

CASE REPORT

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Cerebral Traumatism With a Playground Rocking Toy Mimicking Shaken Baby Syndrome

ABSTRACT: Shaken baby syndrome (SBS), one of the most deadly and devastating forms of child abuse, is caused by violent shaking. The combination of subdural hematoma, retinal hemorrhage, brain swelling, and diffuse axonal injury is highly typical of this syndrome and faced with these autopsy findings, induced traumatic lesions are strongly considered. However, it is known that motor-vehicle accidents and falls from great height can also produce this pattern of injury. Nevertheless, stories of arms fall, couch fall, or bumped head while the baby is being carried are generally considered incompatible with SBS. We here report a case of a 2-year-old boy presenting with all the classic autopsy findings of SBS from a playground rocking toy shaken by an older child.

KEYWORDS: forensic science, forensic pathology, traumatic brain injury, shaken baby syndrome, child abuse

In the last decade, the literature on shaken baby syndrome (SBS) has proliferated enormously. A Medline database search for the term “shaken baby syndrome” between 1966 and 1998 identified 71 articles while a similar research covering the 1999–2006 period revealed 235 articles. There were therefore three times more papers published on that subject in the last 8 years than there were for the previous 33-year period. Consequently, forensic clinicians can sometimes feel overwhelmed by all this literature, often contradictory from one paper to another.

The SBS refers to the severe brain injury caused by the violent shaking of an infant (1–5). Classic signs of shaking-induced damage (the “classic triad”) include subdural hemorrhage, brain swelling, and retinal hemorrhages (3,4,6). Additional signs can be encountered and further support the diagnosis: diffuse axonal injury, subarachnoid hemorrhage, retinoschisis, ribs and long bones fractures, and bruises on the child grabbing areas (2,4,5). However, in 2003, a controversial paper contested the long-held belief that the classic triad indicated that an infant had been subjected to non-accidental head injury (7). The authors of this study, based on a pathological study demonstrating microscopic hemorrhage in 36 of 50 dura of intrauterine, neonatal, and infant deaths, speculated that the triad was not caused by shaking forces but by a combination of cerebral hypoxia, raised intracranial pressure from brain swelling, raised arterial pressure, and raised central venous pressure. Although some enthusiastically embraced this theory (8,9) and used it in courtrooms, most specialists were highly skeptical (10–14). Finally, this theory known as the “unified hypothesis” was refuted by its own initiator: under cross-examination in the Court of Appeal, Dr. Geddes accepted that her hypothesis was meant to stimulate debate and not to be taken as a fact, and furthermore, that the hypothesis was inconsistent with the lack of subdural and retinal hemorrhages seen in the majority of clinical circumstances of

acute severe hypoxia associated with a sudden rise in central venous pressure and intracranial pressure (6). At last, the classic triad of SBS as indicator of head injury has stood the test of time (6).

The classic lesions of SBS are caused by an acceleration–deceleration mechanism (4,15). The child is seized by the chest or shoulder and violently shaken for an average of 5–15 sec, the head being whipped back and forth in the anterior–posterior direction (4). This head-whipping movement, combined with the disproportionate head dimension of infants and poor neck muscle strength, generates repetitive movement of the brain within the skull and tearing of bridging veins (4,15). Similar acceleration–deceleration biomechanical forces can be generated in other forms of traumatic circumstances like motor-vehicle accidents and falls from great height (4,15). These special circumstances are therefore possible SBS mimickers.

We here report the case of a 2-year-old boy who presented autopsy findings strongly suggestive of a SBS. However, investigation revealed that lesions were secondary to a playground rocking toy accident.

Case Report

The day preceding his death, a previously healthy 2-year-old boy was at a public playground with his two older brothers, of 3 and 6 years of age, and his 12-year-old caregiver. The boy, sitting on a rocking toy shaped as a motorcycle and mounted on a large spring (Fig. 1), apparently asked his caregiver to swing the playground toy. The 6-year-old brother was shaking the toy from behind while the caregiver was holding the handlebars of the toy to push back. After about 4–5 min of intense violent rocking, the boy apparently lost grip and his head struck the motorcycle handlebars. The child immediately started crying and a bruise appeared on his forehead. A few minutes later, he had stopped crying and went back to play until his carer decided to bring everybody home.

