

# Delayed Sudden Death in an Infant Following an Accidental Fall

## *A Case Report With Review of the Literature*

*Scott Denton, MD, and Darinka Mileusnic, MD, PhD*

**Abstract:** Several controversies exist regarding ultimately lethal head injuries in small children. Death from short falls, timing of head injury, lucid intervals, presence of diffuse axonal injury (DAI), and subdural hematoma (SDH) as marker of DAI are the most recent controversial topics of debate in this evolving field of study. In this area of debate, we present a case of delayed death from a witnessed fall backwards off a bed in a 9-month-old black male child who struck his head on a concrete floor and was independently witnessed as "healthy" postfall for 72 hours until he was discovered dead in bed. Grandmother, babysitter, and mother all independently corroborated under police investigation that the child "acted and behaved normally" after the fall until death. Autopsy showed a linear non-displaced parietal skull fracture, diastasis of adjacent occipital suture, subgaleal hemorrhage with evidence of aging, small posterior clotting SDH, marked cerebral edema, and a small tear of the midsuperior body of the corpus callosum consistent with focal axonal injury (FAI). No DAI was seen, and there were no retinal hemorrhages. All other causes of death were excluded upon thorough police and medical examiner investigation. Although this seems to be a rare phenomenon, a delayed, seemingly symptom-free interval can occur between a clinically apparent mild head injury and accidental death in a young child.

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### CASE REPORT

A 9-month-old black male child weighing 22 pounds (10 kg) and measuring 28 inches (71 cm), 80<sup>th</sup> percentile and 50<sup>th</sup> percentile for age, respectively, with a history of asthma

treated with nebulizer, was witnessed by his grandmother to fall backwards off the edge of a queen-sized bed, 30 inches off the floor. The child was sitting on the edge of the bed as the grandmother dressed her 2-year-old daughter. The child fell backwards and rotated from the sitting position, striking the midback of his head on a vinyl-covered concrete floor at 8:00 AM. He immediately began crying, and the grandmother placed ice on a knot on the back of his head. He stopped crying and was consolable within a few minutes. The child was taken to the babysitter's residence, where the babysitter was told of the fall and to watch for any behavioral changes. The mother was at work the morning when the fall occurred. When the mother picked the child up at the babysitter's in the afternoon, he appeared well. The babysitter reported no problems and that he acted, ate, and behaved as usual. For the next 2 days, the grandmother, mother, and babysitter did not notice any abnormalities in either behavior or appearance of the child.

Approximately 72 hours after the fall off the bed, the child was found at the foot of the mother's bed, where he usually slept, prone, cold, and unresponsive. Paramedics were called, and in spite of resuscitative efforts, he was pronounced dead upon arrival at the hospital. He was last seen alive 8 hours prior when he was fed by his mother and given his usual albuterol breathing treatment. No abnormalities on the child were seen in the emergency department. There was no evidence of overlying or asphyxia.

Medical and family history revealed that the child was born full-term weighing 7 pounds 4 ounces. He was diagnosed with asthma after complaints of wheezing episodes and was prescribed an albuterol nebulizer twice daily. The child's father, who does not reside in the home, has asthma. The mother and grandmother reside together in a public housing development. Department of Child and Family Services records revealed no prior incidents concerning the deceased, but the mother had 1 report of being a victim of prior abuse. The grandmother has a remote history of cocaine abuse. Her 2-year-old daughter is well and lives with her.

Autopsy revealed a well-developed and well-nourished black male child appearing the stated age and without exter-

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From the Cook County Office of the Medical Examiner, and Department of Pathology, Rush College of Medicine, Chicago, Illinois (J.S.D.), and Knox County Office of the Medical Examiner, and Department of Pathology, Graduate School of Medicine, University of Tennessee, Knoxville, Tennessee (D.M.).

Reprints: Darinka Mileusnic, MD, PhD, Regional Forensic Center, University of Tennessee Memorial Hospital, 1924 Alcoa Highway, Knoxville, TN 37920. E-mail: dmileusn@mc.utmc.edu

