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SPECIAL ISSUE: ABUSIVE HEAD TRAUMA

Alternate theories of causation in abusive head trauma: What the science tells us

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Abstract When cases of suspected abusive head trauma are adjudicated in courts of law, several alternative theories of causation are frequently presented. This paper reviews common theories and examines their scientific basis.

Keywords Abusive head trauma · Alternative theories · Evidence · Infants

Introduction

When cases of suspected abusive head trauma are adjudicated in courts of law, several non-traumatic origins of the physical signs and symptoms are often presented by experts. This paper reviews common theories presented in cases of alleged abusive head trauma and examines their underlying scientific basis.

Theory: Normal, healthy infants die suddenly many months after birth because a birth-related subdural hematoma rebleeds spontaneously after minimal trauma [1]

Subdural bleeding is commonly seen after normal, uncomplicated deliveries [2–7]. These bleeds are usually asymptomatic. They are more likely to be seen around the falx cerebri, in the posterior aspects of the cerebrum, and in the posterior fossa [1, 3, 5]. Subdural hematomas are found more commonly in infants delivered by Caesarean section and more commonly in infants delivered by forceps or ventouse [3, 6, 7]. In two studies that followed asymptomatic newborns with subdural hematoma, investigators found that almost all the bleeds resolved completely within 4 weeks [3, 5]. In one case, the subdural hematoma was documented to have resolved in 3 months [5].

When infants with pre-existing head injuries are followed over time, small rebleeds within or around the original bleed are often identified on repeat imaging studies [8]. The infant's clinical status does not change, however. In other words, the rebleed is an incidental finding on the imaging study and is not reflected in the infant's neurological status. These rebleeds are more commonly found in infants who have brain atrophy. This most likely reflects the fact that "nature abhors a vacuum." Because the brain tissue is shrinking, the chronic subdural can expand to fill the space.

On occasion, infants with pre-existing head injuries have a gradual change in their neurological status as continued rebleeding occurs and the subdural hematoma gradually expands [8]. These cases do not present as an acute collapse in a previously healthy infant. In fact, there exists no single well-documented case in which a completely normal infant who had had normal head growth suddenly collapsed and sustained severe brain injury because of a rebleed into a pre-existing subdural hematoma.

Subdural neomembranes are sometimes seen in infants who die from non-traumatic causes. In these infants, subdural collections of fluid are not noted, and the neomembranes are an incidental finding [9]. In addition, once a subdural bleed occurs, iron staining of the dura can persist for years in the absence of continued bleeding [10]. The presence of a neomembrane or iron staining of the dura does not necessarily indicate a death was caused by rebleed into an old subdural hematoma.

Certainly, both chronic and acute subdural hemorrhage can be seen in infants who have been victims of repeated episodes of abusive head trauma [11]. Not uncommonly, the identified

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abuse episode is not the first time the infant has been abused [12]. In fact, episodes of abusive head trauma are frequently misdiagnosed as accidental trauma or natural illness [13].

Prospective studies of the clinical state changes in cases of known infant head trauma when rebleeds are identified would be helpful in resolving the bleed/rebleed controversy in cases of suspected abusive head trauma.

Theory: Subdural hematomas in infants are caused by episodes of hypoxia

The theory that subdural hematomas in infants are caused by hypoxia was initially presented by Geddes and colleagues [14]. In their study the dura from 50 infants who died from natural causes were compared with the dura of 3 infants who died from abusive head trauma. Seventy-two percent of the natural-cause group had "intradural bleeding." Of this group, only one infant had a visible subdural hematoma on radiologic studies or at autopsy. This child died of complications of extreme prematurity, sepsis and presumed disseminated intravascular coagulation. The authors also speculated that hypoxia could account for retinal hemorrhages as well, even though the results of retinal exams were not actually reported in the non-traumatic death group.

This paper has been widely criticized. Limited clinical data were presented in the paper. The investigators did not know how the dural specimens had been collected. This is important because bleeding within the dural leaflets has been reported as an artifact caused by traction on the dura when it is pulled off the skull [15]. Even Geddes herself acknowledged, "This was only a theory" when questioned about the paper in court [16].

Since this paper was published other authors have presented papers advocating this theory [17–19]. In reality, however, when cases of known death from hypoxia in infants and children have been reported, subdural hemorrhages were not present [20–22]. Many infants who experience episodes of confirmed hypoxia are imaged to determine the extent of brain damage. Subdural hematomas are not seen in drowning and suffocation.

