuroimaging techniques to elucidate the mechanisms associated with PTE to identify an accurate biomarker that could provide prognostic information regarding both PTE and cognitive development following TBI. In this first of a series of studies, we have reviewed and analyzed clinical variables in children who had suffered TBI to understand the epidemiologic and clinical characteristics of PTE in our urban population.

Study design and methods

A retrospective electronic chart review was performed on patients who had suffered TBI and subsequently referred

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* Communications should be addressed to: Dr. Park; Division of Pediatric Epilepsy; Department of Pediatrics; Rainbow Babies & Children's Hospital; Case Western Reserve University School of Medicine; Cleveland, Ohio.
E-mail address: jun.park@uhhospitals.org

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to a child neurologist (any one of the seven neurologists in both inpatient and outpatient) at Children's Hospital of Michigan from 2002 to 2012. These patients were identified after interrogating a large database of all new patients maintained in the Division of Pediatric Neurology. A separate database was then constructed on patients who had received various diagnoses that included PTE, focal neurological deficit(s), postconcussive syndrome, and persistent headaches. A total of 321 patients' charts were reviewed and, of these, 47 patients were diagnosed with PTE. These 47 subjects form the basis of the current report.

Multiple clinical variables in these 47 subjects were reviewed: age at TBI, sex, perinatal complications, type of trauma, that is, accidental (AT) and nonaccidental (NAT), imaging and neurophysiologic data, degree of TBI (mild, moderate, severe), surgical interventions, time of first seizure, seizure types and semiology based on descriptions and/or neurophysiologic data, antiepileptic medications, treatment response, follow-up duration, and outcome. Seizure onset time was classified as immediate (<24 hours after injury), early (<1 week after injury), or late (>8 days after injury), as in previous studies.[9] Severity of injury was classified as mild, moderate, or severe. Mild injury was defined as loss of consciousness or amnesia lasting <30 minutes. Moderate injury was defined as loss of consciousness from 30 minutes to 1 day or a skull fracture. Severe injury was defined as loss consciousness for >1 day, presence of subdural hematoma, or brain contusion.[10,11]

Seizures were categorized using the semiological classification based on the history and, when available, review of the seizures on video electroencephalography (VEEG; Table 1). Using this approach, instead of the International League Against Epilepsy classification, allowed us to study the seizures based exclusively on ictal clinical semiology.[12,13] When classifying the seizures, we chose to be as precise as possible depending on the availability of the seizure descriptions. For instance, hypomotor seizure category was used if the salient feature was immobility or a reduction in movements, as in infants and young children where consciousness cannot be assessed.[14] In this age group hypomotor seizures are likely a bland form of "complex partial" seizures with no or minimal automatisms.[15] Tonic-clonic seizure category was used if there was a report of "body stiffening followed by jerking" without any indication to a specific body part.

Methodology of subject identification

The database includes both outpatient and inpatient evaluations. Patients who were referred to the outpatient neurology clinic were either previously hospitalized and seen by a neurologist or were referred by a physician for an initial evaluation of concussion and/or TBI and related comorbidities. The on-call inpatient neurologist evaluated patients after a head injury if the hospitalist, neurosurgeon, physiatrist, or intensivist saw a specific indication, which typically included a prehospital spell or a seizure. If there was mention of a seizure or any other type of seizure-like event immediately after the TBI and before hospital admission, these patients would be seen, as requested by the treating physician. Therefore not all patients who had suffered a "head injury" were necessarily observed by a child neurologist. All patients with diagnosis of a head injury with or without associated comorbidities seen in the outpatient neurology clinics (which includes outlying satellite clinics) were reviewed and followed by the neurologist. We excluded patients who only had one seizure in the acute/subacute stages of recovery, but did not have additional unprovoked seizures before the cutoff time period. All pediatric neurologists in our division had approximately equal participation in evaluating the previously mentioned patients.

It is possible that there were patients who suffered TBI and may have had subclinical seizures in the pediatric intensive care unit. These patients may have had hypoxia and/or asymptomatic stroke during the recovery phase in the intensive care unit. However, unless they were referred to one of our child neurologists for evaluation, these patients were not monitored for development of PTE. Also, those children who had a concussion but were not referred to our pediatric neurology division were not included in our database, as we would not have had knowledge of such patients. Our study only looked at patients who were initially referred to our center for evaluation of clinical seizures (and subsequently electrographically confirmed) after a head injury. Those who were initially referred for a clinical seizure, but confirmed to have nonepileptic events were not included in this study. Lastly, neonates with

TABLE 1.
Semiological Seizure Classification*

Epileptic Seizure
Aura
Somatosensory aura†
Auditory aura†
Olfactory aura
Abdominal aura
Visual aura†
Gustatory aura
Autonomic aura†
Psychic aura
Autonomic seizure†
Dialeptic seizure‡
Typical dialeptic seizure‡
Motor seizure†
Simple motor seizure†
Myoclonic seizure†
Epileptic spasm†
Tonic-clonic seizure
Tonic seizure†
Clonic seizure†
Versive seizure†
Complex motor seizure‡
Hypermotor seizure‡
Automotor seizure‡
Gelastic seizure
Special seizure
Atonic seizure†
Hypomotor seizure‡
Negative myoclonic seizure†
Astatic seizure
Akinetic seizure†
Aphasic seizure‡
Paroxysmal event

* Semiological classification as published by Lüders et al.[12]
† Left/right/axial/generalized/bilateral asymmetric.
‡ Left hemisphere/right hemisphere.