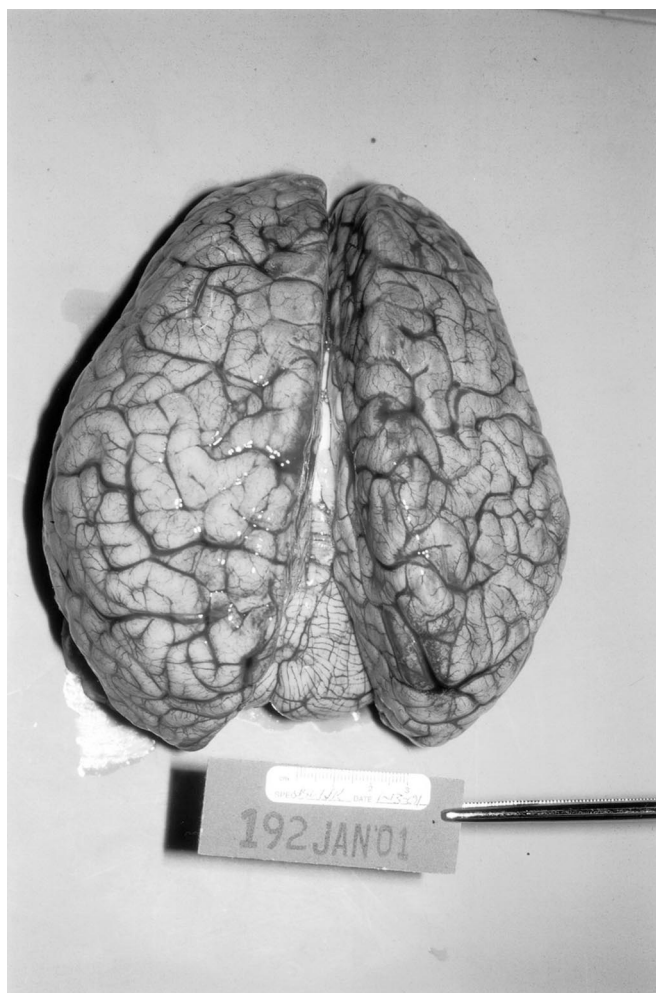
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nal abnormality. There was no swelling or contusion of the back of the head. Complete postmortem radiographs revealed a linear, nondisplaced, posterior right parietal skull fracture. Internal examination confirmed the skull fracture, as well as a patch or right posterior subgaleal hemorrhage that was centrally red with yellow margins. The underlying right posterior linear skull fracture was 3.0 inches (9.0 cm) long and extended to the right parieto-occipital suture, causing mild diastasis of the suture, 2.5 inches (7.5 cm) long (Fig. 1). There was a thin adherent clotted SDH underlying the fracture,  $2.0 \times 2.0 \times 0.1$  cm. The brain weighed 1035 g (expected average weight for age, 750 g) and showed severe edema with flattening of the gyri, loss of sulci, and notching of both unci and cerebellar tonsils (Fig. 2). After formalin fixation, serial sectioning of the brain revealed diffusely dusky white matter and a focal tear of the midsuperior corpus



**FIGURE 1.** View of the linear skull fracture involving the posterior right parietal bone, after removal of the overlying subgaleal hemorrhage.



**FIGURE 2.** Severely edematous brain demonstrating flattening of the gyri and narrowing of the sulci.

callosum, 1 mm, with surrounding hemorrhage, 2 mm. There were no other gross neuropathologic findings. The remaining internal organs were unremarkable, without other new or old fractures, petechiae, or gross asthma changes. Comprehensive toxicologic screening using gas chromatography and mass spectrometry was negative. Microscopically, the corpus callosum tear showed hemorrhage with intact red blood cells, FAI, and microglial activation without inflammation (Fig. 3). Extensive sections of the brain showed only edema without evidence of DAI. Sections of the subgaleal hemorrhage showed hemorrhage of coalescing red blood cells with neutrophilic inflammation. Decalcified sections of the parietal fracture showed an acute fracture with early periosteal reaction at the fracture margin. Lung sections showed mild focal peribronchial lymphocytic aggregates consistent with bronchitis without asthma changes. There were neither eosinophils nor mucus plugs. Sections of remaining organs were



**FIGURE 3.** Coronal section of the corpus callosum showing wedge-shaped laceration (right upper corner) surrounded by a rim of hemorrhage, FAI and activated microglia (100 $\times$ , hematoxylin-eosin).

without pathologic changes. The eyes were examined by an ophthalmic pathologist consultant and were normal. A forensic radiologist consultant also reviewed postmortem radiographs and reported no additional findings.

### Follow-up Investigation

Prior to the autopsy, Chicago police detectives were notified of the skull fracture and attended the examination. After autopsy, police remanded the grandmother and mother to the police station, where they were interviewed separately about the injuries. Upon extensive questioning about any possibility of inflicted trauma and abuse that the baby could have sustained, they both spontaneously gave the similar story of the fall 3 days prior. The babysitter was questioned and confirmed the accounts and timing of the reported events. Police detectives and evidence technicians accompanied the mother and grandmother back to their residence and verified the scene and reenactment of the fall. A week later, the prosecutor pathologist (JSD) and a specialist child death scene investigator of the Medical Examiner's Office went to the residence and again inspected the residence, interviewed the grandmother and mother, and reenacted the fall. As with the police detectives, all felt the grandmother and mother to be truthful and grieving appropriately for the circumstances. After consideration of the autopsy, toxicologic, histologic, consultative, and investigative findings, the death was certified as craniocerebral injuries due to a fall from the bed backwards onto a concrete floor. The manner was determined accidental.

