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Comparison of Different Kinds of Traumatic Head Injury in Children on Computed Tomography

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Abstract: Mixed-density convexity subdural hematoma and interhemispheric subdural hematoma suggest nonaccidental head injury. The purpose of this retrospective observational study is to investigate subdural hematoma on noncontrast computed tomography in infants with nonaccidental head injury and to compare these findings in infants with accidental head trauma for whom the date of injury was known. Two blinded, independent observers retrospectively reviewed computed tomography scans with subdural hematoma performed on the day of presentation on 9 infant victims of nonaccidental head injury (mean age: 6.8 months; range: 1-25 months) and on 38 infants (mean age: 4.8 months; range: newborn to 34 months) with accidental head trauma (birth-related: 19; short fall: 17; motor vehicle accident: 2). Homogeneous hyperdense subdural hematoma was significantly more common in children with accidental head trauma (28 of 38 [74%]; nonaccidental head trauma: 3 of 9 [33%]), whereas mixed-density subdural hematoma was significantly more common in cases of nonaccidental head injury (6 of 9 [67%]; accidental head trauma: 7 of 38 [18%]). Twenty-two (79%) subdural hematomas were homogeneously hyperdense on noncontrast computed tomography performed within 2 days of accidental head trauma, one (4%) was homogeneous and isodense compared to brain tissue, one (4%) was homogeneous and hypodense and 4 (14%) were mixed-density. There was no statistically significant difference in the proportion of interhemispheric subdural hematoma, epidural hematoma, calvarial fracture, brain contusion, or subarachnoid hemorrhage. Homogeneous hyperdense subdural hematoma is more frequent in cases of accidental head trauma; mixed-density subdural hematoma is more frequent in cases of nonaccidental head injury but may be observed within 48 h of accidental head trauma. Interhemispheric subdural hematoma is not specific for inflicted head injury.

Key words: Abuse, subdural hematoma, brain imaging, accidents, retinal hemorrhage

INTRODUCTION

Inflicted trauma is the cause of head injury in 7-10% sof children under the age of 2 years but accounts for a disproportionate share (≤56%) of serious brain injury (Bruce and Zimmerman, 1989). Vigorous shaking, with or without directly impacting the skull, is the postulated mechanism of injury to the brain or calvarium (Bruce and Zimmerman, 1989; Duhaime *et al*., 1998, 1987; Hadley *et al*., 1989). Forceful shaking of an infant creates an angular acceleration-deceleration force that can stretch and tear cortical veins that course to the dural venous sinuses through the subarachnoid space and results in hemorrhage into either the subdural or subarachnoid spaces (Bruce and Zimmerman, 1989; Duhaime *et al*., 1987; Billmire and Myers, 1985). Axons coursing from gray to white matter or to areas of denser packing may also be stretched and torn, resulting in diffuse axonal or shear injury. It is hypothesized that with rotational acceleration of the ocular globe, traction on the retina by the firmly attached vitreous can lead to retinal hemorrhage, folds and separation (Chen *et al*., 1999). As a result, a constellation of findings has been observed from injury by inflicted head trauma, including interhemispheric subdural hematoma (SDH), shear injuries, diffuse axonal injury, contusional white matter tears and retinal hemorrhage (Hymel *et al*., 1997; Zimmerman and Bilaniuk, 1994; Zimmerman *et al*., 1978, 1979). In contrast, the most common mechanism of accidental head injury is a linear or translational impaction force that may cause a linear skull fracture, epidural hematoma, localized SDH, or cortical contusion. Falls from heights produce higher impact forces that can produce depressed or comminuted skull fracture, subarachnoid hemorrhage, or cortical contusion (Chan *et al*., 1990). Depressed skull fracture can also result from short falls when the head impacts a small surface area (Williams, 1991).

Noncontrast Computed Tomography (CT) is often performed as the initial imaging test for the evaluation of the infant with suspected head injury from trauma. It has been suggested that the finding of a heterogeneous or mixed-density SDH on CT implies multiple episodes of traumatic injury and, therefore, a pattern of repetitive head injury (Brown and Minns, 1993; Chabrol *et al*., 1999; Hart *et al*., 1996). Similarly, others have stated that the finding of an interhemispheric SDH is pathognomonic of the shaking or shaking-impact mechanism of head injury (Duhaime *et al*., 1998; Zimmerman *et al*., 1978; Barnes and Robson, 2000).

