Abusive and Nonabusive Traumatic Brain Injury: Different Diseases, Not Just Different Intent

The current clinical approach to abusive head trauma (AHT) largely follows the diagnostic process for other types of traumatic brain injury (TBI), except when it comes to social management. Although it is important to protect the child from further injury, the argument goes, the brain injury is the brain injury, right? With these assumptions, it is logical to suspect abuse based largely on factors like an inconsistent or implausible history, to diagnose AHT with similar methods used for other TBI, and to manage AHT using the same medicines, procedures, and algorithms.

See related articles, p 170 and p 176

However, 2 studies in this volume of *The Journal* add to a growing field of evidence suggesting that these assumptions are misguided, and that outcomes for victims of AHT might be dramatically improved if we begin to understand and treat AHT as a different disease from other forms of TBI.

For decades, we have known that the outcomes of AHT, measured in mortality, resource use, and costs, are worse than other forms of TBI.[1-3] Although it is plausible that these differences could be explained by patient factors (eg, victims of AHT are younger), or the preferential identification of severe AHT, imaging and biochemical evidence suggests that there may be more at play.

Blackwell et al[4] add osteopontin to the large list of proteins that are detected in significantly different concentrations among children with AHT compared with other forms of TBI.[5-8] Nevertheless, the goal of identifying a biochemical test to improve AHT recognition remains elusive. Although osteopontin concentrations differed statistically between the groups, the overlap in individual values between abused and nonabused children in this and other studies is too much to support clinical use in this high-stakes diagnosis. At this time, there is no "troponin of brain injury," much less of AHT. Hope remains that biological screening might someday be achieved with the use of multiple markers and sophisticated analyses that go beyond a single positive/negative threshold.[9]

However, Blackwell's early, provocative study may yet have important implications. Serial marker testing, a key strength of the study, suggests that osteopontin—an inflammatory protein—may continue to increase in the days after AHT, but not other forms of TBI.[4] This finding comports with other evidence of AHT-specific patterns of inflammatory, ischemic, or excitotoxic secondary injury processes found in animal and imaging studies.[10-12] If further study confirms inflammatory processes particular to AHT, such information could improve both AHT recognition and our understanding of the processes of secondary injury; that is, when damage occurs after the initial trauma. Coupled with improved prevention, mitigation of secondary injuries is likely to be our most promising target for therapy. A traditional model of TBI treatment, based largely in preventing mass effect and herniation from intracranial hemorrhage, is unlikely to produce optimal outcomes, because many children with the most devastating forms of AHT present with only thin-film subdural hematomas and no mass effect.[11]

This recent attention to secondary injury to the brain parenchyma of children with AHT follows increased use of MRI, and especially nonsedated rapid MRI, in the evaluation of AHT.[13-16] Also in this issue, Noorbakhsh et al[17] demonstrate the cost effectiveness of rapid MRI as a case-finding method for relatively well-appearing children with AHT. As with Blackwell's study, this analysis depends on recognizing the specific aspects of AHT that distinguish it from other forms of TBI, specifically that AHT is difficult to identify and is likely to be a chronic or escalating disease if it goes unrecognized. For these reasons, traditional diagnostic approaches that are highly successful for other forms of TBI are not recommended for AHT.[18,19] In centers without access to rapid MRI, cost-effectiveness analysis supported screening with the Pittsburgh Infant Brain Injury Score and a relatively low threshold for imaging in children with high-risk complaints.[17,20] Their overall conclusion is clear: the need for secondary prevention implies a much lower testing threshold for AHT than other forms of TBI.

These advances in imaging and biochemical testing suggest that we are entering a new and more sophisticated era in AHT research, one in which we can begin to solve the vexing problem of stubbornly poor outcomes from AHT. The first step of this new era, surely, is to identify the factors most associated with secondary injury. Leading candidates include a delay in seeking care, repetitive trauma, social determinants of health, and particular mechanisms associated with AHT (ie, shaking, and/or prolonged hypoxia). At the same time, we need to

AHT Abusive head trauma
MRI Magnetic resonance imaging
TBI Traumatic brain injury

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identify specific biological processes (eg, hypoxia, inflammation, seizures, excitotoxicity) that predict poor outcomes for brain tissue and for patients.

From these insights, research into TBI therapy must recognize AHT as a unique disease from other forms of TBI. As difficult as it has been to test interventions, such as hypothermia, anticonvulsants, or decompressive craniectomy for pediatric TBI in general, we should push for similar studies or sub-group analyses specific to AHT.[21-23] Such studies would require innovation in processes of informed consent for young, abused children, who are often excluded from research for lack of an appropriate surrogate.

Finally, it is time to become serious about research focusing on social and policy interventions when there is a concern for abuse. From mandatory reporting to alternative responses, the ways that different jurisdictions address abuse vary widely, and are determined more by the most recent high-profile disaster than by any data about outcomes for families and children.[24,25] Leveraging the advances of Noorbakhsh, Blackwell, and others will require at least a bare minimum of data regarding the outcomes of children reported to child protective services, such as mortality, long-term family separation, health, educational achievement, and patient and family satisfaction.

For the youngest children, AHT is the most important source of morbidity and mortality from TBI. It is time to reject the assumption that if we could only improve awareness and recognition of AHT, we could end the outcome gaps for abused children relative to those with other TBI. Rather, from diagnosis and treatment, to pathophysiology and policy, we should begin to treat AHT as the separate disease that it surely is, and devote the resources and attention commensurate with its impact. ■

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