Once at home, ice was placed on the victim’s forehead. The child seemed well and was put to sleep about an hour and a half

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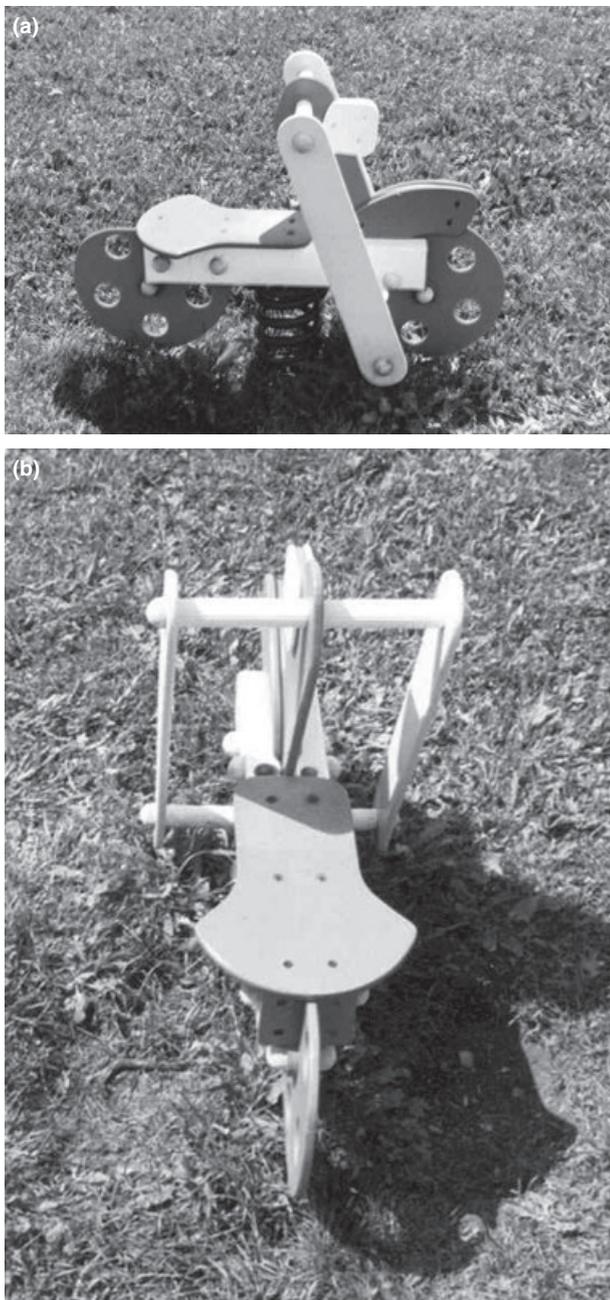


FIG. 1—(a), (b) Playground rocking toy shaped as a motorcycle and mounted on a large spring where the boy was shaken.

later. Later in the night, it was noticed that the child was breathing irregularly and the boy was thus sent to the hospital. The boy died during acute subdural hemorrhage surgical drainage. The body was transferred to the forensic institute for autopsy, considering strong SBS suspicion.

At autopsy, except for the postsurgical findings, external examination revealed a large 10×9 cm bruise on the forehead and a 2.4×2 cm bruise on the lateral part of the right eyelids. A few other bruises of different ages were found on the arms and legs, near elbows and knees, compatible with minor trauma of child's play.

At internal examination, there was evidence of surgical drainage of a left subdural hematoma. Residual right acute subdural hemorrhage, bilateral subarachnoid hemorrhage, and diffuse cerebral

swelling were also noticed macroscopically. Besides, microscopic exam demonstrated diffuse axonal damage (grade I/III of the Adams' grading system) (16). The ophthalmic examination pointed out the presence of multiple bilateral retinal hemorrhages, extending to the anterior part of the retina, as well as bilateral meningeal hemorrhagic infiltration of both optic nerves. There were no other significant autopsy findings. Radiological skeletal survey as well as toxicological analyses were negative.

Cause of death was determined as a severe cerebral traumatism and SBS was strongly considered, given the presence of the SBS classic triad. However, despite classic triad findings, further investigation oriented otherwise.

Witnesses interrogation clearly demonstrated consistent testimonies on repetitive interviews. Given the fact that these witnesses were all children of 12 years of age or less, it is barely conceivable that they could have come with such an elaborated scenario to hide an abusive trauma. Hence, it was concluded that the 2-year-old boy sitting on the rocking toy suffered from an intense back and forth movement, thus creating acceleration/deceleration forces similar to those encountered in SBS. Ultimately, considering the autopsy findings along with the elements of investigation, it is possible to consider that the boy's death was related to an accidental head injury involving a rocking toy.