### DISCUSSION

Certain issues in pediatric head trauma, such as lethality of short falls, timing of head injury, and presence of DAI

in the majority of lethal events, are still controversial. One widely held belief is that short falls are almost never fatal. Second, if a child is going to die following head trauma, either accidental or abusive, he or she is severely impaired and most likely immediately unconscious, without a lucid interval. Finally, in severe injuries where children are immediately comatose and die shortly after the incident from either shaking and/or direct impact, it is believed that DAI is the mechanism. Certain reviews have gone so far to identify subdural hemorrhage, frequently present in certain forms of early childhood abusive head trauma, as a "marker" of undetectable DAI.<sup>1</sup> If this were true, then reports describing radiologically present old and/or new subdural hemorrhages, with or without focal shear hemorrhages in the white matter, in living children would be a rarity rather than a common place.<sup>2</sup>

In this present case, we discuss the death of a 9-month-old child who died 3 days after a witnessed backward fall from a bed on a concrete floor. Main pathologic findings consisted of a linear nondisplaced skull fracture, minimal clotting subdural hemorrhage, severe brain swelling with tonsillary herniation, and a small tear in the body of the corpus callosum, which appeared histologically as FAI. Analysis of the fall revealed a rotational component of the body and head movement, which could account for the described injuries. The location and appearance of the primary injury was consistent with flipping backward and striking the back of the head. There was no diffuse axonal damage or retinal hemorrhage. Thorough workup, including scene investigation and independent police questioning of all individuals involved in the care of the infant, prior to, during, and after the accident, were unanimous. There were no inconsistencies, and the stories have never varied from the beginning to the conclusion of the investigation. There were no other instances of trauma to the head observed by the caretakers. Based on several independent accounts, the infant's behavior following the head trauma up to his sudden death was ordinary and did not require medical attention, qualifying as a lucid or symptom-free interval.

Deciding whether head injury in a very young child is accidental or nonaccidental has always been problematic for clinicians and forensic pathologists alike.<sup>3,4</sup> We realize that a number of child abuse experts would have a problem with the accidental determination of the manner of death in the present case. We do not argue the widely noted observation that simple falls from low heights rarely result in significant primary brain injury.<sup>5</sup> However, every fall is different, as well as the individual reaction to the primary insult. Some experts in head trauma consider the term *minor head injury* an oxymoron.<sup>6</sup> We believe that a series of secondary injuries, known to occur after a primary insult, resulted in the extreme swelling of the brain and death of the child. What is widely understated and sometimes forgotten about is secondary brain

injury, which occasionally may be the principal force determining the outcome after a seemingly trivial head injury.<sup>7-12</sup> Another frequently forgotten factor is the influence of age and sex on the presentation and the outcome of head injury. The grouping together of different pathologies such as subdural hemorrhages, cerebral contusions, FAI, and DAI, as well as lumping together of infants, toddlers, and preschool children, needs to be addressed. It has been shown that infants and young toddlers lose consciousness less frequently, and a smaller proportion of their head injuries lead to immediate coma in comparison to other children with the same grades of traumatic energy.<sup>13</sup> Pohl et al<sup>9</sup> demonstrated that evolution of posttraumatic brain damage after head trauma in developing rodents is a highly dynamic process exhibiting age-dependent excitotoxic and distant apoptotic cell death.

Reviewing the literature on childhood head trauma, one can clearly see that a gradual sideways drift or evolution of findings and conclusions of the original reports, research, and data had taken place. One of them, also frequently encountered in court, is that very young children, especially infants, are automatically assumed to be the victims of "shaken baby syndrome."<sup>14-16</sup> However, from the literature and from personal experience, findings of direct impact to the head prevail. The problem is not only semantic in nature but has major and far-reaching consequences since the character, location, and clinical presentation of the injuries are different from the rare purely shaken babies.<sup>17,18</sup>

Another encountered fallacy is that the children who die of head trauma, especially abusive head trauma, sustain DAI. Going back to some of the original research, it is clear that the authors explicitly stated that the 2 worst types of head injury are SDH and DAI. These 2 have different mechanisms of causation: SDH occurs much more commonly in nonvehicular injuries, such as falls and abusive head trauma, while DAI is caused almost exclusively by vehicular mechanisms.<sup>19-22</sup> Although both injuries frequently share a common mechanical cause such as angular acceleration, they differ in degree. SDH usually occurs with a rotational injury of short duration and a high rate of acceleration. Conversely, motor vehicle accidents tend to cause longer-duration, lower-acceleration-rate injuries leading to DAI rather than SDH.<sup>8,21</sup> SDHs occur in a greater number in children with inflicted versus noninflicted traumatic brain injury, whereas shear injuries are commonly visualized in the noninflicted injury group.<sup>22</sup> Therefore, current supposition that the presence of SDH is a marker of DAI is likely inaccurate.