The concept that the sudden collapse of an infant resulted from hypoxia was again presented in a 2007 paper by Barnes et al. [23]. The authors presented a case of an infant who died after choking on his formula. Several aspects of the paper, however, are problematic. First, the authors did not disclose that they had been hired as expert witnesses to review the case for the defense. They had not actually taken care of the child in a clinical setting. They failed to completely report the infant's injuries. They failed to note inconsistencies in the histories given by the caretaker. Finally, they failed to note that the alleged perpetrator in the case was found to be guilty of child abuse by the jury and that the conviction was upheld on appeal.

Theory: Unexplained subdural hematomas in infants can be the result of venous sinus thrombosis

In a growing number of court cases, unexplained subdural hematomas in infants are attributed to primary venous sinus thrombosis. In rare cases this has been reported in adults with severe underlying illnesses [24–28]. Newborn infants are known to be more susceptible to venous sinus thrombosis and often are found to have coexisting subdural hematoma, as well [29]. Clinical examples of well-documented subdural hematoma in infants resulting from venous sinus thrombus (in the absence of trauma) have not been reported after the post-natal period. In fact, a recent study specifically examined imaging studies on infants who presented with primary venous sinus thrombosis and found that none of the infants who were not newborns had subdural hematoma [30].

Venous sinus thrombosis can certainly be found in infants and children who die of severely elevated intracranial pressure causing venous stasis. In cases where the severely increased intracranial pressure is caused by trauma, the coexistence of venous sinus thrombosis and subdural hematoma are not likely to indicate causation [31].

Frequent causes of venous sinus thrombosis in children include malignancies, dehydration, metabolic acidosis, central nervous system infections, cyanotic heart diseases, head injury, craniotomy, renal disorders and thrombophilic disorders [32].

Theory: Subdural hemorrhages in infants are caused by immunizations

Because most infants receive vaccinations multiple times during the first year of life, it is not surprising that sometimes abusive head trauma occurs after immunizations. Some anti-vaccine proponents claim that brain swelling and intracranial bleeding diagnosed as shaken baby syndrome are actually caused by vaccines [33, 34].

The Health Resources and Services Administration (HRSA) recently commissioned the Institute of Medicine to review the epidemiological, clinical and biological evidence regarding adverse health events associated with vaccines. The resulting 892-page book published in 2012 by the National Academies Press contained a comprehensive review of available cases and literature analyzed by a panel of experts [35]. The reviewers identified several cases of head injury resulting from falls occurring because of vasovagal responses to vaccines. These injuries included subdural hemorrhages in a few cases.

There is no evidence in the medical literature that vaccines cause infants to have any condition that could be confused with abusive head trauma.

Theory: The signs and symptoms of abusive head trauma can actually be caused by benign extra-axial fluid of infancy

Benign extra-axial fluid of infancy is a common condition in which infants have unusually large heads caused by expansion of the cerebral spinal fluid within the subarachnoid space [36]. It is thought to be caused by immature balance of the production and absorption of cerebral spinal fluid. The excess fluid resolves in a year or two and is not specifically associated with persistent developmental delays and disabilities. The question is, can this otherwise benign phenomenon put children at risk of presenting with neurological collapse, subdural hematoma and retinal hemorrhages mimicking abusive head trauma? Several investigators have reported subdural hemorrhages occurring in infants with benign extra-axial fluid after minor trauma [37–40]. In these cases the subdural hematomas are often localized rather than generalized. In addition, neurological collapse and retinal hemorrhages do not accompany the subdural hematoma. Benign extra-axial fluid might predispose infants to get subdural hematomas after insignificant trauma, but it does not explain the signs and symptoms of abusive head trauma.

One factor that complicates the interpretation of increased subarachnoid spaces and subdural hematoma is that bleeding from head trauma can cause expansion of subarachnoid space secondary to the clogging of the cerebral spinal fluid absorptive mechanism by red blood cells. Serial imaging of infants with severe head trauma and subdural hematoma often results in transient secondary expansion of the subarachnoid space.

Theory: Retinal hemorrhages are not indicative of abusive head trauma

Very specific patterns of retinal findings have been associated with abusive head trauma [41–45]. These include extensive retinal hemorrhages extending to the ora serrata, multilayer retinal hemorrhages (intraretinal, pre-retinal and sub-retinal), retinoschesis and retinal tears [44].

These findings are rarely found in infants and children unless they have underlying illness such as leukemia, bleeding disorders, severe accidental trauma or sepsis [46].