The purpose of this study is to investigate the incidence, location and density-pattern of SDHs and other brain injuries on noncontrast CT scan in infants with accidental head trauma and to compare these findings in infants with nonaccidental head injury.

MATERIALS AND METHODS

This investigation was performed at a tertiary care teaching hospital complex with a level-1 trauma center and a separate labor and delivery hospital. The institutional review board of both institutions approved the conduct of this investigation.

Subjects: Through, a computer-assisted search of the radiology information system, we identified 162 noncontrast CT examinations performed in 106 children ≤3 years of age in which the term "subdural hematoma" or "subdural hemorrhage" was contained in the body or conclusion of the report. After assigning a unique, random identification number, reports were uploaded into a database. After review of these primary interpretations, subjects were excluded because the SDH was secondary to neoplasm, aneurysm, or congenital malformation (n = 11). Subsequently, additional subjects were excluded because medical charts or CT scans could not be located for review (n = 40), the mechanism of injury had not been witnessed (n = 4), or the CT scan had not been performed within 2 weeks of witnessed injury (n = 4). The remaining 73 noncontrast CT examinations, performed on 47 children, are the subject of this investigation.

CT scans: After masking all of the identifying information on hard-copy images, CT scans were reviewed, first independently and then jointly, by a neuroradiologist and a pediatric neurologist, each with 15 years of clinical experience, who were blinded to the clinical history.

SDH: Observers determined the location and the attenuation of each SDH. The location of the SDH was first classified as convexity or falcotentorial. If located along the cerebral convexity, it was further described by location as frontal, temporal, parietal, or occipital. If falcotentorial, the location was further classified as interhemispheric if the hematoma was adjacent to the interhemispheric falx or tentorial if the SDH was next to the cerebellar tentorium. The attenuation of each SDH was classified as homogenous or heterogeneous in reference to the number of different areas of attenuation within the SDH on noncontrast CT. If the attenuation of the SDH was uniform, it was classified as homogeneous. If the hematoma had components of different attenuation, that is, dependent sediment different in attenuation from a nondependent supernatant, it was classified as heterogeneous. If heterogeneous, the observers then further classified the different components of the attenuation using cortical brain and Cerebrospinal Fluid (CSF) as reference tissues. For example, a heterogeneous lesion could have one component that was isodense compared with CSF and another that was hyperdense relative to cortical brain tissue.

Other traumatic brain injuries on noncontrast CT: Other brain injuries were also documented on each noncontrast CT scan. Epidural hematoma was diagnosed if there was a biconvex, hyperdense hematoma that displaced the gray-white matter brain interface away from the calvarium. Subarachnoid hemorrhage was diagnosed if there was high-attenuation blood in the cisternal or convexity subarachnoid space. Skull fracture was diagnosed as when a linear or branching radiolucent area with sharp, well-defined margins was identified in the calvarial bone. Brain contusion was diagnosed when there was a discrete focus of cortical hyperdensity, with or without adjacent white matter edema.

Classification of head injury: The classification of head injury relied on the available hospital medical charts and the forensic evaluation by the hospital child protection team. Cases of Nonaccidental Head Injury (NAHI) were determined by the child protection team after review of the clinical history and past medical charts, performance of a physical examination. A pediatrician with special expertise in child maltreatment leads this child protection team. Factors considered by the child protection team include the caretaker's confession of inflicting injury, inconsistent or inadequate history from the caretaker, delay in seeking health care and other injuries consistent with inflicted trauma presenting contemporaneous with the head injury. These other injuries include posterior rib fracture, classic metaphyseal lesion (i.e., microfracture across the metaphysis that is oriented parallel to the physis), unexplained fractures of different age, spiral

fracture in nonambulatory child, spinal fracture and bowel or pancreatic injury. A decision that the observed head injury was nonaccidental required consensus by the multidisciplinary child protection team. Accidental Head Trauma (AHT) as a result of a single traumatic event was determined from a corroborated clinical history of birth-related trauma, fall from a height, or accident involving a motor vehicle. Confirmation of the clinical history in these cases was obtained from review of the medical chart in cases of birth-related trauma and the traffic accident report in cases of motor vehicle accident. Neonates who had presented with symptoms within 3-5 days of birth were classified as birth trauma. Furthermore, children classified as having suffered AHT did not meet the aforementioned diagnostic criteria for NAHI.