A frequently asked question is whether delayed mental status deterioration can occur following head injury in children. This is critically important in unwitnessed circumstances such as child abuse. A widely held dogma is that if a child becomes unresponsive while in the care of an individual who is reporting the onset of unconsciousness, that same individual must be the perpetrator. Currently, some special-

ists involved in the care of abused children accept as true that all children who eventually die, regardless of the type of the head injury, must be severely disabled, usually comatose from the very moment the injury occurred.<sup>23</sup> From personal experience and based on the literature review, this tenet is not necessarily true.<sup>24,25</sup> Although there are clearly scenarios in which this principle could be applicable, there is undoubtedly a subpopulation of infants and especially toddlers with a completely different constellation of injuries and a dissimilar presentation. Occasionally, these children have nonspecific symptoms for several hours to a day prior to the onset of either coma or seizure followed by coma. Common observations include reduced physical activity, lethargy, drowsiness, irritability, temperature irregularities, poor feeding, and gastrointestinal symptoms.<sup>23,26,27</sup> Careful analysis of the history and the events leading to the critical symptoms indicate that there was a certain progression of symptomatology.

Occult intracranial injury in infants younger than 12 months of age is not uncommon.<sup>28</sup> Clinical symptoms and signs are insensitive indicators of intracranial injury in infants.<sup>29</sup> Radiologic observations can sometimes be of limited value as well.<sup>30</sup> Also, slow deterioration following mild head injuries in children have been reported.<sup>31</sup> Furthermore, 1 of the most frequently cited articles on restricting the time of injury in fatal inflicted head injuries draws its pediatric population mainly from motor vehicle accidents, with the average age of the study group patients being 8.5 years, with a SD of 4.0 years.<sup>32</sup>

Although many studies have offered guidelines for determining the age of cerebral injuries, various factors limit the reliability of these methods; for example, reduced cerebral blood flow may impede the cellular response. Not infrequently, injured children survive in the hospital for additional 2 to 3 days or even longer, sometimes undergoing craniotomy, rendering timing of the injuries based on the autopsy findings, including histologic examination of the cerebral injuries, extremely difficult.<sup>8,23,33-35</sup>

DAI is most likely a rarity in nonaccidental head trauma, and the term is misleading.<sup>17,18,36</sup> Coma may be more of a reflection of the severity of axonal damage in particular regions of the brain, most notably the brainstem, rather than the total sum of axonal injury distributed throughout the brain. Furthermore, the plane of head rotational acceleration plays an important role in determining both the distribution of axonal damage and the production of coma.<sup>36</sup> The localized axonal damage demonstrated in corticospinal tracts in the lower brainstem and rostral cervical cord, presumably caused by stretch to the neuroaxis produced by cervical hyperextension, may be more significant. This finding also provides an explanation for the frequent occurrence of apnea at presentation. In many of the cases reported by Geddes et al,<sup>18</sup> the axonal damage at the craniocervical junction was survivable; what was life-threatening was the subsequent hypoxic injury

and brain swelling. In addition, true contusional tears, which are peculiar to the brains of young infants, represent localized “shearing” between gray and white matter after an impact and should not automatically imply DAI.<sup>17</sup>

Cerebral hypoperfusion, followed by hypoxia/ischemia and diffuse brain swelling, characteristic in injured children younger than 24 months of age, are key pathophysiological findings associated with poor outcome following severe traumatic brain injury.<sup>8,37–39</sup> Primary brain damage occurs at impact and appears immediately or shortly after injury. Secondary brain injury may be more important, particularly in delayed fatalities, and occurs distant to the impact. Secondary events may not become apparent until several hours after injury. The largest controlled neuropathological study of nonaccidental infant head injury showed that axonal damage occurred in the brain of both head-injured subjects and in controls in the same distribution. This is not DAI but rather diffuse vascular or hypoxic-ischemic injury due to brain swelling and raised intracranial pressure. The study demonstrated that the diffuse brain damage responsible for loss of consciousness is a hypoxic secondary reaction and argues against DAI. One of the main conclusions of the study was that focal, localized axonal injury and secondary vascular-hypoxic changes characterize the mechanism of brain death.<sup>18</sup>

In conclusion, we present a case of a seemingly minor brain injury in an infant with a symptom-free interval, which resulted in delayed, sudden death. The importance of the mechanism of injury, location of injury, age of the child, and secondary brain injury with special reference to nonaccidental head trauma of childhood, as well as need for further research, are discussed. Although this is a rare presentation of a traumatic brain injury, based on recent advances in traumatic neuropathology, it is conceivable, as in this case, that a delayed asymptomatic deterioration to death can occur.

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