It is often claimed in the literature that retinal hemorrhages do not indicate child abuse. This is true, because retinal hemorrhages have been documented in a wide range of conditions including exposure to high altitude, high blood pressure, metabolic disease and genetic conditions [47]. In most cases, however, other causes of retinal hemorrhage result in small scattered hemorrhages in the posterior pole of the retina rather than widespread, multilayered hemorrhages [45]. When hemorrhages have the typical characteristics of abusive head trauma hemorrhages, the differential diagnosis is much more limited, and the other causes of this type of hemorrhages are not easily mistaken for abusive head trauma [47].

Many types of retinal hemorrhage have been associated with normal delivery in newborns. These clear spontaneously, usually within 6 weeks. Interpretation of retinal hemorrhages in newborns should take this into account [48]. Conditions that have not been found to cause retinal hemorrhages similar to those found in abusive head trauma include seizure, resuscitation, hypoxia and increased intracranial pressure [47].

Theory: Short falls can cause signs and symptoms of abusive head trauma

In 2001 Plunkett [49] published a series of 18 children who died after falling from playground equipment. These cases were collected from all of the cases reported to the Consumer Product Safety Commission from Jan. 1, 1988, to June 30, 1999, of children who fell on playgrounds and were seen in emergency departments. Out of more than 75,000 cases, 18 children died. None of the children was younger than 1 year. Limited autopsy or neuropathological data were presented. Some of the children had pre-existing medical conditions (arteriovenous malformation, bleeding disorder), one of the falls was unwitnessed and no autopsy was done. Several falls were substantially farther than the distance of 1.4 meters generally considered to define short falls. Finally, Plunkett stated, “Several of the children had retinal hemorrhages similar to those claimed to be caused by shaken baby syndrome” in spite of the fact that none of the children had documented indirect ophthalmoscopic exams done by an ophthalmologist.

Other studies have documented that short household falls very rarely cause death in children. Chadwick et al. [50] reviewed the literature and determined that the risk of death from short falls was less than 1 in 1 million. The rate of death in multiply witnessed childhood short falls documented in the literature is zero.

Theory: Biomechanical studies have proved that violent shaking of infants does not cause serious head injuries

It is claimed in courts that manual shaking has been shown in scientific experiments to be incapable of causing brain injury or subdural hematoma in infants in the absence of impact. Biomechanical experiments that claim to prove infants cannot be harmed by shaking have been based on rather primitive mechanical models [51, 52]. When models are constructed that are more biofidelic, the results of the effects of impact on the infant head are quite different [53].

Injury thresholds for the infant head and brain have not been determined [53]. Biomechanical calculations that claim that shaking cannot injure infants’ brains are based on the supposition that thresholds for injuries such as subdural

hematomas and axonal injuries can be calculated solely by size scaling from adult data; that is, small brains are less susceptible to injury than large brains. The assumption is made that the only difference between infant brains and adult brains is their mass. In fact, infant brain tissue is extremely different than adult brain tissue. Infant brains have a much higher water content than adult brains and they contain more vulnerable, unmyelinated axons. For example, the shear modulus for infant piglet brain tissue is significantly lower than that of the adult pig brain [54]. The infant brain is much more deformable when a load is applied.

Coats and Margulies [53] have stated that "no pediatric threshold data exist relating infant head [maximum peak angular acceleration] and [maximum peak-to-peak change in angular velocity] to traumatic brain injury. . . . The prediction of injury in the pediatric population using . . . adult data are further complicated by the paucity of information regarding scaling injury thresholds from adults to children. Based on size alone, tolerable levels of acceleration might be expected to be higher in children, but tissue properties also play a critical role in scaling adult data to children. We conclude that a more complete comparison between adult and pediatric tissue injury tolerances is required before any adult head injury data can be appropriately scaled to infants" [53].

The developing brain's biochemical response to trauma is much different from that of the adult brain. Traumatic brain injury sets in motion a number of metabolic cascades that cause secondary neuronal injuries. These secondary insults include excitotoxicity, ischemia, inflammation, oxidative stress, free radical damage, and apoptosis. All of these insults have been shown to be worse in infants than in adults [55]. The response of the infant brain to injury is exaggerated, causing more harm and more tissue damage in infants compared to adults after comparable injuries.

When all of the above factors are considered, there is extensive evidence that violently shaking young infants can cause serious head injury [56].

Conclusion

The evidence base supporting many of the explanations offered to disprove abusive head trauma is often quite weak. A critical reading of the relevant literature is required to make informed decisions about the diagnosis of abusive head trauma. Continuing research is expected to be helpful in making these decisions. Certainly in every case of suspected abusive head trauma alternative explanations for the medical findings must be sought, and a thorough workup for accidental causation or organic disease should be pursued.

Conflicts of interest None

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