Dating of SDH: To study the attenuation of blood in the SDH on noncontrast CT, both at presentation and over time, the time of AHT and the date of the CT scan were used to determine the "age" of the SDH. A definite date and time of head injury was recorded for subjects who had sustained witnessed or corroborated injury from birth, fall from a height, or motor vehicle crash. For the birth-related injury cohort, the time of delivery was recorded.

Statistical methods: Isher's exact test was used to determine the statistical significance of the data. Statistical significance was defined using a 95% confidence interval with a $p \leq 0.05$.

RESULTS

The group of children in this study consisted of 30 boys and 17 girls with a mean age of 5.2 months (range: 0-34 months). The type of head injury was classified as AHT in 38 children and as NAHI in 9 children. Based on the clinical history, the children who sustained AHT were further divided into those whose traumatic head injury was associated with birth (n = 19), fall from a height or other blunt traumatic head injury (n = 17), or motor vehicle crash (n = 2); there were 26 boys and 12 girls in this group with a mean age of 4.8 months (range: 0-34 months). The 9 children who had sustained NAHI consisted of 4 boys and 5 girls with mean age of 6.8 months (range: 1-25 months).

The average number of noncontrast CT scans performed per child was 1.5±0.7 (mean±SD; range: 1-4); for the children with birth-related head injury, 1.2±0.4 (range: 1-2); for children with accidental, nonbirth-related injury, 1.8±0.8 (range: 1-4); and for children with abusive head trauma, 1.7±0.7 (range: 1-3). The average time between AHT and initial noncontrast CT scan was 2.0±2.8 days (range: 0-13 days). The mean time between witnessed fall or motor vehicle crash and initial NCCT scan was 1.5±2.5 days (range: 0-10 days). The average time between birth-related trauma and initial NCCT scan was 2.6±3.1 days (range: 0-13 days).

Noncontrast head CT scans in 38 cases of AHT: Of the 13 noncontrast CT scans that were performed within 1 day of AHT, 9 (69%) demonstrated homogeneous, hyperdense SDH, 1 (8%) was homogeneous and isodense compared with brain tissue and 3 (24%) were heterogeneous in attenuation. Of these heterogeneous SDHs, 1 had a sediment that was hyperdense and a supernatant that was hypodense compared with brain, another also had a sediment that was hyperdense to brain but a supernatant that was isodense compared with CSF and the third heterogeneous SDH had a sediment that was isodense to brain and a supernatant that was isodense to CSF.

Of the 15 noncontrast CT scans that were performed >1 day but <2 days after witnessed AHT, 13 (87%) demonstrated homogeneous, hyperdense SDH and 1 (7%) was homogeneous but hypodense compared with brain tissue. There was only 1 (7%) heterogeneous SDH; it had a sediment that was isodense compared with brain and a supernatant that was isodense to CSF. Ten of these 15 cases had been secondary to birth-related trauma.

Comparison of traumatic brain injuries: On noncontrast CT scans performed at presentation, homogeneous hyperdense SDH was identified on 28 (74%) scans in 38 cases of AHT and in 3 (33%) scans in 9 cases of NAHI; this difference was statistically significant ($p = 0.045$). Heterogeneous SDH was demonstrated on NCCT scan in 6 (67%) cases of NAHI and in 7 (18%) cases of AHT; this difference was also statistically significant ($p = 0.008$). There were significantly more heterogeneous SDHs containing hyperdense blood in cases of NAHI; this was demonstrated in 5 (56%) cases of NAHI and in 4 (11%) cases of AHT ($p = .007$). Interhemispheric SDH was found on 3 (33%) NCCT scans in 9 cases of NAHI and in 9 (24%) scans in 38 cases of AHT; this difference was not statistically significant ($p = 0.67$). There was also no statistically significant difference in the proportion of epidural hematoma (NAHI, 3 of 9 [33%]; AHT, 4 of 38 [11%]; $p = 0.12$), calvarial fracture (NAHI, 1 of 9 [11%]; AHT, 16 of 38 [42%]; $p = 0.18$), brain contusion (NAHI, 1 of 9 [11%]; AHT, 7 of 38 [18%]; $p = 0.68$), or subarachnoid hemorrhage (NAHI, 0 of 9; AHT, 4 of 38 [11%]; $p = 0.57$).

