Cerebral infarct in head injury: relationship to child abuse

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Abstract

Background: To determine whether CT-detected cerebral infarct in young children is associated more often with abuse or unintentional head injury.

Methods: Retrospective case-control study of injured children under age 6 who had abnormal initial head CT scans and who were admitted to the only Level I pediatric trauma center in Washington State for closed head injury (CHI) from January 1, 1992 to December 31, 1998.

Results: Fifteen children developed cerebral infarct after CHI during the 7 year period. These cases were compared to 53 controls (those who did not develop infarct). After adjusting for the presence of SDH and for severity of injury, patients with infarcts were six times more likely to have been abused than patients without infarcts (OR 6.1; 95% CI, 1.02–36.0).

Conclusions: Cerebral infarct after CHI appears to result more frequently from abuse than unintentional injury in young children.

Keywords: Infarct; Unintentional injury; CT scan; Child abuse
Introduction

One rare but recognized complication of closed head injury (CHI) in children is cerebral infarct (Duval, Van Coster, & Verstraeten, 1998; Gilles & Nerlson, 1998; Mirvis, Wolf, Numaguchi, Corradino, & Joslyn, 1990). It is often not apparent at the time of presentation to the hospital, but develops one to several days after the injury occurred (Auer, Kreeck, & Butt, 1994; Duhaime, Christian, Rorke, & Zimmerman, 1998). Infarcts prolong recovery, increase length of stay in hospital and rehabilitation facilities, and are often associated with at least temporary if not permanent motor, sensory and speech deficits (Dharker, Mittal, & Bhargava, 1993; Garg & DeMeyer, 1995). A 1999 study of traumatic brain injury in children under age 6 found that patients with cerebrovascular infarct had significantly decreased mental and motor function after the injury (Ewings-Cobbs, Kramer, & Prasad, 1999).

An association between head injury due to abuse and cerebral infarct has been described in several case studies (Bird, McMahon, Gilles, Senac, & Apthorpe, 1987; Cohen, Kaufman, Myers, & Towbin, 1985; Suh, Davis, Hopkins, Fajman, & Mapstone, 2001). However, these studies were uncontrolled and did not attempt to discern whether head injury from unintentional causes have the same risk of cerebral infarct. We reasoned that if the mechanisms of injury associated with abuse is a factor in the development of posttraumatic cerebral infarct, then child abuse would be found more frequently among those patients who developed infarct than among those who did not. The finding that cerebral infarct develops more often after abuse than unintentional injury would have important medical implications, both in the management of known abuse and in the understanding of the potential mechanisms of ischemic injury.

Methods

This was a case-control study of posttraumatic cerebral infarct in young children. The study was approved by the Human Subjects Review Committee of the University of Washington. We studied children under age 6 who had an abnormal initial Computed Tomography (CT) scan and were admitted to the only Level I pediatric trauma center in Washington state for a closed head injury between January 1, 1992 and December 31, 1998. We considered skull fracture, intracranial hemorrhage, cerebral contusion or edema, and shear hemorrhages as abnormal findings on CT.

Cases were all children who were found on CT scan to have cerebral infarct at some time during their acute hospital stay. The cases were identified by searching a database of all head CT reports for the text words “infarct” or “ischemia.” The CT scans of all patients found by CT report to have cerebral infarct were independently reviewed by a trauma radiologist (FAM), who was blinded to any identifying information about the patients, including cause of injury.

The diagnosis of infarct was based on morphology of the CT abnormalities and the temporal course of the lesions on CT scan (Gean, 1994). We defined infarct as any region in the brain with lower density than normal brain and either diffuse cerebral ischemia characterized by decreased or loss of gray-white interface OR a wedge-shaped or rounded region occupying recognized vascular territory (Wardlaw, Lewis, Dennis, Counsell, & McDowall, 1998).
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