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Seminars in
Pediatric
Neurology

Ethical Issues in Neuroprognostication after Severe Pediatric Brain Injury

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Neurologic outcome prediction, or neuroprognostication, after severe brain injury in children is a challenging task and has many ethical dimensions. Neurologists and intensivists are frequently asked by families to predict functional recovery after brain injury to help guide medical decision making despite limited outcome data. Using two clinical cases of children with severe brain injury from different mechanisms: hypoxic-ischemic injury secondary to cardiac arrest and traumatic brain injury, this article first addresses the importance of making a correct diagnosis in a child with a disorder of consciousness and then discusses some of the clinical challenges with deducing an accurate and timely outcome prediction. We further explore the ethical obligations of physicians when supporting parental decision making. We highlight the need to focus on how to elicit family values for a brain injured child, how to manage prognostic uncertainty, and how to effectively communicate with families in these challenging situations. We offer guidance for physicians when they have diverging views from families on aggressiveness of care or feel pressured to prognosticate with in a "window of opportunity" for limiting or withdrawing life sustaining therapies. We conclude with a discussion of the potential influence of emerging technologies, specifically advanced functional neuroimaging, on neurologic outcome prediction after severe brain injury

Semin Pediatr Neurol 22:187-195

Case 1—Hypoxic-Ischemic Brain Injury Secondary to Cardiac Arrest

A 4-month-old girl with no significant medical history had a cardiac arrest at home. Her parents found her unresponsive, face down in her playpen, and not breathing. She was last seen 20 minutes prior. Her father performed cardiopulmonary resuscitation for 5 minutes and emergency medical personnel continued cardiopulmonary resuscitation (CPR) for an additional 2-3 minutes. Spontaneous circulation returned after a dose of epinephrine. She was intubated and taken to the emergency department, where she was noted to have seizure-like movements in her right arm and eye twitching. She received 2 doses of lorazepam and was started on treatment with dopamine before transfer to the pediatric intensive care unit, where she was placed on a therapeutic hypothermia protocol. The computed tomography (CT) scan of her head was unremarkable. On initial examination she was unarousable, pupils were 1-2 mm and sluggishly reactive, vestibular-ocular reflexes were absent, and she had weak cough and gag reflexes. She had no spontaneous movements, and no reaction to noxious stimuli. Over the next several days she developed seizures that were difficult to control, and required treatment with a combination of levetiracetam, phenytoin, and midazolam and ketamine infusions. Her electroencephalogram (EEG) consisted of highly attenuated and slow activity, and it showed that she was not clearly reactive to stimulation. Clinically observed myoclonic jerks were associated with epileptiform sharps. She became hemodynamically stable on

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1071-9091/15/$-see front matter
http://dx.doi.org/10.1016/j.spen.2015.05.004

a dopamine infusion and was able to tolerate nasogastric feeds. A work up result for abusive head injury and metabolic diseases was unremarkable. The child's magnetic resonance imaging (MRI) of the brain was compatible with hypoxic-ischemic injury, showing multifocal areas of restricted diffusion in the posterior limbs of the internal capsules and the cerebral peduncles bilaterally. Her examination 1 week after the cardiac arrest showed her to be awake, with occasional spontaneous eye opening. Her pupils were equal and reactive, but she did not fix on or follow objects, and she did not blink to threat. Her vestibular-ocular reflexes were intact. Her tone was increased throughout. She had cortical thumbs as well as palmar and plantar grasp reflexes. She moved all extremities spontaneously. Occasionally, she exhibited lip smacking and bicycling movements of the lower extremities as well as extensor posture of the extremities.

Case 2—Traumatic Brain Injury

A 2-year-old boy with no significant medical history sustained a traumatic brain injury when a thick tree branch falling from several stories high struck him on the head. He had immediate loss of consciousness but did not have seizures or a cardiac arrest. He was intubated by emergency medical personnel and brought to the emergency department. On the initial examination he was unresponsive to voice and painful stimuli. His right pupil was reactive and his left pupil was dilated with minimal reactivity. Corneal reflexes were present, and he had a weak cough and gag. Head CT showed extensive calvarial and skull base fractures, left frontal subdural and subarachnoid hemorrhages, and small left lateral intraventricular hemorrhage. A CT angiogram of the brain was normal. Over the next several days his intracranial pressure was well controlled with sedation and hyperosmolar therapy; infrequent subclinical seizures were managed with levetiracetam. He required mechanical ventilation and had no major hemodynamic or respiratory complications. He was tolerating full nasogastric feeds. Brain MRI scan demonstrated multicompartmental intracranial hemorrhage with bilateral supratentorial and infratentorial small subdural collections, subarachnoid hemorrhage, intraventricular hemorrhage, and hemorrhagic contusions in the frontal and the temporal lobes. Widespread changes from diffuse axonal injury were apparent, including hemorrhage and restricted diffusion in the superolateral midbrain and cerebellar vermis, likely owing to Duret hemorrhages secondary to compression against the tentorial incisura at the time of injury. His examination approximately 10 days after injury demonstrated no spontaneous eye opening and no purposeful interaction with his environment. Cranial nerves were intact, except a persistent traumatic left dilated and nonreactive pupil. His extremities moved spontaneously and in response to painful stimuli, more vigorously on the left, with intermittent bilateral extensor posturing that had no EEG correlation.

Introduction

Neurologic outcome prediction after severe brain injury is one of the most difficult tasks neurologists and intensivists face, and this process is filled with a unique set of clinical and ethical challenges. With the advancement of technology and clinical practices in critical care and neurology aimed at preserving and restoring neurologic function, prognostication has evolved from estimating the probability of survival to the more complicated task of predicting functional recovery. Despite limited outcome data and inevitable prognostic uncertainty, neurologists are often compelled to make management recommendations and counsel parents about the potential for neurologic recovery when a child remains critically ill.

The cases presented in this article describe children with severe brain injury and resultant disorders of consciousness from 2 different mechanisms—hypoxic-ischemic injury secondary to cardiac arrest and traumatic brain injury. Although the etiologies of the brain insults are quite different, the clinical status of the children at the conclusion of these vignettes is comparable. However, the projected recovery may be quite divergent based solely on injury mechanism.

Using these cases as a backdrop, we address the importance of making the correct diagnosis in a patient with a disorder of consciousness after severe brain injury and discuss some of the challenges in deducing an accurate prognosis in these situations. We then explore some of the clinical and ethical challenges relating to the obligations of physicians to inform families about the predicted trajectory of neurologic recovery after severe brain insult and discuss how the certainty of a prognosis may appropriately or inappropriately influence clinical management decisions. Finally, we examine the potential effect of new technologies, specifically advanced functional neuroimaging, on our current and future prognostic abilities.

Making the Correct Diagnosis

The adage often quoted by medical ethicists—"good facts make good ethics"— aptly applies to children after severe brain injury and a disorder of consciousness. Consciousness comprises 2 clinical components—wakefulness and awareness of one's self and the environment.[1] A disordered state of consciousness, which can vary from acute and transient to irreversible and permanent, results when 1 or both of these components are compromised. Disordered states of consciousness are a spectrum of clinical syndromes that encompass a range of cognitive dysfunction, from confusional states such as delirium, to vegetative and minimally conscious states, coma, and locked-in syndrome.[2] Although the diagnosis of these syndromes can be significantly more challenging in infants and children, it is essential to properly identify both the etiology of brain injury and the correct disorder of consciousness syndrome before attempting to prognosticate recovery. The etiology of the impaired consciousness is easily identified in the vignettes presented