Ten noncontrast CT scans were performed >2 days after witnessed AHT. Six (60%) demonstrated homogeneous SDHs, that were hyperdense compared with brain and 1 of these cases was performed on the

tenth day after birth-related head trauma. One (10%) SDH was homogeneous and isodense compared with CSF and this scan was performed 5 days after a motor vehicle crash. Three (30%) SDHs were heterogeneous in attenuation and were demonstrated on CT scans performed within 3 days, 7 days and 11 days, respectively, after AHT.

DISCUSSION

One of the most common manifestations of traumatic brain injury in abused children is SDH (Duhaime *et al*., 1987; Ewing-Cobbs *et al*., 2000; Reece and Sege, 2000; Vinchon *et al*., 2005). In a series of 287 children between the ages of 1 week and 6.5 years, SDH was found in 46% of children with NAHI but in 10% of children with accidental head injury (Reece and Sege, 2000). In another study of 150 cases of head-injured children under the age of 2 years, Vinchon *et al*. (2005) found SDH in 81% of 57 victims of child abuse compared with 28% of children with AHT; child abuse accounted for 64% of all SDHs. With rapid to-and-fro motion, the brain and bridging superficial cortical veins move at a different rate than the calvarium and attached dural venous sinuses. As a result, the rupture of cortical veins may create a hematoma in the subdural space (Chen *et al*., 1999; Zimmerman *et al*., 1979). Ewing-Cobbs *et al*. (2000) report that interhemispheric, convexity and infratentorial SDH were significantly more prevalent after inflicted brain injury than accidental traumatic brain injury. Some authorities posit that the interhemispheric SDH is particularly characteristic of the shaking or shaking-impact mechanism of injury (Duhaime *et al*., 1998; Zimmerman *et al*., 1978; Barnes and Robson, 2000). In our study, interhemispheric SDH was found in one third of children with NAHI, but also in nearly one quarter of children with AHT. Therefore, we cannot conclude that interhemispheric SDH is pathognomonic of NAHI. Furthermore, we found no statistically significant difference in the prevalence of calvarial fracture, epidural hematoma, subarachnoid hemorrhage, or brain contusion between children with AHT and NAHI.

When SDHs of mixed attenuation are discovered in victims of inflicted head injury, the most commonly cited explanation has been repetitive episodes of head injury over time (Hymel *et al*., 1997; Zimmerman *et al*., 1978, 1979). In this study, heterogeneous or mixed-density SDH was significantly more common in children with abusive head injury. The mixed-density SDH on noncontrast CT reflects the combination of high-attenuation blood from acute hemorrhage or clot retraction and lower density fluid from unclotted blood, serum, or CSF (Bergstrom *et al*., 1977; Lee *et al*., 1997; Kao, 1983). Foci of high attenuation in SDH on noncontrast CT are related to the relatively higher concentration of hemoglobin in blood during hemoconcentration and clot retraction (New and Aronow, 1976; Norman *et al*., 1977). The classic teaching is that an acute SDH is hyperdense relative to the attenuation of normal brain tissue and, over the ensuing 4 days to 3 weeks, becomes isodense compared with normal brain (Bergstrom *et al*., 1977; Moller and Ericson, 1979; Scotti *et al*., 1977). Hypodense SDHs are usually >3 weeks of age and appear grossly like crank-case oil (Scotti *et al*., 1977). In adults, a heterogeneous or mixed-density SDH may result from recurrent hemorrhage into a chronic SDH in the absence of known traumatic head injury. It is postulated that the rupture of fragile neovessels in the organizing subdural membrane may explain this "spontaneous" mixed-density hematoma (Friede and Schachenmayr, 1978; Schachenmayr and Friede, 1978). However unlike adults, spontaneous rebleeding into a chronic SDH is much less common in infants (Block, 1999; Case *et al*., 2001; Krous and Byard, 1999). Therefore, many authors believe that a heterogeneous, mixed-density SDH in an infant implies an acute-superimposed-on-chronic head injury, a pattern that suggests repetitive head injury (Hymel *et al*., 1997; Zimmerman *et al*., 1979; Sinal and Ball, 1987).