Discussion

In the present case, although the victim presented the classic triad of subdural hemorrhage, brain swelling, and retinal hemorrhages, death turned out to be related to an accidental brain injury. Some may argue that the victim was a little old for SBS. However, although SBS is generally encountered in infants (17), SBS has been encountered in older children as well (18). Furthermore, although diffuse axonal damage was present in this case, it was likely the result of anoxic encephalopathy rather than the primary traumatic event itself, as the victim remained conscious for a few hours after injury.

Current forensic practice of child maltreatment is still largely based on the concept described by Kempe et al. when they first introduced the "battered child syndrome": cornerstone of nonaccidental-induced injury stands in great part on inconsistency between clinical observations and reported event history (19). Likewise, to be able to evaluate if the given history could have generated similar forces to SBS, a good understanding of the biomechanical forces that come in play to create SBS classic triad is thus mandatory.

Biomechanical forces implicated in the pathophysiology of SBS are rotation and acceleration of the head by whiplash shaking, with sudden acceleration and deceleration of the head (4,5,15). Although an experimental model suggested that shaking alone may not be sufficient to produce the angular acceleration necessary to cause the classic triad (20), most authors believe that this model was faulty and that pure shaking (without an associated impact) can cause death in infants and young children (21–24). Nevertheless, it seems that while pure SBS does exist, most cases present with impact injuries as well (20,23,25). As a matter of fact, shaking can occur in combination with impact, children being violently shaken and then forcefully thrown or the head being bumped against a surrounding surface or object during the shaking movements (4). Those cases are often referred to as shaking-impact syndrome. The present case belongs to this last category, because apart from the shaking on the rocking toy, an impact of the forehead was also present. Nevertheless, apart from shaking itself and shaking with impact, SBS classic triad is not compatible with a

minor trauma history such as simple falls from a couch or from parent's arms, or a story of a bumped head during baby carrying (3,4,6). On the other hand, as previously mentioned, motor-vehicle accidents and falls from great height are considered possible mimickers of SBS (4,15).

One unusual case has been reported in 2004 by Lantz et al., describing a particular situation that seems to also have mimicked SBS (26). It is the case of a 14-month-old child found on the floor with a television covering his head and chest. Autopsy revealed symmetrical parietal skull fractures, bilateral subdural and subarachnoid hemorrhages, thin epidural hematoma, brain contusions, severe cerebral edema, diffuse axonal damage, optic hemorrhages, perimacular retinal folds, retinal hemorrhages, and retinoschisis. The pediatric ophthalmologist was absolutely convinced that perimacular retinal folds coincident with retinal hemorrhages were specific for SBS, and Child Protective Services removed the 3-year-old sibling from the victim's home. However, Lantz et al. pointed out that the consistent history given by both the father and sibling should have been taken into account: the television, which was mounted on a homemade unstable stand, fell on the child's head (26).

A case with some similarities to ours was reported in 2003 by Jones et al. It is the case of an 8-week-old infant who was admitted to the hospital and found to have bilateral subdural hemorrhages, brain swelling, and retinal hemorrhages (27). The child died the following day. The carer stated that he saw the victim's 14-month-old sibling vigorously shaking the baby-rocker. Experimentation with the baby-rocker, using Duhaime's model, was performed to assess the possibility of this history. They asked a child of an approximate age (12.5 months) and then an adult to vigorously rock an anthropomorphic infant dummy in the rocker. Based on such models, they established that experiments did not support the account proffered by the carer which involved the shaking of the baby-rocker in the manner described. However, they modulated this statement with the mention that investigation of the child's death was far-reaching but inconclusive, with no definite answer as to what really happened. Furthermore, the point of their paper was not to attempt to determine what actually did cause this child's injury but rather to demonstrate the potential role for biomechanical investigation. On the other hand, experimental limitations of biomechanical analysis are obvious and often hard to deal with. For example, no quantitative head injury tolerance data exist specifically for children while anthropomorphic dummies respond differently from human tissues in terms of dynamic response (27). Even though SBS models have been developed based on animal experimentation, mathematical modeling, accident reconstruction, and cadaver studies (27–30), great caution should be exercised when applying these models to assess consistency of given history with clinical observations (27).

Taken individually, none of the SBS classic triad signs is pathognomonic of the syndrome. It is the gathering of autopsy findings consistent with SBS combined with the exclusion of every other differential diagnosis that can lead to a final diagnosis of SBS being put forward. Hence, the importance of thorough investigation before concluding whether a head injury is caused by abuse cannot be emphasized enough.

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