In a prospective case series of 66 children with SDH, Feldman *et al* . (2001) reported that only abused children and children whose injuries were of indeterminate cause had mixed acute and chronic or chronic SDH. However from our study, we would like to urge caution about the diagnostic implications of a mixed-density SDH. Although it was significantly more common in cases of NAHI, we report heterogeneous SDH in 4 of 28 noncontrast CT scans performed within 2 days of well-documented AHT. In 2 separate studies, Vinchon *et al*. (2002, 2004) report that infantile SDH after a witnessed traffic accident may have mixed density and attribute this to early sedimentation of clotted blood. Other investigators have also reported mixed-density extra-axial hematoma with active bleeding and no evidence of previous hemorrhage and in patients with a coagulopathy (Greenberg *et al*., 1985; Sargent *et al*., 1996). There are several possible explanations for a heterogeneous SDH after a single episode of traumatic head injury. A traumatic tear in the arachnoid membrane or granulations may create a communication between the subarachnoid and subdural spaces. Zouros *et al*. (2004) studied CSF flow in 5 infants who had undergone external drainage after evacuation of a mixed- or low-density SDH. Indium radiotracer was detected in a cranial subdural catheter within 24 h after the radiotracer was instilled in the lumbar subarachnoid space and indicated free

communication between the cranial subarachnoid and subdural spaces. Through such a communication, created by a traumatic tear of the arachnoid membrane or granulations, CSF in the subarachnoid space could mix freely with radiodense blood from the acute SDH to create a mixed-density hematohygroma. Alternatively, when hypodense CSF in the subarachnoid space (e.g., benign extraaxial fluid collections of infancy) or in the subdural space (e.g., chronic subdural hygroma) predates head trauma, a superimposed acute SDH may create a heterogeneous attenuation pattern on noncontrast CT. Rapid clotting and clearance of blood or severe anemia might also produce an SDH of relatively low density (Vinchon *et al*., 2002, 2004; Kaufman *et al*., 1980; Smith *et al*., 1981). Acute, clotted blood is hyperdense compared with the gray and white matter of the brain because of the globin moiety of hemoglobin and may actually increase in radiodensity over the first 3 days because of clot retraction (Bergstrom *et al*., 1977; Kaufman *et al*., 1980). Early studies on the appearance of subacute hemorrhage showed that its radiodensity on noncontrast CT decreases ~1.5 Hounsfield units (H) per day because of clot resorption and liquefaction (Scotti *et al*., 1977). If one assumes that the initial density of acute clotted blood could be as high as 120H and the density of gray matter is ~35H, then in theory it might take as long as 57 days for the blood to become isodense with cerebral gray matter. However, isodense SDHs have been detected between 7 and 22 days after traumatic head injury (Moller and Ericson, 1979; Scotti *et al*., 1977). In this study, 2 isodense and 1 hypodense SDHs were found within 5 days of AHT. Dias *et al*. (1998) reported the development of a hypodense SDH within 17 h of inflicted head injury. Conversely, a homogeneously hyperdense SDH, which was more commonly observed in cases of accidental traumatic head injury, was found as late as 9 days after birth-related head trauma in this study. Others have reported high-density SDH on CT scans performed up to 11 days after accidental traumatic head trauma (Vinchon *et al*., 2002). Thus, it is important to emphasize the uncertainty in making precise inferences about the date or cause of head injury from a single noncontrast CT scan performed at the time of presentation.

There are several limitations to this study. First, this was a retrospective study performed before the implementation of a digital picture archiving and an electronic medical chart system. As a result, we were not able to obtain the original CT scan or pertinent medical chart in all of the cases and this compelled the exclusion of many eligible subjects from our study (participation bias). Second, the number of infants with NAHI in this study is relatively small, particularly compared with the number of subjects with AHT. In this regard, it is important to mention that this was not a study of all infants suspected of child abuse but only of those with suspected head injury from inflicted injury. Because of this selection bias, it is probable that the incidence of brain injuries that we have reported is higher than if all suspected cases of abuse had been imaged.

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